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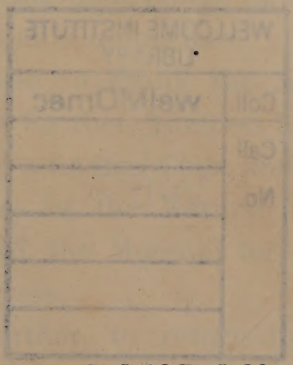
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XVIIth International Congress of Medicine

LONDON: 1913



General Volume



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PREFACE

THE Officers of the Congress desire to offer their heartiest thanks to :

The University of London,

The Authorities of the Imperial College of Science and Technology,

The Authorities of the Royal College of Science,

The Authorities of the Victoria and Albert Museum,

The Authorities of the Royal College of Art,

The Authorities of the City and Guilds Central Technical Institute,

The Board of Education,

The Royal College of Physicians,

The Royal Society of Medicine,

The Governors of St. Thomas's Hospital,

The Governors of the Royal Dental Hospital, Leicester Square, and

The Commandant of the Royal Army Medical College, for generously placing their buildings at the disposal of the Sections and National Committees.

They desire also to thank the Authorities of Queen Alexandra's House for their kindness in lending their rooms for the purposes of a Ladies' Club, and

The Authorities of the Natural History Museum for allowing the use of the Museum for the purposes of the Soirée of the Congress.

They have, further, to return their best thanks to many individuals and corporations, who were kind enough to entertain the members of the Congress. In especial they would desire to mention their gratitude to H.M. Govern-

ment, to the Lord Mayor and Corporation of the City of London, to the Grocers' Company, to the Royal College of Surgeons, to the Society of Apothecaries, to the Royal Society of Medicine, to the Royal Army Medical College, and to Lord Strathcona, G.C.M.G., G.C.V.O., for their generosity in this respect.

Other corporations and individuals were good enough to contribute towards the General Expense Fund. The Officers of the Congress desire to express their gratitude to the Royal College of Physicians of London, the Royal College of Physicians of Edinburgh, the Royal College of Surgeons of Edinburgh, the Society of Apothecaries, the Goldsmiths' Company, the Merchant Taylors' Company, the Salters' Company, the Clothworkers' Company, the Proprietors of the *Lancet*, Lady Butlin, and Lady MacCormac for their generosity.

The full members of the Congress numbered 5,546, including

British	1,823
Overseas Dominions	222
United States	675
Foreign	2,826

CONTENTS

PAGE

RULES OF CONGRESS 7

RULES OF SECTIONS 9

OFFICERS OF CONGRESS 11

DELEGATES OF GOVERNMENTS 15

REPRESENTATIVES OF UNIVERSITIES 19

NATIONAL COMMITTEES 23

GOVERNMENT DINNER 35

OPENING MEETING 36

GENERAL ADDRESSES :

Professor CHAUFFARD, 'Le Pronostic médical' . . . 57

Professor HARVEY CUSHING, 'Realignments in Greater Medicine' 71

W.G.R. Professor Dr. PAUL EHRLICH, 'Chemio-therapy' . 93

Professor W. BATESON, 'Heredity' 113

Right Hon. JOHN BURNS, M.P., President Local Government Board, 'The Relationship between Medicine and Public Health' 125

SERMONS :

Right Rev. THE DEAN OF ST. PAUL'S, 'The Medical Profession and Social Reform' 147

Right Rev. Bishop RYLE, Dean of Westminster, 'The Body of our Humiliation' 151

His Eminence Cardinal BOURNE, 'I was sick, and you visited me' 155

CLOSING MEETING 159

RULES OF CONGRESS

Article 1.—The Seventeenth International Congress of Medicine will be held under the august patronage of His Most Gracious Majesty George V, King and Emperor.

Article 2.—The Congress will be opened on Wednesday, August 6, and will close on Tuesday, August 12, 1913.

At the time of Congress, the Central Bureau will be located in the Royal Albert Hall, Kensington Gore, W., and will be opened for the inscription of Members on Tuesday, August 5, at 10 a.m.

Article 3.—The object of the Congress is exclusively scientific.

Article 4.—The Members of the Congress will be : (*a*) Qualified members of the medical profession, who have made formal application, and have paid the subscription hereinafter fixed ; (*b*) Scientific men who have been nominated by a National Committee or by the Executive Committee, and have paid the same subscription.

Article 5.—The subscription is : £1 sterling ; 25 kroner (Austro-Hungary) ; 25 francs ; 20 marks ; 15 rupees ; 20 kroner (Norway) ; 5 dollars (United States or Canada).

The wives and daughters of Members of Congress desiring to profit by the advantages accorded to them, must pay half the subscription fee. No male person is admitted except as a full Member.

Subscriptions should be sent by postal order or cheque payable to : The Treasurers, Seventeenth International Congress of Medicine, 13 Hinde Street, W., and the Section in which each member wishes to be inscribed must be indicated.

In the case of any country in which there is no system of international postage exchange, the services of a banker must be employed. No one can be enrolled as member of Congress before the receipt of his subscription.

An applicant when sending his subscription should enclose his visiting card indicating his medical qualifications and titles and his full postal address. Any change of address must be immediately notified. Cards of Membership will be sent out from the Central Office of the Congress within eight days following the receipt of the subscription.

Article 6.—Members of Congress may take part in the proceedings of Sections other than that in which they have been inscribed, but have the right to receive only the volume of the Transactions recording the proceedings at the general Sessions, as well as the Transactions of the Section in which they have been inscribed. The Transactions of other Sections can be purchased on order. Members of sub-sections will have the right to receive either the Transactions of the Main Section or the Transactions of the sub-section at their option. Members of the XVth and XVIth Sections will receive the Transactions of each Section.

Article 7.—The Sections of the Congress are twenty-three in number, besides which three sub-sections are established, namely : I, Anatomy and Embryology ; II, Physiology ; III, General Pathology and Pathological Anatomy ; III (*a*), (Sub-section) Chemical Pathology ; IV, Bacteriology and Immunity ; V, Therapeutics (Pharmacology, Physiotherapy,

Balneology); VI, Medicine; VII, Surgery; VII (*a*), (Sub-section) Orthopædics; VII (*b*), (Sub-section) Anæsthetics; VIII, Obstetrics and Gynæcology; IX, Ophthalmology; X, Diseases of Children; XI, Neuropathology; XII, Psychiatry; XIII, Dermatology and Syphilography; XIV, Urology; XV, Rhinology and Laryngology; XVI, Otology; XVII, Stomatology; XVIII, Hygiene and Preventive Medicine; XIX, Forensic Medicine; XX, Naval and Military Medicine; XXI, Tropical Medicine and Hygiene; XXII, Radiology; XXIII, History of Medicine.

Article 8.—The organization of the Congress is in the hands of the Organizing and Executive Committees.

Article 9.—There will be two General Meetings of the Congress, the Inaugural Meeting and the Closing Meeting. At these meetings the speakers will be the Government Delegates who have been invited by the Organizing Committee or designated as such, and these alone. At the Closing Meeting the President will announce the city in which the next Congress will be held. This will be determined by the Permanent Commission which will sit during the Congress.

Article 10.—The scientific work of the Congress will consist in: (*a*) general sessions; (*b*) sectional sessions; (*c*) combined sessions of two or more Sections.

Article 11.—The number of general sessions and the number of speakers will be fixed by the Executive Committee. There will be no debates in the general sessions.

Article 12.—The sessions of the Sections will be occupied in formal discussions on the Reports and in the reading and discussion of papers on subjects chosen by individual members of Congress. The work of the Sections is dealt with in separate regulations.

Article 13.—Two or more Sections may hold combined sessions.

Article 14.—The speeches delivered at the Opening and Closing General Meetings, as well as the Reports opening formal discussions, will be published in full. As to communications on subjects selected by individual members, only those papers will be published which the authors have personally presented to the Congress, and the Executive Committee, after consultation with the Sectional Committees, has decided to publish.

Article 15.—The manuscripts of speeches delivered at the General Meetings must be sent to the General Secretary. The manuscripts of remarks made in discussions, and papers read by individual members at the Sectional Sessions, must be delivered immediately to the Secretary of the Section concerned.

Article 16.—The Central Office of the Congress will use the English, French, and German languages for international business. In the General Meetings Italian and Russian may be used as well as these languages.

Article 17.—All correspondence should be addressed to the offices of the Congress as follows: The Hon. General Secretary, Seventeenth International Congress of Medicine, 13 Hinde Street, London, W.

Article 18.—On the envelopes of letters relative to the scientific work of the Sections, the Section to which they refer should be specified.

Article 19.—Information concerning the reductions in fares granted by railway companies, hotel and boarding accommodation, excursions, &c. will be published before April 30, 1913.

RULES OF SECTIONS

Article 1.—The Sections will meet at 9.30 a.m. and at 3 p.m.

Article 2.—The first Session of each Section will be opened on Wednesday, August 6, at 3 p.m.

Article 3.—The President of the Section will be responsible for the conduct of the discussions, for the application of the rules, and for the punctual accomplishment of the work of the Secretaries. If the President is prevented from being present at any Session of his Section, his place will be taken by one of the Vice-Presidents or by a Member of the Council of the Section.

Article 4.—There will be no Honorary Presidents of Sections.

Article 5.—The Sessions will be conducted according to the Parliamentary regulations in general usage.

Article 6.—The Sessions will include discussions on the Reports and the reading and discussion of papers on subjects selected by individual Members.

Article 7.—Reports. In each Section the morning Session will be reserved for the discussion of important questions which have been previously selected by the Council of the Section. Each discussion will be introduced by one or two Reporters chosen by the Council of the Section with due regard to the International character of the Congress. The manuscripts of the Reports must be typewritten, and must be sent to the Central Office of the Congress by February 28, 1913, at the latest. The Reports of each Section *which have arrived by the date fixed* will be printed and distributed as soon as possible, and before the opening of the Congress, to all members of the Section who have then been enrolled. Those that arrive later cannot be so distributed.

The Reports will not be read *in extenso* at the Session. Each Reporter will, however, be allowed a maximum of fifteen minutes for an opening *résumé*, and ten minutes for a reply at the end of the discussion. Other speakers taking part in the discussion will be allowed a maximum of ten minutes only for their remarks.

Article 8.—Members of Congress who desire to take part in the discussion of any Report may enter their names before the Congress by giving written notice to the General Secretary. During the Session they must communicate directly with the Secretary of the Section.

Article 9.—Speakers will be called upon by the President according to the order of their inscription on the Agenda.

Article 10.—Independent Papers. The afternoon Sessions will be devoted to the reading and discussion of independent papers. The titles of such papers should be announced to the Central Office of the Congress by April 30, 1913. The Council of the Section has the right of selection from among the papers offered, and of declining any that they do not consider desirable. The Council of the Section will arrange the order in which the selected papers shall be read. Any papers offered after April 30, 1913, will only be placed upon the agenda after the discussion of those which have been announced before this date and have been chosen by the Council of the Section. No paper will be accepted unless the title

has been received by the Secretaries of the Section before July 1, 1913. The text of every communication must be typewritten.

A maximum of fifteen minutes will be allowed for the reading of a paper, and five minutes for each speaker who takes part in the discussion. The author of the paper will be allowed five minutes for a reply.

Article 11.—Language. Reports and Independent Papers may be written in English, French, German, or Italian. Speakers may avail themselves of other languages, but only if one of the members present translates into one of the above four languages the sense of the speech.

Article 12.—Speakers will receive two intimations from the President as to their time limit; notice will be given two minutes before, and at the moment of expiry of the period allowed.

Article 13.—For certain communications of particular importance and general interest, the President may, with the consent of the Section, prolong by five or ten minutes the periods already indicated.

Article 14.—If a speaker wanders from the subject under discussion or indulges in personalities, the President may call upon him to sit down. If several members ask to speak upon a paper, and the hour is late, the President may on his own authority, or upon the proposition of a member, defer further discussion upon that paper to the end of the Session, if time permit.

Article 15.—The text of the remarks made in the course of discussions will only be inserted in the Transactions of the Congress if the speaker sends it in writing, condensed into twenty lines of print, to the Secretary of the Section before the end of the Session. (Block note-sheets will be placed for this purpose at the disposal of members by the Secretaries.)

Those who omit to conform to this regulation will lose the right to have their remarks published in the Transactions.

Article 16.—The Executive Committee reserves to itself the right to abridge the report of any discussion, and to omit any remarks of a personal character.

Article 17.—Private resolutions can only be proposed after previous notice given to the President, and when the business on the Agenda has already been disposed of. For the proposal of any special resolution a maximum period of five minutes only will be allowed.

The President will authorize only such resolutions as come within the limits of the work of the Section.

Article 18.—No vote may be taken, nor any resolution passed, upon questions of science or theory, but only on such questions as possess a practical or administrative character. On such questions the sense of the meeting will be taken by the majority standing or remaining seated.

The President will transmit such resolutions as shall be passed, through the General Secretary, to the Permanent Commission of the Congress. The Commission will decide whether the resolutions in question ought, or ought not, to be put to the vote at the Closing Meeting of the Congress.

Article 19.—The Secretaries of each Section will send an account of its transactions for the daily journal. This account will mention in chronological order the subjects of the Reports and discussions, the papers read, the names of the speakers, and any resolutions submitted to the Section.

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DELEGATES OF GOVERNMENTS AND OF UNIVERSITIES

INVITATIONS were addressed in 1911 by the British Government to suzerain states, and by the officers of Congress, acting on behalf of the organizing committee, to the various universities of the world.

No invitations were addressed to learned societies, corporations, or towns. It was considered that invited guests would have a claim to special privileges, and that the inclusion of societies, corporations, and towns would raise the number of privileged persons to a total which would deprive ordinary members of Congress of the chance of participating in any entertainments or excursions.

In response to the invitations, the following representatives were nominated. Many were nominated by Governments as representing universities. Such have been included in the Government lists, but the delegates nominated by universities will be found in a second list.

DELEGATES OF GOVERNMENTS

UNITED STATES OF AMERICA

Dr. T. F. Harrington.
Assistant-Surgeon-General J. W. Kerr.
Dr. J. Dawson.
Dr. Edward Parker Davis.
Dr. A. R. Allen.
Professor Arthur Bevan, University of Chicago.
Professor Frank Billings, University of Chicago.
Dr. Norman Bridge, University of Chicago.
Professor Harvey Cushing, Harvard University.
Professor George Dock, Washington University.
Dr. Simon Flexner, Director Rockefeller Institute.
Professor Theodore Janeway, Columbia University.
Professor Alexander Lambert, Cornell University.
Professor Thomas McCrae, Jefferson Medical College, Philadelphia.
Professor Stuart McGuire, University College of Medicine, Richmond.
Professor Louis C. McMurtry, University of Louisville.
Professor Rudolph Matas, Tulane University.
Professor Ramon Guiteras, New York Postgraduate Medical School.
Professor M. Allen Starr, Columbia University.
Professor W. S. Thayer, Johns Hopkins University.
Professor William H. Welch, Johns Hopkins University.
Dr. J. A. Witherspoon.
Dr. Cunningham Wilson.
Dr. George W. Crile.
Dr. W. J. Mayo.
Professor Frank Farrow Simpson, University of Pittsburg.
Dr. James Riddle Goffe.
Dr. John B. Murphy, University of Chicago.
Dr. William L. Baum.
Dr. Charles Enfield.
Dr. Hermann Biggs.
Dr. R. W. Craig.
Dr. Moses Greeley Parker.

ARGENTINA

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Professor Dr. R. Kraus.
Professor Eliseo Segura.
Dr. Gennaro Sisto.

AUSTRIA

Dr. Franz Ritter von Haberler, Ministerialrat (Minist. des Innern).
Professor Dr. Anton, Freiherr von Eiselsberg, Obersanitätsrat u. Hofrat.

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New South Wales. Dr. Ashburton Thompson.

New Zealand. Dr. Robert Haldane Makgill, Public Health Department.
Dr. F. Truby King.

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Comm. Merletti, Professeur à l'Université Royale de Ferrare.
Comm. Rossi, Professeur à l'Université Libre de Pérouse.
Comm. Ferrannini, Professeur à l'Université Libre de Camerino.
Comm. Perrando, Professeur à l'Université Royale de Gênes.
Comm. V. Ascoli, Professeur à l'Université Royale de Pavie.
Comm. Lucatello, Professeur à l'Université Royale de Padoue.

LUXEMBURG

Président : M. le Dr. G. Fonck, président du Collège médical à Luxembourg.

Secrétaire : M. le Dr. A. Praum, directeur du Laboratoire bactériologique à Luxembourg.

PORTUGAL

Président : M. le Professeur Bello Moraes, Lisbonne.
Secrétaire : M. le Dr. Antonio de Azevedo, Lisbonne.
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M. le Professeur Adelino Vieira, Coïmbre.
M. le Professeur Alberto Aguiar, Oporto.
M. le Dr. Alberto Mac-Bride, Lisbonne.
M. le Professeur Almeida Ribeiro, Coïmbre.
M. le Professeur Alvaro de Mattos, Coïmbre.
M. le Professeur Angelo de Fonseca, Coïmbre.
M. le Dr. Anciães Proença, Lisbonne.
M. le Professeur Annibal Bettencourt, Lisbonne.
M. le Dr. Antonio de Lencastre, Lisbonne.
M. le Professeur Antonio de Padua, Coïmbre.
M. le Dr. Armando Gonçalves, Coïmbre.
M. le Professeur Augusto Brandão, Oporto.
M. le Professeur Augusto Monjardino, Lisbonne.
M. le Professeur Augusto de Vasconcellos, Lisbonne.
M. le Professeur Ayrés Kopke, Lisbonne.
M. le Professeur Azevedo Neves, Lisbonne.
M. le Professeur Basilio Freire, Coïmbre.
M. le Professeur Bettencourt Laposo, Lisbonne.
M. le Dr. Bissaya Barreto, Coïmbre.
M. le Professeur Carlos Lima, Oporto.
M. le Professeur Candido de Pinto, Oporto.
M. le Professeur Custodio Cabeca, Lisbonne.
M. le Professeur Daniel de Mattos, Coïmbre.
M. le Professeur Dias d'Almeida, Oporto.
M. le Dr. Domingues d'Oliveira, Oporto.
M. le Professeur Egaz Moniz, Lisbonne.
M. le Professeur Elysio de Moura, Coïmbre.
M. le Dr. Fernando M. Chaves, Lisbonne.

M. le Professeur Francisco Gentil, Lisbonne.
M. le Professeur Gama Pînto, Lisbonne.
M. le Dr. Henry Mouton, Lisbonne.
M. le Dr. Jayme d'Almeida, Oporto.
M. le Professeur José Gentil, Lisbonne.
M. le Professeur José de Magalhães, Lisbonne.
M. le Dr. José Rodrigues, Coïmbre.
M. le Professeur Julio de Mattos, Lisbonne.
M le Dr. Lopo de Carvalho, Guarda.
M. le Professor Magalhães Lemos, Oporto.
M. le Professeur Maximiano Lemos, Oporto.
M. le Dr. Mello Breyner, Lisbonne.
M. le Dr. Mendes Correia, Oporto.
M. le Professeur Moreira Junior, Lisbonne.
M. le Professeur Oliveira Feijad, Lisbonne.
M. le Professeur Philomeno da Camara, Coïmbre.
M. le Professeur Pires de Lima, Oporto.
M. le Professeur Raposo de Magalhães, Coïmbre.
M. le Dr. Reio Stromp, Lisbonne.
M. le Dr. Reynaldo dos Santos, Lisbonne.
M. le Professeur Ricardo Jorge, Lisbonne.
M. le Professeur Roberto Prias, Oporto.
M. le Professeur Sabino Coelho, Lisbonne.
M. le Professeur Sergio Calisto, Coïmbre.
M. le Professeur Serrase Silva, Coïmbre.
M. le Dr. Silva Carvalho, Lisbonne.
M. le Professeur Silva Telles, Lisbonne.
M. le Professeur Sobral Cid, Lisbonne.
M. le Professeur Sousa Junior, Oporto.
M. le Professeur Thiago d'Almeida, Oporto.
M. le Dr. Virgilio Machado, Lisbonne.
M. le Dr. Pito Fontes, Oporto.
M. le Dr. Zeferino Falcad, Lisbonne.

RUSSIA

Président du Comité : D. Ott.
Vice-Président : J. Raptchevski.
Secrétaires : A. Korobkoff, B. Preobrajensky.

Membres :

Bekhtereff, W., St. Pétersbourg.
Bertenson, L., St. Pétersbourg.
Chapiroff, B., St. Pétersbourg.
Chidlovsky, S., St. Pétersbourg.
Dogel, A., St. Pétersbourg.
Dolganoff, W., St. Pétersbourg.
Dianine, A., St. Pétersbourg.
Enkhoff, T., St. Pétersbourg.
Ewdokimoff, A., St. Pétersbourg.
Fedoroff, S., St. Pétersbourg.
Fenomenoff, N., St. Pétersbourg.
Gubbenet, W., St. Pétersbourg.
Janovsky, M., St. Pétersbourg.
Khlopine, G., St. Pétersbourg.
Kolossoff, A., Doyen de la Faculté de Médecine à l'Université de Varsovie.
Lawroff, D., St. Pétersbourg.
Levacheff, W., St. Pétersbourg.
Likmatcheff, A., St. Pétersbourg.
Maximovitch, J., St. Pétersbourg.
Malinovsky, L., St. Pétersbourg.
Moisseeff, A., St. Pétersbourg.
Netchaeff, A., St. Pétersbourg.
Obolonsky, A., Doyen de la Faculté de Médecine de l'Université de Kief.
Pawloff, J., St. Pétersbourg.
Pawloff, T., St. Pétersbourg.
Podwyssotzky, W., St. Pétersbourg.
Popoff, M., Doyen de la Faculté de Médecine de l'Université de Tomsk.
Popoff, N., Doyen de la Faculté de Médecine de l'Université d'Odessa.
Prjibitek, S., St. Pétersbourg.
Razoumovsky, B., Doyen de la Faculté de Médecine de l'Université de Saratof.
Rauchfus, C., St. Pétersbourg.
Rein, G., St. Pétersbourg.
Simanovsky, N., St. Pétersbourg.
Sirotinine, B., St. Pétersbourg.
Tchistovitch, N., St. Pétersbourg.
Tikhomiroff, N., St. Pétersbourg.

Tonkoff, W., Doyen de la Faculté de Médecine de l'Université de Kasan.
Troianoff, A., St. Pétersbourg.
Weliaminoff, N., St. Pétersbourg.
Werkhovsky, B., St. Pétersbourg.
Wladislawleff, S., St. Pétersbourg.
Wreden, R., St. Pétersbourg.
Zernoff, D., Doyen de la Faculté de Médecine de l'Université de Moscou.
Zoueff, A., St. Pétersbourg.

SPAIN

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Vice-Président : Dr. Simonena, professeur de clinique médicale.
Secrétaire : Dr. Isidro Covisa, médecin général de l'hôpital.
Membres : Dr. Goyanes, chirurgien général de l'hôpital.
Dr. Elicegaray, médecin général de l'hôpital.
Dr. Hernando, professeur de thérapeutique à la Faculté de Médecine.
Dr. Castro, professeur d'anatomie à la Faculté.
Dr. Gomez Ocaña, professeur de physiologie à la Faculté.
Dr. Azua, professeur de dermatologie à la Faculté.
Dr. Covisa (D. José), syphilographe à l'hôpital de San Juan de Dios.
Dr. Cifuentes, urologo, chirurgien à l'hôpital de la Princesa.
Dr. Marquez, professeur d'ophtalmologie à la Faculté.
Dr. Fernandez Sanz, neurologue à la Faculté de Médecine.

SWEDEN

Président : M. le Dr. S. E. Henschen, professeur de médecine interne à l'Institut de médecine de Stockholm.
Secrétaire : M. le Dr. E. G. Nyström, professeur de chirurgie à l'Institut de médecine de Stockholm.
Membres : M. le Dr. G. Ahlström, professeur d'ophtalmologie à l'Université de Lund.
M. le Dr. K. Petrén, professeur de médecine interne à l'Université de Lund.
M. le Dr. G. Essen-Möller, professeur de gynécologie à l'Université de Lund.
M. le Dr. S. G. Hedin, professeur de chimie physiologique à l'Université d'Upsala.
M. le Dr. O. M. Ramström, professeur d'anatomie normale à l'Université d'Upsala.
M. le Dr. U. Quensel, professeur d'anatomie pathologique à l'Université d'Upsala.
M. le Dr. H. Petersson, membre du ' Board of Health '.
M. le Dr. H. Köster, chef de l'hôpital de Gothembourg.
M. le Dr. J. E. Johanson, professeur à l'Institut de médecine de Stockholm.
M. le Dr. G. Torssel, professeur à l'Institut de médecine de Stockholm.

SWITZERLAND

Président : M. le Professeur Dr. Th. Kocher, Berne.
Membres : M. le Professeur Dr. Roux, Lausanne.
M. le Professeur Dr. Sauerbruch, Zurich.
M. le Professeur Dr. d'Espine, Genève.
M. le Dr. Feurer, chirurgien en chef de l'hôpital cantonal, St. Gallen.
M. le Dr. Häberlein, président de la Caisse médicale suisse, Zurich.
M. le Dr. von der Mühll, président de la Commission médicale suisse, Bâle.
M. le Professeur Dr. Sahli, Berne.

GOVERNMENT DINNER

On the night of Tuesday, August 5, the British Government gave a dinner of 500 covers, to which members of the Congress were invited. Lord Morley took the chair, and was supported by Lord Beauchamp and Mr. John Burns.

Lord Morley proposed the health of the Congress, to which the President, Sir Thomas Barlow, M. Landouzy, and His Excellency Professor Dr. von Schjerning responded.

OPENING MEETING

On Wednesday, August 6, at 11 a.m., a general meeting was held in the Albert Hall.

The organist played a recital from 10.30 to 11 a.m. At that hour a procession, formed of officials of the Congress, preceded His Royal Highness Prince Arthur of Connaught up the hall to the platform. The procession was composed as follows :

Mace of the University of London.

Secretary-General (Vice-Chancellor of the University), Secretary of the Reception Committee.

Chairman of the Finance Committee, Chairman of the Reception Committee.

The Treasurers.

Chairman of the Executive Committee.

Master of the Society of Apothecaries
(Preceded by the Mace of the Society).

President of the Royal College of Surgeons
(Preceded by the Mace of the College).

The President of Congress and the Prince's Equerry
(Preceded by the Mace of the Royal College of Physicians).

The Secretary of State for Foreign Affairs.

H.R.H. Prince Arthur of Connaught.

When His Royal Highness had taken his seat at the table, the President asked him to open the Congress.

Prince Arthur spoke as follows :—

I feel that it is hardly necessary for me to assure you what very great pleasure it affords me to be present on this occasion to welcome in our midst, in the name of the King, the representatives of all branches of medical science from every quarter of the globe, who are gathered here to-day.

His Majesty, your Patron, has been graciously pleased to command me to give expression to his earnest hope that such international meetings may conduce to the advancement of the great science of medicine and to the general well-being of mankind.

His late Majesty King Edward VII, as Prince of Wales, opened this Congress in London in 1881, and it is a source of peculiar satisfaction to me that it is my privilege to follow in his footsteps and open this world-wide Congress on this occasion.

In 1881 Sir James Paget was President, and M. Pasteur, to whose wonderful discoveries the whole world is so much indebted, was present.

Marvellous as those discoveries were considered at that time, the discovery of Röntgen rays and radium within the last few years has furnished the medical world with powerful weapons for the diagnosis and treatment of disease, and I feel that I am voicing the opinion of all those here to-day in saying that we have worthy successors of the President and representatives of the 1881 Congress in the persons of Sir Thomas Barlow and the representatives from foreign countries and the British dominions beyond the seas assembled here to-day.

May I remind you that, although this Congress is meeting in London, it is not England alone which is the host. Canada, Australia, New Zealand, South Africa, and India are represented on the various committees with Englishmen, so it is really the Empire and not the United Kingdom which is giving this Congress—thereby forging another link in the imperial ideal.

I have had some experience of international exhibitions and am fully sensible of the good they have done in promoting friendly relations with foreign nations, and therefore I am quite convinced that even greater and more far-reaching international benefits may result from such a Congress as this, which affords opportunities for its members of acquiring and imparting knowledge to one another in a mutual exchange of ideas and discoveries.

A Congress with a membership of 6,000 persons constitutes a meeting of huge dimensions, and must surely appeal to the imagination, and although much of the work must necessarily be of a technical character, there will be many subjects of general interest, and the best men from all over the world have been chosen to introduce them.

I will not dwell on the various problems to be discussed, which will be explained by Sir Thomas Barlow; but I take this opportunity of congratulating the Reception Committee on the success of their labours, of which we have such evident proof to-day, and I have no doubt that, at the close of the Congress, they will feel fully repaid.

In conclusion, gentlemen, I have the greatest pleasure in welcoming you here to-day, and am further desired to inform you that His Majesty is very pleased that you are to be his guests at Windsor, and I venture to hope that you will find yourselves as much at home in this country as in your own homes, which, in some cases, are so very far away.

In the name of the King I declare this Congress to be open.

Sir Edward Grey, the Secretary of State for Foreign Affairs, then spoke as follows:

It is my most agreeable duty to offer to this Congress a welcome on behalf of His Majesty's Government.

I need scarcely say that that welcome is most whole-hearted and sincere. The Congress includes so many men of great distinction that we feel it an honour to have you assembled amongst us. Besides that, we know this to be a Congress that, while it accepts our welcome, cannot possibly have occasion to invite our intervention. Science is in the true sense of the word international. It has its controversies, but they are not national controversies. Men of all nations, who have risen to the plane of knowledge, thought, and research that is worthy to be called science, are not separated in their work on that plane by political or national rivalries, however much they may share the politics and feelings of their respective nationalities or parties in other departments of life.

We all have an individual interest in your work to an extent that hardly exists in the case of any other science. To some sciences we look

for much to add to the variety or amenity of life ; to yours we have learnt to look for something that comes home to us still more : the relief of suffering, the relief that takes from life one of its greatest terrors. Nor does your science appeal to the general thought of mankind in a less degree than any other. Medicine and surgery are, I suppose, in their scientific work at least as near to the study of what a lay mind would call the secret of life, or the secret of the universe, as any conceivable form of science can be. The imagination of men waits upon your work with the expectation that at any moment you may stimulate the thought and quicken the pulse of mankind by lifting still further a corner of the veil, and showing us yet a little more of what has hitherto been mysterious, unexplained, and unintelligible.

As far as the public mind is concerned, science is in one respect fortunate to-day : the rock of lay ignorance is not so intractable as it was in earlier generations. As far as the public is concerned, opposition has given way to expectation. We are more ready to welcome a new discovery than to oppose it with an old error. At least, I think that this is so with regard to those forms of science with which this Congress is especially concerned. If politics is included as a science, I should speak with considerable hesitation : for no one is very teachable in a subject which every one thinks that he understands. But as regards your work I think I may claim that the public is teachable, and not only well-disposed and grateful. The sovereign, the government, and the nation would like to welcome you with the greatest cordiality. We are all unfeignedly glad that London should be honoured as the meeting-place of the Congress and by the presence of so many men of world-wide distinction, who come from abroad to attend the Congress and to join in giving knowledge, lustre, and fame to its discussions.

The PRESIDENT then spoke as follows :

Your Royal Highness, My Lords, Ladies, and Gentlemen,—My first duty is to express our gratitude to His Majesty the King for graciously consenting to be the Patron of the Seventeenth International Medical Congress. My second duty is to thank His Royal Highness Prince Arthur of Connaught for representing His Majesty on this occasion in the inauguration of the Congress and for the important address which he has given to us. My third duty is to acknowledge the kindness and the consideration of Sir Edward Grey, the Minister of Foreign Affairs of this country, for the official and hearty welcome which he has given to our foreign members.

IN PRAISE OF FAMOUS MEN

A whole generation has passed away since the International Medical Congress last met in London. What a magnificent galaxy of talent in medicine, surgery, and pathology was gathered round the Prince of Wales, who was our Royal Patron at that time ! It is fitting that we should follow the admonition of Ecclesiasticus and praise famous men and the fathers that begat us.

Our President, Sir James Paget, was a great clinical pathologist. His mind was stored with all that was then known of the morbid anatomy of surgical disease and injury, and of the family relationship of the different diatheses. He was a splendid teacher and possessed a lucid eloquence and a moral fervour not excelled by any of his contemporaries.

Jenner and Gull, Wilks and Gairdner, were our great teachers of clinical medicine. Each of them based his knowledge on the sure foundation

of the post-mortem room and the hospital wards. We shall not see their like again, for their careers began before the days of specialization, and they were amongst the last of the great general physicians of our time.

Hughlings Jackson was the philosophical exponent of the new neurology. Many of his forecasts were verified by the experiments of David Ferrier, of which I may say there was a remarkable demonstration at the 1881 Congress.

Jonathan Hutchinson was the patient, accurate recorder of the natural history of disease in multitudinous departments, and, characteristically enough, he was the organizer of our Congress clinical and pathological museum.

The pioneers of abdominal surgery—Spencer Wells, Thomas Keith, and Lawson Tait—were with us. Huxley, the most brilliant expositor of natural science of his time, discoursed to us on the relations of medicine and biology.

William Bowman, whose work on the minute anatomy of the eye was the foundation of modern English ophthalmology, was one of our most useful members.

Last of all the Englishmen whom I will mention was our great Lister, then in the zenith of his grand career. He has but lately been taken to rest in the fullness of years, and we commemorate him to-day in the medal of our Congress.

Our foreign brethren were not less illustrious in the bede-roll of medical and surgical achievement.

Virchow, the Nestor of morbid anatomy, honoured and beloved by us as by his own countrymen, delivered a fine historical discourse on the value of pathological experiments. Volkmann gave a critical survey of the recent advances of surgery.

Robert Koch gave what may truly be called a path-breaking demonstration of the microbial findings in several morbid conditions, and he illustrated their characteristic growth on different organic media.

Von Langenbeck and Esmarch spoke for military surgery; Donders and Snellen for ophthalmology. Baccelli, Murri, and Pantaleoni represented Italian medicine. From the United States came Austin Flint, the accomplished physician and master of physical examination; Billings, prince of medical biographers; and Bigelow, the famous surgeon.

The great French school was represented by Brown-Séquard and Charcot, Lancereaux and Bouchard, and Verneuil and a host of others; but there was one great Frenchman with us who towered aloft amongst all his contemporaries, and who, though not a medical man, exercised by his discoveries a profound influence on the medicine of the world, and that was Louis Pasteur. In his address on vaccination in relation to chicken cholera and splenic fever he gracefully linked his most recent researches with the time-honoured labours of Edward Jenner on cow-pox.

Time fails me to speak of other great and honoured names, but surely we may say there were giants in those days.

The Parting of the Ways

Now let us realize to ourselves that the Congress of 1881 marked not the parting of the ways, but emphasized the notable fact that the parting of the ways had already been passed. The times of superstition, of empiricism, and of transcendental speculation had vanished. But what of the period of accurate and detailed observation? That was neither superseded nor completed, but it was already supplemented and redirected

into more fruitful channels by the new development of *experimental methods*.

If it had not been for the work of Pasteur, Lister, and Koch, which was expounded to us thirty years ago, how poverty-stricken would have been the output of medicine and surgery in this our Congress of 1913.

The great men—both observers and experimenters—of whom I have spoken were like mountain peaks towering above the plain of ordinary medical humanity, and we sometimes sadly ask, where are the mountain peaks now ? That is a shallow and unenlightened question. For, indeed, thanks to the unremitting labours of workers in multitudinous paths, we have attained a glorious heritage, not of high mountain peaks and deep valleys, but a lofty and magnificent tableland of well-ordered and correlated knowledge.

Consider the bare fact that the fifteen sections of the 1881 Congress have, by the inevitable specialization and concentration of work, become twenty-three sections and three subsections in 1913 ; but so imperative is the demand for mutual conference that we have no less than fourteen meetings arranged in which sections have found it desirable to discuss various problems in joint session.

In what ways have we pursued and expanded the work of our fathers?

The Developments in Microbiology and Parasitology

First, unquestionably in the development and application of bacteriology. Koch's great discovery of the life-history of the tubercle bacillus was published in the year after the London Congress, and what an enormous body of knowledge has grown out of that discovery ! We are learning to discriminate between the essential and causal factors of disease and the concomitants, such as combined and terminal infections. The by-products and the antibodies developed to neutralize bacterial life, of which we see the beneficent rôle in Nature's own cure of an acute specific disease, have been made to yield their share in two important methods of treatment—viz. serotherapy and vaccinotherapy. We have also faced the problem of strengthening the phagocytosis of the patient.

I need not dwell on the history of the Klebs-Löffler bacillus and the causation of diphtheria, nor on the indubitable efficacy of the most important of all the antitoxins, nor on the singular parallelism between the bacteriological findings in atypical throat exudations with the ambiguous symptomology which clinical observation reveals. Nor need I dwell on the extension of bacteriological investigation of typhoid which has been fruitful in new measures of prophylaxis and defence of the community.

We have learned something about the natural history of the ultra-minute organisms which as ' filter passers ' elude our microscopic investigation.

There are still great gaps in our knowledge of the infective organisms of the specific diseases, but it is a gain to have learnt, from the study of recent epidemics, that infantile paralysis must be grouped with the infective diseases, and thanks to Flexner we know many of the reactions of its elusive organism.

Great advances have been made in protozoology, in helminthology, and indeed in the whole subject of the relation of the parasites to the diseases of man and animals.

In tropical diseases these studies as well as bacteriology have brought about a rich harvest.

Mediterranean fever, plague, malaria, sleeping sickness, have all

yielded more or less of their secrets. Sometimes the whole cycle of the disease has been discovered, rationalized in every respect, and its successful treatment has been evolved. In other cases, as in malaria, sleeping sickness, and yellow fever, where only part of the natural history of the disease has been elucidated, nevertheless enough real knowledge has been acquired to enable important though sometimes costly hygienic measures to be successfully employed. Here it is fitting that we should offer our homage to our American brethren for their splendid hygienic work in Cuba, in Panama, in the Philippines, and in Costa Rica, and for the efforts which they are organizing for a world-wide crusade against ankylostoma disease.

An Outline of Progress in Many Directions

Chemical pathology has widened our knowledge and our resources, and the mystery of immunity has been to some extent illuminated.

The detailed examination of the morphological elements and the chemical characters of the blood and other body fluids has eventuated in the rewriting of some of our physiology, and the pathological extension of the knowledge thus gained has improved the diagnosis and the treatment of several diseases.

Thirty years ago Ord demonstrated to the Congress of that time examples of the disease which he had defined as myxœdema, but which, with surer instinct, Gull had described as a cretinoid state in adults. The gradual evolution of the doctrine of thyroid insufficiency and of its therapeutics is a model of induction. And this important discovery has given a great impetus to the whole study of internal secretions as well as to the employment of organic extracts, of which the last and most interesting is that of the pituitary body.

The empirical and then the experimental study of small variations in the ordinary diets of adults and children and infants in different social strata and in different countries has been fruitful in many unexpected ways. The great milk problem is still with us, but we have learnt the blunders of our early generalizations. Cleanliness in the milk-supply from start to finish has a far more exhaustive meaning than in days gone by.

The curious disease beriberi, which some of us have long thought had parallelisms with scurvy, has been shown, at all events among rice-eating people, to depend on the loss of the nutritive material just internal to the pericarp which the ordinary process of milling removes.

The patient study of chronic alcoholism has opened up a new chapter in nervous diseases. The routine traditional employment of alcohol in disease has happily been largely discredited. The open-air treatment of all forms of tubercular lesions has had a wide indirect influence not only on the treatment of other chronic ailments but on the daily life of the people.

The recognition and radical treatment of oral sepsis due to damage to the gums in consequence of various disorders of teeth has been followed by remarkable benefit. A strong case has been made out for intestinal stasis as a cause of various forms of malnutrition and for operative measures in dealing with slight mechanical obstructions, and on this subject we hope for further evidence.

The additions to diagnosis yielded by X-ray exploration are like the creation of a fifth sense, and its curative applications and those of radium are the opening of a new chapter of therapeutics.

I ventured to hint that medicine had now and then led to the rewriting

of some chapters of physiology, and I may add that recent researches on diseases of the heart have led to the re-editing of neglected knowledge of the minute structure of heart muscle and of orderly and disorderly mechanism of its movements.

The Triumphs of Surgery

Of the magnificent triumphs of the surgery of this generation it is beyond my power adequately to speak, but I can refer to the wide fields opened up through the beneficent protection of Listerism. We are staggered by the reasoned and calculated audacity of our brethren when sinuses of the skull are drained, cerebral abscesses evacuated, cerebral tumours removed, the pituitary body even being investigated; when pleuro-pericardial adhesions are freed to the great relief of the heart; when different parts of the alimentary canal are short-circuited; and when one or other damaged viscus is removed either entirely or in part. The active co-operation of surgeons and physicians has gained for us some knowledge of what Moynihan and others have happily described as 'living pathology', and we gratefully acknowledge the invaluable information of correlated symptoms, signs, and morbid conditions, and the statistics of comparative frequency which surgical experience has brought to the common store.

The supreme gain, after all, is that many more useful lives are saved than in the last generation, that the realm of grave and hitherto incurable disease is invaded on every side, and that the danger of operation *qua* operation is retreating to a vanishing point.

It is impossible even to enumerate the varied ways in which medicine has co-operated with economics, social legislation, and philanthropy, which we sum up briefly as public health. The school-house and the scholars, the home of the poor, the colliery and the factory, the dangerous occupations, the sunless life of the mentally deficient, have benefited, and will benefit still more, by its friendly invasion. And I venture to foretell that not many years hence every department of life and work shall be strengthened and purified and brightened by its genial and penetrating influence.

Conclusion

Surely I have said more than enough to justify my contention that we have come into a goodly heritage, and that that heritage is like a lofty and magnificent tableland of knowledge and efficiency.

The gaps are being filled; we are no longer isolated, but are working side by side on adjacent areas which are inseparably connected. Every day we gain fresh help from the auxiliary sciences, and we realize more and more the unity and the universality of medicine.

Brethren from foreign lands, we thank you for the treasures new and old of observation and experiment and of a ripe experience which you have brought to this Congress for the common weal. I venture to affirm that the output of work of the Congress week in its twenty-three goodly volumes will astonish civilized countries by the amount of its solid worth. I welcome you to our dear country, this ancient home of freedom, and I speak not only for the medical men of the British Isles, but for our brethren of the overseas dominions of this great Empire, who join with us in our cordial greeting.

May this Congress add to the common store of fruitful and useful knowledge, may it increase our good fellowship, our mutual understanding and co-operation, and may it help to break down the barriers of race and country in the onward beneficent march of world medicine.

The SECRETARY-GENERAL then presented his report, and as Vice-Chancellor of the University added a few words of welcome to the Congress.

The President then named the following as honorary presidents of Congress :

M. Chauffard
Professor Harvey Cushing
W.G.R. Professor Dr. Paul Ehrlich
Professor W. S. Thayer (America)
Professor Dr. Franz Ritter von Haberler (Austria)
Professor Paul Heger (Belgium)
Professor Rovsing (Denmark)
M. Landouzy (France)
Professor Waldeyer (Germany)
Dr. Pel (Holland)
Dr. Dollinger (Hungary)
Professor Bianchi (Italy)
Professor Kitasato (Japan)
Professor Uchermann (Norway).
Dr. de Ott (Russia)
H.E. Dr. Camilo Calleja (Spain)
Professor Essen-Möller (Sweden)
Professor Kocher (Switzerland)

The delegates of foreign Governments then rose in turn to greet the Congress.

UNITED STATES OF AMERICA—Professor William S. Thayer spoke as follows :

Your Royal Highness, Mr. President, Ladies and Gentlemen,—'Tis my happy privilege to bring to you to-day the greetings of the United States of America, to offer you our thanks for the warm and cordial reception which you have accorded us, to assure you of our earnest co-operation, and to express the hope that the efforts of our fellow countrymen may, in some measure, further the objects of this Congress.

There are certain emotions familiar to all who have taken part in these great international assemblies, which are very stimulating—the peculiar pleasure and satisfaction which arise from the realization of the unity of science, and of the inevitable fellowship of disinterested searchers for truth, no matter how divergent their social or political habits and ideals. The quickening impulse which springs from this consciousness, from these associations, promises here to be unusually strong.

Again, it is very inspiring to meet, here in Britain, the men upon whose shoulders has fallen the mantle of Linacre and Harvey and Sydenham, of Hook and Lower and Mayow, of Glisson and Boyle and Willis, of Jenner and Addison and Bright and Hodgkin, of Stokes and Graves and Corrigan, of Darwin and Wallace and Huxley, of Hunter and Cooper and Colles, and Simpson and Paget and Lister !

I need not refer to the pride which we Americans take in the thought that we too have a share in this great heritage.

And so, sir, gratefully conscious of the precious threads which bind us to a common past, we from across the sea bear to you in the dear old mother country something a little warmer than a mere formal greeting.

May that success attend the deliberations of this body which your generous efforts so richly merit !

ARGENTINA—Professor Eliseo Segura spoke as follows :

Au nom du Gouvernement de la République Argentine, que j'ai l'honneur de représenter ici, et me faisant le fidèle interprète de ses

désirs, je veux avant tout adresser mes plus grands hommages à Sa Majesté le Roi Georges V, sous l'Auguste Patronage duquel se trouve réuni ce Congrès ; à Son Altesse royale le Prince Arthur de Connaught, qui très gracieusement a consenti à présider cette séance inaugurale ; au Comité d'organisation, qui par son zèle ardent a assuré à l'avance la réussite de ce Congrès ; au très illustre corps médical anglais, qui depuis notre arrivée dans cette imposante capitale, tête du monde, nous a comblés de toutes sortes d'attentions ; et, enfin, à la grande nation britannique pour laquelle mon pays éprouve la plus vive sympathie.

Nous voici, médecins des cinq parties du monde, une fois de plus réunis, attirés par le même objet et animés de la même pensée.

En présence des différentes nationalités qui se trouvent représentées ici, on pourrait penser que cette assemblée se compose d'éléments hétérogènes entre lesquels persistent les divisions qui à un certain moment ont menacé la tranquillité du monde, mais heureusement il n'en est pas ainsi, et je crois ne pas me tromper, Messieurs, en affirmant que nous tous, membres de ce Congrès, nous avons abandonné momentanément notre cher pavillon national pour arborer le drapeau universal de la science médicale, qui porte comme devise : soulager celui qui souffre. Ceci est notre but et c'est pour sa réalisation que nous affrontons toujours avec courage les sacrifices que la clinique et le laboratoire exigent et c'est pour cela aussi que nous avons franchi de grandes distances pour venir participer à cette illustre réunion d'hommes de science venant chacun y apporter le fruit de son expérience personnelle.

Si notre mission est grande et noble considérée dans les rapports avec le malade, plus grande et plus noble encore est-elle par rapport à la collectivité. La plus grande richesse d'un pays se trouve dans la santé de ses enfants. L'homme sain lutte et triomphe de toutes les difficultés de la vie, et la vigueur de ses muscles et le fonctionnement normal de son cerveau sont un levier plus puissant que celui que voulait appliquer Archimède pour soulever la terre, car il possède en soi-même dans sa propre énergie le point d'appui que le savant chercha vainement.

Son effort ne connaît pas de limite ; de même qu'il cultive la surface de la terre, de même il pénètre hardiment dans ses entrailles pour en extraire soit les métaux précieux soit les matières que plus tard sa fertile intelligence transformera en force et mouvement ou mieux encore en vapeur et électricité qui sont les véritables facteurs du progrès universel.

Au contraire l'homme malade est une quantité négative, qui au lieu de produire, consomme, devenant en conséquence une lourde charge matérielle et morale : matérielle, parce qu'une bonne partie des éléments d'activité et de travail, au lieu de coopérer efficacement à la marche du progrès, doivent être consacrés à ses soins, et morale parce que la peine que le malade occasionne à sa famille enlève les forces et supprime les énergies, complétant de cette façon le tableau de désolation et de malheur du foyer.

Pour lutter contre cela, notre science est là. En soignant le malade, nous rendons à la collectivité un élément utile dont elle se trouvait privée et nous remenons le bonheur au foyer affligé. Son action va plus loin encore, car non seulement elle soulage les malades, mais encore elle s'occupe de faire l'homme fort en surveillant l'enfant dès sa naissance.

De plus, Messieurs, elle s'occupe de lutter contre les maux d'ordre social, tels que d'éviter la cruelle injustice de voir venir en ce monde des êtres d'un organisme imparfait et condamnés à payer toute leur vie la faute d'autrui, de même d'arrêter le bras du criminel qui tue, parce que ce bras est animé soit par les molécules d'alcool, soit par le germe de la

syphilis, qui ont préparé dans l'organisme de ses antécédents les éléments de dégénérescence et de destruction de l'homme futur.

Par conséquence notre champ d'action est très étendu et les Gouvernements désireux de coopérer au bien-être matériel et moral de leurs sujets, doivent accorder l'appui le plus grand à notre science en la dotant sans parcimonie de tous les éléments qu'exige son développement, tout spécialement, de vastes laboratoires et des hôpitaux modernes ; en échange, elle rendra au monde la richesse la plus grande, la santé de l'homme.

AUSTRIA—Professor Dr. Franz Ritter von Haberler spoke as follows :

Your Royal Highness, Mr. President, Members of Congress, Ladies and Gentlemen,—In the name of the I.R. Austrian Government, which I have the honour to represent at the Seventeenth International Medical Congress, I beg to render you the warmest greetings. It was a great pleasure to our country to receive the kind invitation of this country to send representatives to the Seventeenth International Medical Congress, and I therefore fervently wish that its deliberations may be crowned with success.

The importance of this scientific meeting consists not only in the great number of papers on medicine to be read at this Congress, but also in the personal contact by which so many states and nations are united for professional competition.

The fight against disease is indeed the most natural agent of concord between all nations ; all the branches of medicine are united in the pursuance of this task, which is the common object of scientific research and medical practice.

Public health, the special field to which I have to devote myself in my official capacity of referee for sanitary matters, expects, before all, assistance and co-operation from all the branches of medicine, and equally appeals for assistance to physicians and to the public in general, as it is the link between medicine and the institutions for public welfare.

The treatment of disease, the therapeutical line of medicine, has laid the foundation-stone of public health ; every progress of medicine, the development in the prophylactic line of medicine, seems to indicate that public health will make great progress in the near future.

The public sanitary administration as well as medical research and the institutions for medical education are consequently all highly interested in the efforts of this Congress, and I therefore beg to reiterate in this sense my best and most cordial wishes for the complete and lasting success of your deliberations.

BELGIUM—Professor Paul Heger spoke as follows :

Altesse royale, Monsieur le Président, Mesdames, Messieurs, — L'étranger qui aborde en Angleterre par Folkestone aperçoit en haut de la falaise la statue du grand physiologiste dont le génie domine depuis deux siècles l'histoire de la médecine.

Harvey se dresse sur le rocher ; il regarde vers le continent ; il semble imposer à tous ceux qui mettent le pied sur le sol anglais le respect de vos grandes traditions médicales ; son image fait naître en nous des sentiments d'admiration et de reconnaissance.

A mesure que nous pénétrons davantage dans votre pays, que nous visitons vos cathédrales, vos universités et vos collèges, ces sentiments grandissent et dans une occasion comme celle-ci, le souvenir de vos grands hommes est si présent à notre esprit que nous avons l'illusion de croire

qu'ils revivent un instant pour célébrer avec nous les progrès merveilleux de la médecine moderne. Harvey, Hunter, Sydenham, Jenner, Lister — et d'autres non moins illustres — occupent les places d'honneur dans ce Congrès où sont accourus les médecins du monde entier : ils vivront dans les siècles, aussi longtemps que des Congrès de médecine se réuniront sur la terre anglaise.

Au nom du laborieux petit peuple que je représente, je salue la grande patrie qui nous reçoit si généreusement.

BRAZIL—Professor Marcos Cavalcanti spoke.

CHINA—Dr. Wu Lien Teh spoke.

CUBA—Dr. Aristides Agramonte spoke as follows :

Your Royal Highness, Mr. President of the Congress, Ladies and Gentlemen : From the Government of the Republic of Cuba I have the honour to tender you sincere and warmest greetings. It is a long way to come 4,000 miles across the Atlantic, but we think that the distance is not too great when compared with the intense desire for moral and intellectual betterment which animates our people, and we feel that no national or topographical boundaries can limit the scope of science, nor can any difference of race or language render impossible the intercourse of men who seek for truth in the highest plane of the human activities.

We have banished from my country, I hope for ever, two of the worst infections which terrorized mankind — small-pox and the yellow fever. We harbour but a few of the so-called tropical diseases, we have no sleeping sickness, no beriberi, no leishmaniasis, and our sanitary conditions may be favourably compared with those of the most enlightened nations on the globe ; having performed this work in the field of preventive medicine, we thought that in spite of our modest political and territorial significance, our representation might not be considered entirely out of place amongst you.

Everything points to an equal if not greater display of hospitality on the part of our hosts than we enjoyed four years ago when we met in the city of Budapest, for which a sense of gratitude still abides with us ; may we celebrate many meetings such as this. Personally, I rejoice at the opportunity which this Congress affords to renew old acquaintances and to make new friends. I repeat, sir, my Government's salutation and my personal best wishes for the success of our sessions.

DENMARK—Dr. T. Madsen spoke as follows :

May it please Your Royal Highness, Mr. President, Ladies and Gentlemen ! In the name of the Royal Danish Government I have the honour to thank you for the gracious invitation to participate in the work of the Seventeenth International Congress of Medicine.

The intimate racial bonds, which through the centuries have connected the British and Danish nations, have made the latter particularly appreciative of the progress which has raised the great British nation into the front rank of civilization, and the glorious pioneer work which you have inaugurated for humanity has always found in Denmark a most fruitful and stimulating soil.

Therefore my Danish colleagues feel the greatest pleasure and gratification in being present at this magnificent meeting of eminent scientists gathered together from all parts of the earth here in the metropolis of the world, and I would express our heartfelt thanks for the splendid welcome given us to-day, and add the best wishes for the success of the Congress.

FRANCE—M. le Dr. L. Landouzy spoke as follows :

A Sa Majesté Georges V, dont l'auguste patronage devait assurer le succès du dix-septième Congrès international ; à vous, Altesse royale, qui nous accueillez si gracieusement ; à vous, éminent Président, Thomas Barlow, comme aux membres dévoués du Comité d'organisation, j'apporte, Président de la Délégation du Gouvernement de la République française, le salut respectueux, les hommages et les vœux des médecins, des corps universitaires, hospitaliers et académiques de France dont tant d'idées scientifiques et humanitaires, pareilles aux vôtres ont, jour à jour, fortifié l'Entente cordiale qu'elles avaient inspirées.

Certains, parmi nos délégués, sont les envoyés de l'antique université de Lutèce à laquelle se rendaient nombreuses tant de générations de vos 'escholiers'. La rue des 'Anglais', au flanc de la montagne Sainte-Geneviève, garde encore leur souvenir.

Nos vieilles écoles ont vu naître vos célèbres collèges de Cambridge, où grandirent Bacon, Milton et Newton ; d'Oxford, dont les murs connurent les méditations du plus grand médecin du dix-septième siècle, Thomas Sydenham.

Venus de la patrie des Lavoisier, Lamark, Bichat, Laennec, Pinel, Desgenettes, Bouillaud, Claude Bernard, Chauveau, Villemin, Pasteur, Bretonneau, Trousseau, Charcot, Duchenne de Boulogne, Ollier, Berthelot, Wurtz, Becquerel, Curien Lippmann, Branly, d'Arsonval, Sabatier, Armand Gautier, Le Chatelier, Laveran, Bourquelot, Moureu, Ch. Bouchard, Roux, Ch. Richet, etc., nous apportons notre collaboration ardente au pays des Jenner, Faraday, Garrod, Hunter, Cheyne, Bright, Addison, William Stokes, David, Robert Darwin, Lister, Adams, Lawson Tait, MacEwen, Ferrier, Crookes, Lord Kelvin, Rutherford, Gram, Sir W. Ramsay, J. J. Thomson, Horsley, Maxwell, Lord Rayleigh, Osler, Macfadyen, Arbuthnot Lane, Sir Dyce Duckworth, Lauder Brunton, Sir A. E. Wright, etc.

A cette liste incomplète, que de grands noms à ajouter..., ceux de Sir Patrick Manson, Sir W. Leishman, Sir David Bruce, Sir Ronald Ross, Dutton, Rogers, dont les travaux cliniques, bactériologiques et expérimentaux ont illustré l'École coloniale anglaise et éclairé la médecine tropicale. Ne nous ont-ils pas révélé, entre autres, le secret de la maladie du sommeil, des leishmanioses, de la fièvre récurrente et de la mélitococcie ?

Si, empressés, nous venons à Londres, c'est encore que le Royaume-Uni offre à notre étude des leçons de choses que vos hygiénistes, vos édiles, vos économistes et vos savants ont données au monde. Par vos lois d'expropriation pour cause de salubrité publique, par votre volonté de répandre l'air et la lumière dans vos laborieuses cités, n'avez-vous pas, en vingt ans, réussi à faire fléchir le chiffre de la mortalité tuberculeuse de 40 pour cent. La diminution n'a-t-elle pas atteint jusqu'à 50 pour cent à Londres ? C'est que Hyde-Park, Regent's-Park, Victoria-Park permettent à la capitale de respirer par de vastes poumons... n'appelez-vous pas ainsi vos espaces plantés et fleuris ?

Non content d'avoir, en quinze ans, dépensé trois milliards pour la destruction de quartiers insalubres, votre pays faisait, hier, un pas décisif dans la lutte contre la tuberculose, spécialement orientée vers la prévention ?

Votre gouvernement n'obtenait-il pas, l'an dernier, le vote d'une loi d'assurance contre la maladie sociale, en même temps que la déclaration et la désinfection obligatoires ?

Tous ces efforts de la science anglaise appliquée à la thérapeutique,

à l'hygiène, à l'économie sociale, à la pathologie tropicale, sont autant d'exemples nouveaux du sens pratique qui, dans toutes les branches de la biologie, et depuis des siècles, inspire les meilleurs esprits du Royaume-Uni.

C'est ainsi que vos législateurs, traditionalistes, mettent leur point d'honneur à pratiquer la pensée de Disraëli : ' Le souci de la santé publique est le premier devoir d'un homme d'État.'

Pareil souci préside aux grandes assises scientifiques et humanitaires que sont nos Congrès. Notre amour de l'idéal, notre recherche de la vérité, notre désir d'enquêtes et de découvertes aux fins de vaincre la misère morale et physique, s'y dépensent avec émulation pour que, par la médecine (devenue plus empêcheuse de maladies, que guérisseuse de malades), les individus, comme les peuples recueillent plus de soulagement dans la souffrance, et plus de justice dans la santé.

GERMANY—H. E. Professor Dr. v. Schjerning spoke as follows :

Unermüdlich wird die Arbeit in den Werkstätten der medizinischen Forschung fortgeführt. Ein Problem nach dem andern taucht auf, eine Frage nach der andern wird der Lösung zuzuführen gesucht. Und dabei stellen sich oft plötzlich neue und wichtige Aufgaben ein. An Stelle der Arbeiten über das Krebsproblem oder die Salvarsanbehandlung, über Tuberkuloseheilung und innere Sekretion, über Jugendpflege und Rassenhygiene lenken die kriegerischen Wirren auf dem Balkan mit einem Male die Wundbehandlung, die Kriegschirurgie und die Bekämpfung der Seuchen in den Vordergrund des Interesses, und die gemeinsamen kolonisatorischen Bestrebungen in Afrika fordern die Abwehr der Schlafkrankheit. Aber über viele, viele wichtige medizinische Fragen schwankt noch beständig die Meinung der Gelehrten, und nur schrittweise und langsam drängt sich die Wahrheit auf dem Wege der Forschung und Beobachtung vorwärts. Darum ist es für die Fortschritte dieser Wissenschaft von höchstem Wert, wenn einmal wieder die Gelehrten und die Männer der Praxis aus aller Welt das Facit ihrer Erfahrungen ziehen und im Austausch die Ansichten, das Ergebnis ihrer Forschungen mit Gleichgesinnten und Gleichstrebenden besprechen. Verdienstvoll und zukunftverheissend ist es demnach, wenn heute England einen Kongress der Aerzte aller Staaten und aller Nationen veranstaltet, der beraten soll, wie die ärztliche Kunst und Wissenschaft fortschreiten und wie man gegen Not und Krankheit erfolgreich weiter kämpfen kann zum Heil der gesamten Menschheit. In diesem grossen friedlichen Kampfe stehen deutsche Vertreter sowie die Abgeordneten aller Länder Schulter an Schulter neben England. Unsere besten und erleuchtesten Geister der Wissenschaft und Praxis sind mit mir aus meinem Heimatlande gekommen, um mit Ihnen zu arbeiten, damit dieser Kongress ein Merkstein werde in der Geschichte der Medizin, damit er würdig sei des Landes, das einst einen Jenner und Simpson, einen Darwin und Lister der Welt geschenkt hat, Männer, die wir unter die grössten Wohltäter der Menschheit zu rechnen haben.

In glänzender Weise ist Ihr Kongress eröffnet ; ich wünsche von Herzen, dass der Verlauf und der Ausgang dem Anfang entsprechen werden. Wir haben in Deutschland ein Wort, das heisst : Die Erinnerung ist ein Paradies, aus dem man nicht vertrieben werden kann. Ich zweifle nicht, dass dieser Kongress allen Teilnehmern eine solche freudige und wertvolle Erinnerung hinterlassen wird !

Im Namen der deutschen Regierungen, im Namen meiner deutschen Kameraden und Kollegen danke ich herzlichst für die Einladung und

den herrlichen Empfang. Ich glaube meinen Dank, meine Wünsche, meine Hoffnungen für den Kongress nicht besser ausdrücken zu können, als durch den Ruf: Es lebe England! Gott segne den erhabenen Protektor des Kongresses, Seine Majestät den König! Gott schütze Englands erlauchte Königin.

GUATEMALA—Dr. Don José Azurdia spoke as follows:

Your Royal Highness, Ladies and Gentlemen,—London, which was so recently the sanctuary of peace, to-day becomes the temple of science and learning. The echo of the last words pronounced at St. James's Palace, to pacify the near East, is still heard throughout the civilized world; and we are now about to hear from the mouth of science words on behalf of the welfare of mankind, words concerning the promotion and maintenance of health and the greater duration of life.

London has therefore been chosen, within a brief period of time, as the centre for the celebration of two of the most important and memorable acts that have been performed for the benefit of the human race and in the cause of peace and science, which means prosperity and prolongation of life.

May I be allowed to congratulate this city on being the proud seat of such great events.

May the Government of His Majesty King George V be pleased to accept our most cordial thanks for the very great courtesy it has extended to us.

Blessed be science and her representatives here met together, who are continually and earnestly engaged in the work of research with a view to finding practical means to maintain health and to prolong life; and in the name of the President and of the people of the Republic of Guatemala, I beg the delegates of all the nations here represented, as also each one of the illustrious members of this assembly, to accept the most hearty welcome to the labours of the Seventeenth International Congress of Medicine.

HOLLAND—Dr. van Rijnberk spoke.

HUNGARY—Professor Dr. Emil de Grósz spoke as follows:

Altesse royale, Mylords et Messieurs, — J'apporte à cette imposante Assemblée scientifique les salutations et les vœux du Gouvernement royal hongrois, des universités hongroises, des médecins hongrois, de la nation hongroise tout entière. Depuis que le septième Congrès international de Médecine siégea à Londres, il y a vingt-deux années, beaucoup de choses ont changé dans le monde. Tout d'abord, les sciences et les arts ont perdu un de leurs plus augustes et éclairés protecteurs en la personne du prince de Galles, ensuite roi Édouard VII, qui ouvrit alors le Congrès; permettez, je vous prie, que mes premières paroles rendent respectueusement hommage à sa vénérée mémoire. Puis la carte géographique de l'Europe et des autres continents s'est modifiée; les doctrines scientifiques elles-mêmes ont subi, à maints égards, des variations appréciables. La seule, l'unique chose qui n'ait point changé, c'est que l'Angleterre, maintenant comme dans les siècles passés, marche à la tête du progrès et de la civilisation.

Le succès des travaux du Congrès est garanti par le seul fait que nous sommes réunis dans cette brillante et magnifique métropole, dans la capitale du grand pays qui a donné à l'humanité les Newton, les Jenner, les Harvey, les Hunter, de ce pays où les plus belles et les plus nobles

traditions d'un long et glorieux passé trouvent toujours et constamment de dignes continuateurs.

Il est certain que les représentants de toutes les nations ont accouru avec le plus vif empressement et un véritable bonheur ici. Mais d'autres sentiments aussi font battre nos cœurs hongrois avec plus de force. Nous sommes fiers et heureux de remettre à la nation britannique le drapeau des sciences médicales que nous reçûmes il y a sept ans à Lisbonne et que nous gardâmes jalousement comme le palladium de notre profession. Et, *last not least*, nous avons l'insigne joie de déposer nos hommages tout particulièrement respectueux aux pieds de Sa Très Gracieuse Majesté la Reine et Impératrice Mary, dans les veines de qui coule du sang magyar.

D'un bout à l'autre du pays des quatre fleuves et des trois montagnes, comme l'on appelle la Hongrie, mais tout particulièrement dans l'Alföld aux merveilleux mirages et dans la Transylvanie aux alpes tourmentées, la mémoire de l'illustre aïeule de Sa Très Gracieuse Majesté se perpétue, impérissable, dans nos cœurs. Nous souhaitons, vibrants d'enthousiasme, que l'ardent patriotisme des vieux princes hongrois, des ancêtres de la reine Mary, comble la famille royale d'Angleterre et la nation britannique de bénédictions et de bonheurs.

Italy—On. Professor Leonardo Bianchi spoke as follows :

It is a great honour for me to express to the British Government the greetings of the Italian nation on this occasion of the periodic Festival of Medicine and exhibition of scientific progress, which is the holiday of civilization. Many Italians take part in this Congress because they have a keen sympathy with foreign scientific literature, and also because they are very conscious of the contribution which they themselves give to the progress of medicine. And I am so much the more pleased to have been chosen to represent the Italian Government on this solemn occasion in so far as I am one of the southern Italians who have had the privilege of observing the rapid moral and economic development of our native land, and also remember with gratitude the great English statesmen, and especially the late Mr. Gladstone, who, in his speeches and writings, contributed to hasten the day of our liberation from a government which he defined as the ' denial of God '. The biological spirit impresses irresistibly its own direction on civilization and on legislation, and for this reason permit me to express my hope that this Congress may be handed down to posterity as famous for the discussion of many new discoveries in the field of biology, from which emanate the impulses for a new conception of life and the ever changing conditions of existence.

Japan—Dr. Takamine spoke as follows :

Your Royal Highness, Mr. President, Ladies, and Gentlemen,—On behalf of the delegates of the Imperial Japanese Government, I have the honour to express the hearty greeting of our Government and our medical profession at the memorable occasion of the opening of this Congress.

It is very interesting to know that the introduction of modern civilization into Japan was really due to the implanting of a knowledge of medicine. It is true that communication with the rest of the world was formally established by Commodore Perry of the United States in 1854, but the real forerunner of modern civilization was the introduction of medicine by the Dutch, who settled in Nagasaki long before Commodore Perry came to Japan.

The Dutch were skilled physicians, and the natives appreciated the superiority which their methods exhibited over those of the old

Chinese school. Students flocked to Nagasaki from all parts of the country, and the latest medical knowledge spread to the principal towns of the country through the study of Dutch literature, whilst chemical and other branches of science were also made practical use of in relation to saltpetre, gunpowder, &c.

Whilst the attitude of the Government of that day was largely anti-foreign, the value of modern science was appreciated by the progressive spirits of the nation, so that when the formal opening up came there were many who were well prepared for the new era. During the reign of the late emperor, some of the younger statesmen, and others, embraced the spirit of modern civilization. It is also interesting to know that during the early part of his reign many members of the cabinet were medical men. It is evident, therefore, that to the study of medicine the introduction of modern civilization into Japan must be attributed, and I am very glad to be here with you at this international Congress of Medicine, to which science our nation is so much indebted.

Considering the short period which has elapsed since the adoption of modern medicine in Japan, the progress we have made has been very satisfactory, and not a few of my countrymen have enriched medical science by their discoveries and achievements. Amongst these scientists may be mentioned Professors Kitasato, Shiga, Takaki, Hashimoto, Aoyama, Kosaka, Noguchi, and last, though not the least of all, Professor Hata, who is here as one of our delegates.

I take this opportunity to express the earnest hope that the time is not far distant when your Congress will hold its sessions in Japan.

In conclusion I wish to express our hearty congratulations for this successful opening of your Congress.

Mexico—Dr. Larumbé spoke.

Monaco—Dr. Caillaud spoke as follows :

Altesse Royale, Mesdames, Messieurs, — J'ai l'honneur d'apporter à ce magnifique Congrès le salut et les vœux de Son Altesse Sérénissime le Prince Albert de Monaco et de vous exprimer quel vif intérêt Son Altesse prend à vos travaux.

En pourrait-il être autrement d'un prince qui a consacré sa vie à la science et surtout à une science ayant par certains côtés tant d'affinités avec la médecine ?

L'océanographie fouillant les abîmes pour leur arracher le secret de la vie, révélant à nos yeux émerveillés le prodigieux travail par lequel la nature a fait apparaître la vie dans une variété infinie de types et une fantasmagorie inouïe de formes, n'est-elle pas l'auxiliaire indispensable de la médecine qui étudie la vie chez l'être où elle se manifeste dans sa forme la plus parfaite ?

C'est un auxiliaire, certes, précieux et qui a rendu déjà bien des services.

Qu'il me soit permis seulement d'en rappeler un des plus récents : la découverte de l'anaphylaxie qui dérive de l'étude des actinies.

Je n'accomplirais, Messieurs, que la moitié de ma tâche si je ne vous disais que l'intérêt apporté par le Prince Albert à vos travaux comme savant se double d'une sympathie venant du penseur et du philosophe.

L'étude des sciences biologiques prépare naturellement l'esprit aux conceptions philosophiques saines et justes.

Le Prince Albert qui a toujours fait ses efforts pour propager les idées de paix entre les peuples, de solidarité entre les individus, sait que nulle part ces idées n'auront plus d'écho que parmi les médecins.

Ces idées, qui sont les principes d'une sorte d'hygiène morale pour les sociétés et les peuples, ne sommes-nous pas amenés de plus en plus à les défendre et à les propager comme corollaires pour ainsi dire des principes d'hygiène physique pour lesquels nous avons commencé la croisade.

Ainsi notre champ d'action s'étend sans cesse, notre horizon s'élargit et dans la marche incessante de l'humanité il me semble que de plus en plus nous deviendrons les pionniers intrépides qui la dirigeront vers le progrès.

Altesse, c'est pour moi un précieux honneur de pouvoir, comme représentant de Son Altesse Sérénissime le Prince Albert, remercier votre patrie de la belle hospitalité qu'elle donne au Congrès.

A cet honneur s'ajoute un grand plaisir, celui que j'ai comme Français d'adresser ici mon cordial salut à la grande nation amie de la France.

NICARAGUA—Dr. Alejandro César spoke.

NORWAY—Professor Uchermann spoke as follows :

Your Royal Highness, Ladies, and Gentlemen,—In the name of the Norwegian Government I have the honour to greet the assembled Congress, wishing it all success to the great and scientific work in which all the world is interested and partakes.

PORTUGAL—Professor Luiz de Freitas Viegas spoke as follows :

C'est au nom du Portugal que j'ai le bonheur et le grand honneur de déposer respectueusement aux pieds de Votre Altesse Royale, noble représentant de Sa Majesté le Roi d'Angleterre, les hommages respectueux du vieux Portugal et des médecins portugais. Depuis des siècles la grande nation britannique et ma chère patrie ont une alliance qui n'a pas été seulement une alliance diplomatique mais aussi une alliance des cœurs. Nos soldats ont versé leur sang à côté des soldats anglais et sous les ordres du duc de Wellington ont gagné de brillantes batailles. Aujourd'hui, dans la paix, l'alliance des cœurs se manifeste encore parce que nous suivons avec la plus vive sympathie les progrès éblouissants de ce grand pays dans toutes les manifestations de l'activité humaine. Et je suis heureux d'apporter au dix-septième Congrès de Médecine les vœux les plus sincères de la nation et des universités portugaises pour qu'il soit plus brillant encore que ses prédécesseurs en bénéfice de la science et de l'humanité.

RUSSIA—General Dr. Raptchewski spoke as follows :

Votre Altesse royale, Monsieur le Président, Mesdames, Messieurs, —En qualité de délégué du Gouvernement impérial de la Russie j'ai l'honneur de vous présenter nos remerciements de la gracieuse invitation à participer aux travaux du dix-septième Congrès international de Médecine.

Ce n'est point la première fois que sur le sol très hospitalier de l'Angleterre se tient un Congrès international, ayant pour but le progrès de la science médicale et nous sommes les témoins que l'Angleterre ne cesse jamais de travailler à l'avancement de la science et au bien de l'humanité. Sa Majesté royale le Roi George V a daigné de prendre sous son auguste patronage les travaux de ce Congrès, la participation précieuse du Gouvernement et les efforts de plusieurs illustres savants anglais pour l'organisation de ce Congrès, la participation de plusieurs savants éminents du monde entier — tout cela fait naître la vive confiance que les travaux du Congrès seront très féconds et qu'ils porteront des fruits abondants pour la science et pour le bien de l'humanité.

C'est dans ce sentiment que la Russie est très heureuse d'apporter à cette assemblée avec le concours de son dévouement le tribut de ses vœux cordiaux.

SERVIA—Dr. Subbotitch spoke.

SPAIN—Professor Recasens spoke as follows :

Your Royal Highness, Mr. President, Ladies, and Gentlemen,—The Government of His Majesty the King of Spain, Don Alfonso XIII, in accepting your generous invitation has honoured me, together with Dr. Calleja, with its representation at this seventeenth Congress which is now being held, and which unites the work for the progress of science with the sentiments of universal brotherhood, constituting foundation-stones for a happy era of uninterrupted peace.

The community of ideas, feelings, and affections which is established between the different countries contributes more than the power of nations to sustain peace, leading one to admire the most learned, the hardest workers, and those whose sentiments are of the highest.

Even if no other benefit is obtained from these assemblies, the binding close for ever of the feelings of affection, between the peoples of different countries, speaking different languages, is more than sufficient recompense for our united efforts. The work done at these meetings is always of importance, and after each one marvellous progress is noticed which redounds to the collective benefit of mankind.

Spain has sent to this Congress many representatives of scientific bodies ; she does not rest content with the splendours of her past, but waking from the state in which her continued wars had plunged her, endeavours to place herself on a level with those who are working for the progress of science and the benefit of humanity. The names of Cajal, Cardenal, Rubio, Castro, &c., were honoured by the membership of the British Medical Association, and a great many other names figure in the medical societies of other nations.

The feeling of admiration towards the English people which is felt in my country has considerably increased of late, since a beautiful English princess, even still more virtuous than beautiful, shares with our august king the hardships of the throne. Their majesties, enthusiasts in everything that concerns progress, as well as the whole Spanish nation, send through me their best wishes for the success of this Congress.

SWEDEN—Professor Essen-Möller spoke as follows :

As the Swedish representative I am very glad to bring to you the hearty greetings of my Swedish colleagues. In Sweden we feel strong sympathy for Britain, due not only to our common indomitable love of liberty, but to the fact that we are of the same tribe and the same blood. I express my best wishes for this Congress, and hope that it will give progress to science and relief of pain, both in accordance to noble British traditions.

SWITZERLAND—Professor Dr. Sahli spoke as follows :

Your Royal Highness, Mr. President, Ladies, and Gentlemen,—In the name of Switzerland and its medical profession I bring to this international Congress all our wishes for fruitful work leading to the advancement of knowledge and increasing power in combating diseases.

We are especially happy that this Congress takes place in the wonderful English capital. The bands of friendship which exist between the English and the Swiss people have their roots not solely in the international

situation of Switzerland and in the daily intercourse of travellers visiting our beautiful country, but also, for our part, in the profound sympathy with the English spirit and the admiration of the great British nation and its achievements in the struggle for the freedom and progress of humanity.

In this sense I bring you from the city of Albrecht v. Haller the greetings of our Swiss mountains.

The President of the Permanent Commission of the International Congresses of Medicine, Professor Waldeyer, spoke as follows :

Altesse royale, Monsieur le Président, Messieurs et Mesdames, — Permettez que je prenne la parole au nom de la Commission permanente des Congrès internationaux de Médecine comme président ' ad interim ' de ladite Commission et que je m'exprime dans sa langue officielle pour présenter les salutations et les félicitations au dix-septième Congrès de Londres.

C'est pour la première fois que la Commission permanente exerce ses fonctions et c'est pour cela que je vais dire quelques mots sur ces fonctions. La Commission permanente représente, on peut le dire, un point central entre les divers Congrès internationaux médicaux qui changent toutes les années le siège de leurs assemblées et les personnes dirigeantes. Elle prépare les propositions des lieux et des temps pour éviter des collisions, elle prévoit les thèmes quelles auront à discuter et la distribution des prix, etc. Le bureau de la Commission est à la Haye, en Hollande, et son secrétaire général est le Professeur Dr. Burger, d'Amsterdam, que je tiens à remercier vivement pour tous les soins et les travaux qu'il a entrepris pour la Commission.

But—and I shall say the following in the English language—we must remember to-day Dr. Frederick Pavy, the first President of this Commission, who devoted so much labour and time for the organization of the Commission. We all regret, surely, that Dr. Pavy could not occupy this place to-day. Honour for ever to this valuable and highly-estimated man !

Nun bitte ich, mit wenigen Worten in meiner Muttersprache schliessen zu dürfen, die in einen Wunsch ausklingen : Möge die Einrichtung der ' Dauerkommission ' den internationalen medizinischen Kongressen zu wirklicher Förderung gereichen, vornehmlich nach der Seite der Erfüllung ihrer ernsten wissenschaftlichen und praktischen Aufgaben hin ! Dass ihre erste Wirksamkeit auf London fällt, nehme ich als ein gutes Omen. London, die altehrwürdige und zugleich immer junge Stadt, in der ein frisches unvergängliches Leben pulsiert, London, welches der medizinischen Kunst und Wissenschaft sowohl an schaffenden Persönlichkeiten wie an Einrichtungen so viel Grosses und Bahnbrechendes gegeben hat, wird sicherlich der neuen Organisation einen Teil dieses seines frischen Lebens als Patengabe in die Wiege legen.

The President of the International Association of the Medical Press, M. Lucas-Championnière, spoke as follows :—

Au nom de la presse médicale je viens remercier les organisateurs du Congrès de leur bon accueil et de la place qu'ils nous ont réservée et leur affirmer notre entier dévouement à leur œuvre.

Le rôle des journalistes ne doit pas se confondre avec celui des orateurs. Il n'en est pas moins important et les organisateurs des Congrès internationaux l'ont bien compris en nous faisant place dans cette séance solennelle.

Sans la presse, sans son activité illimitée, les manifestations orales d'un Congrès resteraient trop limitées. L'effort énorme représenté ici n'aurait pas sa juste conclusion. Notre Association a fait tout ce qui était en son pouvoir pour se préparer aux comptes rendus sincères et complets. Les organisateurs du Congrès nous ont fait la place voulue pour réunir les documents nécessaires. Nous nous permettons de faire appel à la bonne volonté de tous les orateurs. Il est de leur intérêt comme il est de celui de la presse que tous les documents nous soient remis en résumé succinct et complet et le plus tôt possible pour que les communications puissent avoir, par notre entremise, une diffusion au dehors qui ne sera que la juste suite des travaux importants dont nous avons déjà annoncé la venue.

His Royal Highness then closed the meeting, and the procession, having reformed in reverse order, passed out of the hall a few minutes before one o'clock.

GENERAL ADDRESS IN

MEDICINE

Par M. le Professeur A. CHAUFFARD, Paris

LE PRONOSTIC MÉDICAL, SES MÉTHODES, SON ÉVOLUTION, SES LIMITES

Monsieur le Président, Mesdames, Messieurs,

En m'invitant à prendre aujourd'hui la parole dans cette grande assemblée médicale, le Bureau du XVII[e] Congrès International de Médecine m'a fait un honneur insigne dont je tiens, tout d'abord, à lui exprimer mes plus sincères remerciements. Je me sentirais très indigne de cette haute distinction si je ne pensais qu'elle s'adresse beaucoup moins à ma personnalité qu'à la science médicale française. Les médecins de France sont venus très nombreux à ce Congrès, désireux de s'instruire, de faire connaître leurs travaux, et aussi d'entretenir et resserrer les liens d'amitié ou d'estime qui les unissent à leurs confrères de tous les pays, et en particulier au corps médical de la grande nation qui les reçoit si brillamment aujourd'hui. Puisque, pour un instant, il m'est donné d'être leur porte-parole devant vous, permettez-moi d'aborder immédiatement le sujet que j'aurai l'honneur de traiter dans cette conférence.

La question que je me propose d'examiner est celle du *pronostic médical*, envisagé dans ses méthodes, dans son évolution, dans ses limites.

Le pronostic médical ! n'est-ce pas pour nous le problème de tous les jours, l'interrogation angoissante qui sans cesse nous est adressée et que nous nous adressons à nous-mêmes ? Depuis qu'il y a des malades et des hommes qui essaient de les soulager et de leur rendre la santé, cette même question se pose dans chaque cas : la guérison sera-t-elle obtenue, et par quels moyens thérapeutiques ? Le pronostic, le traitement, voilà, dès les origines, les deux besoins instinctifs, les deux demandes immédiates de l'homme souffrant. Ce n'est que plus tard, et à un degré déjà plus avancé de culture médicale, qu'est apparue la nécessité préalable du diagnostic, c'est-à-dire d'une notion d'ordre purement intellectuel et logique. Mais s'il a été la dernière de nos acquisitions, le diagnostic a pris aujourd'hui, dans nos recherches, la place qui lui revient, la première, et quel est le médecin qui, sans un diagnostic précis, pourrait formuler un pronostic ou prescrire un traitement ? C'est une idée toute moderne, mais dont nous devons aujourd'hui être pénétrés, que celle de définir, dans chaque cas particulier, les rapports réciproques de ces trois éléments fondamentaux du jugement médical : savoir,

prévoir, agir. Instinctif dans les âges lointains, puis longtemps hésitant et cherchant ses voies, l'acte médical s'efforce de plus en plus à devenir raisonné et méthodique. C'est le passage de l'art à la science, et nulle époque plus que la nôtre n'a essayé, et souvent avec succès, de le réaliser.

Dans cette évolution ininterrompue, le pronostic médical n'est pas resté immobile ; presque sous nos yeux, et par le fait même des progrès de la science contemporaine, il s'est peu à peu transformé ; ses méthodes se sont modifiées, ses limites se déplacent chaque jour, il est dans un perpétuel devenir. L'heure est peut-être favorable pour *marquer l'étape*, pour établir le bilan des acquisitions et des lacunes. C'est ce que je voudrais essayer de faire devant vous.

Dans l'histoire du pronostic médical, une grand place doit être faite tout d'abord à *l'œuvre hippocratique*, et on ne peut relire ces textes vénérables sans un profond sentiment d'admiration. Sans doute, le médecin, dépourvu de toute méthode technique d'exploration, n'avait alors qu'une vision purement objective du malade, mais combien cette vision était pénétrante, habile à saisir, à coordonner, à interpréter les moindres détails ! Quelle étude minutieuse, quelle recherche constante des signes pronostiques tirés de l'état général, et révélés par le facies, par la position dans le lit, le mouvement des mains, la respiration, les sueurs, l'état des hypochondres, les hydropisies, le sommeil, les selles, les urines, les vomissements, l'expectoration. Le livre sur *le Pronostic* ne vise que les maladies aiguës fébriles, mais rien n'est à retrancher dans ses descriptions.

' Le meilleur médecin me paraît être celui qui sait connaître d'avance,' ainsi débutent ces pages admirables, si pleines d'observation clinique et de philosophie médicale. Toujours considérer le corps dans son ensemble, et ' juger toutes choses par l'étude des signes et par la comparaison de leur valeur réciproque '. Et c'est encore les mêmes idées que soutenait Platon dans le *Charmide*, quand il disait : ' qu'on ne peut guérir la partie sans le tout.'

Toute l'œuvre hippocratique est pénétrée de ces mêmes principes, et la fameuse doctrine *des crises* n'est qu'une systématisation du pronostic dans les maladies aiguës.

Ainsi le pronostic est envisagé comme une *vue d'ensemble de la maladie* et de l'évolution probable qu'elle suivra. D'emblée, la médecine hippocratique s'était placée au point central, et avait tenté la synthèse du pronostic clinique.

Voilà les sources premières, celles auxquelles il faut toujours remonter. Ces notions primordiales n'ont rien perdu de leur valeur, et, si nous leur avons beaucoup ajouté, nous n'en avons rien retranché. Aujourd'hui encore, toutes ces données objectives sont de celles qu'aucun clinicien n'a le droit de négliger, et l'impression d'ensemble qu'elles nous suggèrent au lit d'un malade nous dicte le même pronostic qu'autrefois.

Pendant plus de vingt siècles, l'observation médicale s'en est tenue

là, réduite à ces mêmes ressources élémentaires, jusqu'au jour où des méthodes nouvelles se sont ajoutées à la pratique ancienne, et sont venues permettre l'analyse des troubles morbides. Dès lors, c'est par *voie analytique*, et à l'aide de méthodes de plus en plus pénétrantes, que va progresser l'enquête médicale, en même temps que le pronostic s'ouvrira des voies plus directes.

L'époque moderne commence avec Avenbrugger et Corvisart pour la percussion, avec Laënnec, l'immortel inventeur de l'auscultation, avec Richard Bright, le créateur de la pathologie rénale. D'emblée, et par ces mémorables découvertes, toute la médecine se trouve transformée, l'exploration directe des organes sains et malades devient réalisable, et, suivant l'expression de Laënnec, on tâche ' de mettre, sous le rapport du diagnostic, les lésions organiques internes sur la même ligne que les maladies chirurgicales '. Reconnaître les lésions sur le vivant, en préciser le degré et l'évolution, c'était à la fois créer le diagnostic moderne et donner au pronostic des bases plus certaines. C'était aussi montrer toute l'importance d'une science nouvelle, *l'anatomie pathologique*, qui, par ses descriptions précises, allait corroborer et contrôler l'enquête clinique, et orienter pour longtemps la médecine vers la recherche exclusive des lésions et des états organiques. Définir la lésion sur le vivant, la retrouver sur le cadavre, en déduire le diagnostic et le pronostic, tel était le programme de ce qui a été pendant plus d'un demi siècle *la médecine organicienne* ; beau programme, et qui contenait une grande part de vérité, mais doctrine trop étroite, et qui tendait à confondre la lésion avec la maladie dont elle n'est que le résultat. Sans doute, et jusqu'à un certain point, la lésion est la maladie en évolution ; mais l'anatomie cadavérique n'en apporte que la conclusion, la statique terminale. Le *pronostic anatomique* trouve donc très vite ses limites ; c'est peut-être dans le domaine de la neuro-pathologie qu'il a fait ses plus belles conquêtes, et toute l'œuvre de Charcot repose sur l'application raisonnée de la méthode anatomo-clinique.

Ai-je besoin d'ajouter que, si nous ne sommes plus aujourd'hui des organiciens purs, nous n'avons cependant rien sacrifié ni rien oublié du labeur de nos prédécesseurs ? Leur œuvre se superpose au terrain antique, comme une nouvelle stratification géologique sur laquelle s'édifient nos constructions modernes, d'autant plus étendues et plus solides qu'elles reposent sur de plus larges et plus profondes fondations.

L'étape suivante devait être franchie sous l'influence des travaux mémorables de Claude Bernard. Avec lui, la pathologie devient une physiologie déviée, et la maladie un trouble des fonctions normales. Nous ne pouvons juger d'un état morbide, apprécier son évolution probable et les risques qu'il fait courir à l'organisme que si nous connaissons les *troubles fonctionnels* qu'il suscite. Comment, en dehors de cette notion capitale, se représenter d'une façon scientifique le pronostic

d'une lésion du cœur, du foie, des reins, des organes digestifs ou respiratoires ?

Tout notre effort moderne tend chaque jour à entrer plus avant dans cette voie ouverte par Claude Bernard. Toute notre instrumentation de plus en plus perfectionnée, toutes nos techniques de laboratoire, toutes nos recherches sur le sang, sur les humeurs, sur les réactions cytologiques, ne tendent qu'à un même but, l'appréciation délicate de la valeur fonctionnelle des organes, des réactions organiques de défense, des suppléances et compensations, des synergies morbides. Les médecins s'efforcent de penser et d'agir en physiologistes, ou mieux encore *en biologistes*. Notre analyse, empruntant à la fois les méthodes aujourd'hui intimement associées de la clinique pure et de la pathologie expérimentale, essaie de dissocier l'infinie complexité des mécanismes pathogéniques, l'enchaînement des actions et des réactions secondaires, et nous ne pouvons, sur un cas donné, porter une conclusion pronostique qu'en tenant compte de tous ces facteurs.

Et cela fait, après ce long détour, après tout ce travail analytique, nous en revenons encore comme aux premiers âges de la médecine, au pronostic hippocratique, à l'appréciation synthétique et globale de ce que nos anciens appelaient l'*état des forces*. Qu'on se place au point de vue historique, ou qu'on n'envisage que le côté pratique des choses, nous voyons ainsi notre enquête pronostique procéder par les étapes successives de la lésion, du trouble fonctionnel, des adultérations chimiques, pour aboutir au jugement d'ordre dynamique et vital. Comment apprécier le pronostic d'une cardiopathie sans tenir compte de la valeur fonctionnelle du myocarde, d'une maladie hépatique en dehors de la fonction uréogénique du foie, d'une néphrite sans connaître le degré de perméabilité du rein et le taux de l'urée sérique ? Que saurions-nous de l'avenir d'un diabétique si nous n'établissions le bilan de ses échanges nutritifs, si nous ne décelions les premiers indices de l'intoxication acide qui le menace ? D'un goutteux, si nous ne connaissions ni sa tension artérielle, ni son fonctionnement rénal ? Si le diagnostic se bornait, comme le voulait Pinel, ' une maladie étant donnée, à trouver sa place dans un cadre nosologique,' il ne serait que le plus stérile et le plus conventionnel des efforts. Nous le concevons autrement, comme la représentation aussi parfaite et précise que nous le permettent nos moyens d'étude, d'un état pathologique de l'organisme, envisagé dans sa genèse, dans son équilibre actuel, dans son évolution future. Le diagnostic complet se confond presque avec le pronostic scientifique, ou tout au moins ce sont deux manières très voisines d'envisager les mêmes faits. Qu'on se rappelle tout ce que nous devons à l'hématologie contemporaine, à la chimie biologique, à l'électrologie, à la radiologie, et on verra combien sont inséparables les notions diagnostiques et pronostiques ainsi obtenues. Nos courbes de température, d'urines, de poids, nos graphiques cardio-vasculaires tels que nous ont appris à les recueillir et à les interpréter Marey et James Mackenzie, la recherche dans le sang des pouvoirs bac-

tériciides, des agglutinines, des opsonines, le dosage et le rapport des multiples éléments chimiques du sérum, tout cela importe à un égal degré pour le diagnostic et pour le pronostic. Toute l'histoire des leucocytes du sang, avec leurs réactions chimiotactiques et phagocytaires, nous conduirait encore à cette même conclusion.

Le pronostic prend ainsi sa part immédiate de toutes les conquêtes techniques du diagnostic, et, à la faveur de ces progrès incessants, il semble que son champ d'extension soit presque infini. Nous sommes loin, à coup sûr, d'en avoir atteint les limites futures, quoique celles d'aujourd'hui nous enserrent de toutes parts. Nous essaierons plus tard de les définir.

Le pronostic médical est donc chose très complexe et qui comporte la mise en œuvre de toute une série d'éléments d'appréciation. Et cependant, ainsi compris, il resterait très incomplet, n'envisageant que l'heure présente, et ne tenant pas un compte suffisant de l'avenir.

Or, l'avenir, c'est, pour nous médecins, une préoccupation inséparable de la maladie actuelle que nous avons à soigner. Il est vrai que c'est là une idée encore récente, et que, bien longtemps, on a cru que la maladie guérie n'avait pas de lendemains. Quelle était notre erreur, et combien nous sommes loin de pareilles illusions ! C'est toute la question du *pronostic éloigné* qui, chaque jour, se pose devant nous, et on peut dire qu'elle est entrée dans la science avec la découverte de l'endocardite rhumatismale par Bouillaud. Sans doute, le clinicien français avait eu déjà plus d'un précurseur, et c'est d'Angleterre qu'étaient partis les premiers travaux. David Pitcairn, médecin de Saint-Bartholomew's Hospital, observe le premier que le rhumatisme articulaire aigu est une cause fréquente des maladies du cœur, et il l'enseigne à Matthew Baillie, un grand anatomopathologiste qui, suivant l'expression de Sir Dyce Duckworth, mérite d'être appelé ' le Morgagni anglais de son temps '. Avec Wells, David Dundas, les documents se précisent, en même temps que, à Genève, Odier et Matthey arrivent à des conclusions analogues. Enfin, en 1818, James Johnson affirme et décrit la métastase du rhumatisme sur le cœur.

Quand Bouillaud intervient à son tour, il le fait mieux armé que ses prédécesseurs, et c'est à la fois par l'auscultation méthodique de ses malades et par les constatations nécroscopiques qu'il découvre et décrit l'endocardite rhumatismale. Dès ce jour, le pronostic du rhumatisme articulaire aigu change du tout au tout, et à côté, ou plutôt *au-dessus* du pronostic immédiat de la maladie aiguë, se place le pronostic éloigné. Ce qui nous importe, quand nous soignons un rhumatisant, c'est, avant tout, de savoir si son cœur est touché ou reste indemne. Au delà de la maladie présente, nous redoutons la valvulite possible, nous cherchons à la prévenir ou à en enrayer les progrès ; le pronostic de demain prime le pronostic d'aujourd'hui.

Cet exemple classique des cardiopathies chroniques, ayant pour

origine un rhumatisme articulaire aigu, et évoluant ensuite pendant de longues années comme des maladies devenues autonomes, avec leur enchaînement si spécial de troubles fonctionnels et de lésions, trouve plus ou moins ses analogues dans tous les chapitres de la pathologie infectieuse. Les découvertes Pastoriennes nous ont apporté une théorie générale des infections, nous ont démontré le rôle prépondérant des toxines microbiennes, et la multiplicité des affections viscérales aiguës ou chroniques que la clinique, aussi bien que la pathologie expérimentale, réalise chaque jour. Combien de malades paraissent guéris de leur infection aiguë, alors que leur santé tout entière reste atteinte, et peut-être pour jamais ! Reins de scarlatineux ou de pneumoniques, système artériel de typhiques, lésions post-infectieuses des glandes endocrines, troubles trophiques et arrêts de développement dans la paralysie infantile, les exemples sont innombrables que l'on pourrait citer. Que de fois, à la guérison apparente obtenue, succède une longue évolution pathologique dont nos efforts n'arrêteront pas toujours le progrès.

Si, des infections aiguës, nous passons aux infections chroniques, la question du pronostic éloigné devient encore plus pressante.

Voici un sujet atteint de syphilis récente : pouvons-nous penser que quand nous aurons guéri ses manifestations secondaires tout sera fini, et que notre rôle sera terminé ? Nous le croyons si peu que, sur ce sujet qui paraît en parfaite santé, nous multiplions nos séries de traitements, nous interrogeons méthodiquement la réaction de Wassermann, nous sommes toujours hantés par la crainte d'un tertiarisme tardif, ou d'une syphilose vasculaire sous forme d'anévrisme, ou d'une de ces terribles localisations cérébro-spinales, qui constituent le chapitre le plus sombre de l'histoire de la syphilis. Nous en sommes même arrivés à nous demander si nombre de lésions, banales en apparence, ne relèvent pas, en tout ou en partie, de syphilis lointaines, et, pour ma part, je suis convaincu que tel est bien souvent le cas pour l'artério-sclérose, pour les lésions chroniques du foie, des reins ou du myocarde.

Ce que nous venons de dire de la syphilis n'est pas moins vrai de la tuberculose, et ces deux grandes infections se partagent les plus vastes domaines de la pathologie.

La tuberculose, comme la syphilis, a ses échéances tardives ; inoculée dans l'enfance, elle se réveille dans l'âge adulte, parfois même chez le vieillard, et souvent ce n'est qu'après de longues années que se dévoile la gravité réelle d'une adénopathie oubliée ou d'une pleurésie guérie.

Bien plus, à côté des tuberculoses typiques, caséeuses ou folliculaires, nous avons appris à connaître la longue série des lésions tuberculeuses atypiques ou inflammatoires, et capables de faire leurs preuves par l'inoculation expérimentale. On a poussé si loin cette généralisation pathogénique que, peut-être, on en a un peu abusé, et que la majeure partie de la pathologie finirait par se confondre avec l'histoire lointaine et déformée de la syphilis et de la tuberculose. Je crois que ce serait trop dire, mais il n'en reste pas moins vrai qu'en matière de tuberculose

et de syphilis, on sait bien où *commence* le pronostic, mais on n'en aperçoit plus les limites ; aucun médecin ne pourrait affirmer à un malade une guérison absolue et qui le mette à l'abri de toute échéance tardive, qu'elle soit nettement spécifique ou de caractère atypique.

Mêmes observations pourraient être faites à propos de la goutte, du saturnisme, de l'alcoolisme, etc. L'accident aigu disparaît, les suites lointaines sont toujours possibles.

On pourrait presque dire que *rien ne s'oublie dans l'organisme*, et que toute adultération d'humeur ou de tissu prolonge les traces de son passage. La découverte de l'anaphylaxie, par Charles Richet, n'en donne-t-elle pas la plus étonnante des preuves, et ne peut-on pas dire, avec ce savant, que ' nous avons une *personnalité humorale* qui nous rend différents de tous les autres ; et cette personnalité de nos humeurs est due précisément aux ingestions et aux intoxications multiples qui ont affecté notre organisme en laissant une trace indélébile '.

Pour qui est pénétré de ces idées, le pronostic des états morbides prend une singulière ampleur ; la vie pathologique d'un sujet n'apparaît plus comme une suite discontinue d'épisodes successifs, mais on essaie d'en reconstituer l'unité, d'en démêler la trame ininterrompue, tout en faisant la part des déviations brusques et parfois définitives, des orientations pathologiques nouvelles que peut créer une maladie intercurrente.

Sur ces points, comme sur bien d'autres, la clinique moderne rejoint et complète les données traditionnelles, et les *personnalités humorales* de Charles Richet ne semblent-elles pas proches parentes des antiques *diathèses*, dont plus d'une fois récemment Sir Dyce Duckworth s'est fait le défenseur convaincu et éloquent ?

Étendons encore un peu plus loin le champ de notre vision médicale, et au delà de l'individu nous ferons entrer dans notre pronostic l'avenir de la *descendance familiale*. Je n'insisterai pas sur ce côté, pourtant capital, de la question, mais il est évident que la possibilité de l'hérédo-syphilis, de l'hérédo-tuberculose, que la transmission des maladies humorales, telles que l'hémophilie ou l'ictère hémolytique congénital, de tares familiales comme celles dont la neuropathologie nous offre de nombreux exemples, aggrave singulièrement le pronostic de chacun de ces états morbides, en le faisant entrer dans le cadre des hérédités pathologiques.

Ce n'est pas tout encore, et, à côté du pronostic envisagé chez l'individu et dans sa descendance, il faut faire une place au *pronostic collectif*.

C'est là une question d'ordre pratique, toute récente encore, et qu'ont introduite dans la science les recherches contemporaines sur les *porteurs de bacilles*.

Voici une école dans laquelle éclatent un ou plusieurs cas de diphtérie. Ces cas resteront-ils isolés, ou deviendront-ils le point de départ d'une petite épidémie locale ?

Les mesures prophylactiques à prendre dépendront en partie du jugement que nous porterons, et c'est l'examen direct des autres enfants de l'école qui nous renseignera. Nous chercherons s'il en est parmi eux qui portent dans leurs fosses nasales, dans leur rhinopharynx, ou sur leurs amygdales, le bacille de Lœffler. Le nombre des porteurs de bacilles nous donnera de précieux éléments de précision.

C'est lui encore que nous interrogerons si, dans une unité militaire, une compagnie, un escadron, apparaît brusquement un de ces cas, souvent si rapidement mortels, de méningite cérébro-spinale. Dans ces dernières années, nous avons eu en France de trop nombreuses occasions de voir naître et évoluer ces foyers locaux de méningococcie, et nos confrères de l'armée sont pleinement avertis de la ligne de conduite à tenir ; dans les faits de ce genre, la recherche méthodique des porteurs de méningocoques est immédiatement pratiquée, et montre très vite si l'on a affaire à des cas sporadiques ou à un début probable d'épidémie plus ou moins grave. C'est donc un *pronostic d'épidémie* que nous établissons, et qui peut, jusqu'à un certain point, rester indépendant des pronostics individuels.

Enfin, plus haut que l'individu, plus haut que la famille, plus haut que les collectivités partielles, il y a *la race*, il y a l'ensemble de l'espèce humaine.

Envisager comme *maladies sociales* la tuberculose, la syphilis, l'alcoolisme, en rechercher les causes d'aggravation et de décroissance, n'est-ce pas atteindre au point le plus élevé où puisse monter le pronostic médical ? Combien cela nous éloigne de nos anciennes traditions où, quand le médecin avait soigné son malade, il croyait avoir tout fait et rempli tout son devoir ! Nous savons bien aujourd'hui qu'il n'en est rien, et que souvent le cas particulier ne peut ni ne doit être séparé de ses multiples connexions humaines.

Toutes nos mesures légales de désinfection, de vaccination préventive, trouvent dans ce pronostic social des maladies leur justification et les preuves de leur nécessité. Ce n'est pas ici, en Angleterre, patrie de Jenner, et pays des prophylaxies efficaces, que de telles notions ont besoin d'être plus longuement rappelées.

N'est-ce pas également dans cette grande cité de Londres qu'a pris naissance, avec les travaux mémorables de Sir Francis Galton et de son École, cette science nouvelle, l'*Eugénique*, qui, il y a un an, y tenait ses premières assises ? Améliorer les qualités sociales, soit physiques, soit mentales, des futures générations, comme le voulait Galton, faire, suivant l'enseignement de Pinard, de la puériculture avant la procréation, pendant la gestation, après la naissance, il me semble que c'est transposer dans le domaine de la prophylaxie, et pour le plus grand bien de la race, la notion du pronostic collectif et social.

Parti de l'observation quotidienne du malade, le médecin n'a donc cessé de voir plus haut et plus loin ; il comprend mieux, chaque jour, qu'il ne peut se désintéresser ni de l'individu, ni de la famille, ni de la

race ; que, dans un état social où tant de forces néfastes convergent vers la dégénérescence de l'espèce, il doit apporter à tous l'appoint bienfaisant de sa science et de son labeur.

Cette extension progressive du pronostic médical, basée à la fois et sur l'apport des méthodes nouvelles et sur une compréhension plus large des répercussions de la maladie, porte-t-elle sur des faits toujours identiques à eux-mêmes ? Le pronostic d'une maladie donnée reste-t-il immuable, ou pouvons-nous préciser les causes et le sens de ses variations ?

Admettre l'immutabilité du pronostic médical serait déclarer que notre thérapeutique est restée immobile et sans progrès, et, certes, ce n'est pas à notre époque qu'une aussi décourageante opinion pourrait être soutenue. Depuis quarante ans, et sous nos yeux, toute la médecine scientifique et pratique s'est transformée, entraînée dans une évolution incroyablement rapide ; pathogénie, méthodes d'exploration clinique, méthodes thérapeutiques, tout a pris une face nouvelle, et le pronostic de tous nos états morbides s'en est trouvé profondément modifié.

Je crois que l'on peut dire qu'il n'est presque aucun groupe de maladies dont la gravité n'ait subi de nos jours une notable atténuation.

Sans doute, il reste trop de cas où le médecin déplore son impuissance, mais même alors celle-ci n'est plus aussi complète. Le problème du cancer se pose toujours devant nous, mais comment nier que par l'intervention des rayons de Rœntgen ou du radium, par le progrès des techniques chirurgicales, par des essais chimio-thérapiques, nos moyens d'action sont devenus moins restreints et plus souvent efficaces ?

Le pronostic de la lésion peut rester impossible à modifier, et l'hémorragie cérébrale, ou l'embolie pulmonaire, n'ont rien perdu de leur gravité. Mais les lésions du système artériel ou veineux, causes de ces graves accidents, nous sont mieux connues, nous pouvons les prévenir ou les traiter avec plus de succès, et ainsi, même dans ces cas les moins favorables, nous avons cependant gagné du terrain.

Mais combien d'exemples pourrait-on citer, surtout dans le domaine des infections, où notre pronostic d'aujourd'hui n'a plus rien de commun avec celui de nos devanciers ! Nous ne pouvons plus comparer notre rhumatisme articulaire à celui qu'observait Bouillaud, et la médication salicylique en a fait une autre maladie. Depuis Brandt, nos typhiques ne sont plus les mêmes, et l'intervention, actuellement à l'étude, de la bactério-thérapie antityphique nous permet d'espérer un prochain et décisif pas en avant.

Pouvons-nous oublier ce qu'était la diphtérie avant la découverte de l'antitoxine diphtérique par Behring et Kitasato, et l'application thérapeutique éclatante apportée par Roux au Congrès de Budapest ? Les courbes de morbidité et de mortalité ont montré depuis lors, dans l'univers entier, combien était transformé le pronostic de la diphtérie.

Même la plus terrible des maladies, celle qui ne pardonnait jamais, la rage, a trouvé son maître, et la vaccination antirabique de Pasteur

est peut-être la plus étonnante conquête thérapeutique de notre époque.

Ainsi chaque jour amène son progrès, atténue ou modifie telle ou telle de nos appréciations pronostiques, nous apporte de nouveaux moyens d'action.

Armés des méthodes modernes, disposant à la fois et du mercure et de l'arsenic sous les formes les plus énergiquement actives, ne traitons-nous pas nos syphilitiques avec des chances de succès bien plus grandes que par le passé ? Je n'insiste pas, et voudrais seulement rappeler deux autres exemples tout récents et bien typiques.

Voici, dans le groupe des trypanosomiases, la *maladie du sommeil* regardée, au début de son étude, comme incurable. Thomas, puis Nicole et Mesnil montrent expérimentalement l'efficacité de l'atoxyl contre les trypanosomiases animales. Ayres Kopke, de Lisbonne, applique le premier la médication à l'homme, et, depuis lors, les résultats obtenus n'ont cessé de s'améliorer. A l'hôpital Pasteur de Paris, Louis Martin a traité et suivi, dans ces dernières années, 40 cas de maladie du sommeil, et il a bien voulu me communiquer sa statistique. Les 20 premiers cas traités ont donné 11 décès, tandis que sur les 20 suivants on ne compte que 4 décès compris de 20 à 30. De 30 à 40, pas de décès, et cependant deux malades de cette série ont été traités plus de trois ans après le début des accidents. C'est que peu à peu la technique du traitement s'est modifiée, et à mesure qu'aux doses massives et espacées de dix en dix jours Louis Martin a substitué des doses moins fortes et plus rapprochées, les succès sont devenus plus complets et plus durables. Nous pouvons maintenant parler de guérison dans une maladie dont le diagnostic paraissait il y a peu d'années un arrêt de mort.

Prenons, d'autre part, l'exemple des *dysenteries*. Nous savons aujourd'hui que derrière l'unité apparente des symptômes se cache la multiplicité des pathogénies, et nous distinguons deux variétés principales de dysenterie, suivant que l'infection causale est de nature bacillaire ou amibienne. Le progrès thérapeutique n'a pas tardé à consacrer la distinction pathogénique, et à deux infections spécifiques nous opposons deux médications spécifiques. La dysenterie bacillaire est presque immédiatement enrayée et guérie par la sérothérapie antidysentérique telle que l'ont instituée Shiga, Kruse, Todd, Rosenthal, Vaillard et Dopter, pour ne parler que des travaux les premiers en date.

Quant à la dysenterie amibienne, son traitement spécifique tout récent a déjà largement fait ses preuves, et il est dû aux recherches persévérantes d'un médecin anglais, de Léonard Rogers, de Calcutta. Depuis douze ans, Rogers s'est attaché à étudier les effets de la médication par l'ipéca dans la dysenterie amibienne, à montrer son efficacité préventive contre les abcès du foie. Puis, s'inspirant des expériences de Vedder, il substitue à l'ipéca un de ses alcaloïdes, l'émétine employée sous forme de chlorhydrate, et il obtient les plus beaux succès de guérison immédiate et complète dans les formes intestinales et hépatiques de l'amibiase.

Alors que pour des dysenteries de même gravité la mortalité, pour les cas traités par l'ipéca en poudre, était de 34,6 pour 100, elle n'était plus que de 8 pour 100 dans les cas soumis à la cure d'émétine ; avec l'ipéca la durée moyenne de traitement est de 16,4 jours, alors que, pour l'émétine, elle tombe à 7,2 jours.

Pour les abcès dysentériques du foie, traités par les injections d'émétine sous la peau et dans la cavité de l'abcès, les résultats ne sont pas moins favorables, et la guérison s'obtient souvent dans les conditions de bénignité et de promptitude vraiment étonnantes.

Les faits nombreux relatés par Rogers, ceux qui depuis ont été observés aux Philippines et à Paris ne peuvent laisser aucun doute ; c'est bien une nouvelle médication spécifique que nous devons à Rogers, une des plus actives connues, et une telle acquisition est un bienfait inestimable, une magnifique conquête thérapeutique. Voilà encore une maladie, et combien grave ! dont le pronostic se trouve, du jour au lendemain, complètement transformé.

Si, du domaine des infections, nous passons aux autres familles morbides, sans doute les progrès sont moins brillants, et l'avenir semble plus limité. La recherche des troubles du métabolisme nutritif nous conduit à des causes plus obscures et d'action plus lente, moins favorables aux interventions thérapeutiques. Et cependant, là aussi, nos conquêtes modernes ne se comptent plus, et tout le chapitre des opothérapies serait à citer, aussi bien que celui des régimes diététiques et de la physiothérapie. Partout la maladie recule devant les efforts associés de la thérapeutique et de l'hygiène, partout le pronostic évolue et nous ouvre de nouvelles espérances.

Nous venons d'examiner quelques-uns des aspects généraux de cette grande question du pronostic médical, et il semble que les conclusions de notre étude ne peuvent être que favorables. Mais cependant, là comme dans toute connaissance humaine, il y a des limites que nous ne devons pas oublier, et qui sont bien faites pour contenir, s'il y a lieu, notre orgueil médical et le ramener à la modestie qui lui convient.

Quelles sont donc *les limites du pronostic médical* ?

Ici une distinction capitale doit être faite entre le *pronostic théorique* et le *pronostic clinique*.

En théorie, presque tout nous est connu, et, étant donnée une maladie, nous pouvons énumérer et décrire, sans grandes chances d'omission ou d'erreur, la plupart des complications ou éventualités qui peuvent en modifier le cours.

Mais tout cela c'est le pronostic *du livre*, et il reste malheureusement bien loin du pronostic vivant, individuel, appliqué non plus à une maladie mais *à un malade*. Que d'inconnues dans la pratique médicale et que de surprises ! Combien il est insuffisant de connaître toutes les complications de la fièvre typhoïde, si, au lit d'un typhique nous ne pouvons prévoir ni l'hémorragie intestinale, ni la perforation qui, demain peut-

être, mettront sa vie en danger. Et pour le syphilitique au début, pour le goutteux, pour l'épileptique qui a sa première crise, pour tant d'autres encore et de tous genres, que d'incertitudes d'avenir ! Quand un état morbide commence, nous savons théoriquement quelles routes il *peut* prendre ; mais quelles prendra-t-il ? Voilà le vrai problème pronostique, et celui dont trop souvent la solution nous échappe. Sans doute, l'étude du terrain héréditaire et des états propathiques peut nous servir de guide souvent, mais le déterminisme local des fixations septicémiques ou toxinémiques nous reste ignoré. Nul ne peut prévoir, au début d'une pneumonie, l'endocardite ou la méningite à pneumocoques qui deviendra cause de mort.

Une fois la localisation faite, nous ne pouvons pas toujours apprécier le degré et l'évolution de la *lésion anatomique*. Nous ne pouvons dire si, chez le typhique, l'ulcération intestinale creuse et menace de devenir perforante, si chez une malade atteinte de rétrécissement mitral l'infarctus hémoptoïque ou l'embolie sylvienne vont se produire. Et ainsi, dans les maladies les mieux connues, notre pronostic reste incertain, fort en théorie, faible en pratique.

A toutes ces causes de doute et de prudente réserve, il en est bien d'autres que l'on pourrait ajouter, et une, entre autres, d'importance majeure, la *réaction du système nerveux*. Je ne sais quel homme d'état disait qu'en politique il faut toujours compter avec *les impondérables* ; à coup sûr, en pratique médicale, un tel aphorisme ne serait pas moins vrai. Que ce soit sous forme d'inhibition ou de dynamogénie, la cellule nerveuse peut toujours ajouter aux processus morbides sa réaction perturbatrice ; dans les sphères de la motricité, de la sensibilité, des algies viscérales, du psychisme, cette réaction ne se calcule ni ne se maîtrise à volonté ; nous essayons bien de la redresser ou de la diriger, mais il peut nous être impossible d'en prévoir la gravité ou la durée.

Enfin, et dominant toute la question du pronostic, reste l'appréciation synthétique de la *valeur de l'organisme* atteint. Trouverons-nous en lui les ressources nécessaires pour triompher de la maladie, répondra-t-il à nos sollicitations thérapeutiques, que peut-il supporter, et que pouvons-nous lui demander ? Voilà le grand problème, celui qui prime et résume tous les autres. Pouvons-nous toujours le résoudre ? Je laisse à chacun de nous le soin de formuler sa réponse, et j'ai bien peur que celle-ci doive rester très modeste.

Il faut conclure, et je m'excuse, tout en ayant paru peut-être trop long, d'être resté aussi incomplet.

Le pronostic médical forme comme le corollaire et l'application pratique du diagnostic ; de celui-ci il emprunte les méthodes et suit les progrès. Au cours de son évolution moderne, il a pris sa part de toutes les conquêtes de la médecine scientifique ; comme elle, il n'a cessé de marcher dans les voies de l'analyse clinique et de la pathogénie expérimentale, il est devenu de portée plus haute, de vision plus étendue. Il

apparaît comme la synthèse du jugement médical, en même temps qu'il précède, contrôle et justifie les traitements et les prophylaxies. Dans son ascension vers le mieux, dans les atténuations de gravité qu'il nous permet de constater, il montre les étapes parcourues et encourage nos efforts. Sans doute, nombreuses sont encore nos incertitudes ou nos déceptions ; mais puisque la science du pronostic est un des plus hauts sommets de la médecine, nous devons, dans notre marche sans arrêt, essayer d'en entrevoir la cime ; même quand celle-ci nous reste lointaine et cachée dans les nuages, ne désespérons jamais de diminuer la distance qui nous en sépare. Une telle ambition nous est un devoir et sera toujours le meilleur stimulant de notre effort médical.

GENERAL ADDRESS IN SURGERY

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REALIGNMENTS IN GREATER MEDICINE: THEIR EFFECT UPON SURGERY AND THE INFLUENCE OF SURGERY UPON THEM

Mr. President, Ladies, and Gentlemen,—Thirty-two years have elapsed—a full generation—since this Congress, under the distinguished leadership of Sir James Paget, last gathered in London. Among the participants on that occasion were five of the immortals in the history of the medical sciences: Huxley—naval surgeon, zoologist, palæontologist, educator, philosopher, public servant; Virchow—pathologist, anthropologist, archæologist, statesman, and sanitarian; Pasteur, the chemist, Lister, the surgeon, and Koch, the bacteriologist—among the foremost contributors of all time to the welfare of their kind. On the foundation which these men laid—a new conception of biology and man's place in nature; the doctrine of cellular pathology; fermentation and immunity; wound-healing without suppuration; bacterial diseases—a transformed medical edifice has been erected. Methods of investigation, the direct outcome of those which they evolved, we still employ; weapons which they devised are still, and always will be, effective, not only in a hand-to-hand conflict with individual examples of many of the disorders to which man and the animals needful to his existence are exposed, but better still in their prevention.

The London Congress of 1881 and its participants.

A few years before that meeting the profession of medicine, and particularly those concerned with its basic science, physiology, had been called upon in this city to defend themselves against the public charge of cruelty in their pursuit of knowledge. There had been no striking practical demonstration of the value to the common weal of animal experimentation such as an unenlightened public demanded—such, indeed, as an unenlightened profession expected. The defence was based chiefly on the need, even at the cost of animal life, of increased knowledge concerning the functions of the human body; but despite the warnings and protests of Huxley and a few others the restrictive legislation of which you know was passed. Since then in the British Isles, and consequently in other English-speaking countries, medicine has been placed in the absurd

The restrictive legislation of 1876.

position of defending the character of the labours necessary for its advancement.

But all this is changing, and, indeed, began to change soon after that memorable Congress of 1881. Virchow, though his great contributions had come through mortisection studies rather than through observations on the living, gave an elaborate historical review of the value of experimentation to general pathology. Pasteur at one of the sessions presented the results of his work on vaccination against chicken cholera and anthrax ; at another Koch gave demonstrations of his newer bacteriological technique. In the surgical section the question of chief moment concerned primary wound-healing, and, in the emphatic discussions which followed, Lister, who had perforce been required to conduct some of his investigations in France, described his experiments demonstrating the bactericidal properties of blood serum. Between the lines of the printed reports one can easily perceive the coming transformation of antiseptic into aseptic surgery.

The seed of knowledge often finds a better soil when blown or carried to a distance, and in this instance it was across the Channel, where no discouraging blight was put upon the sort of research that is as necessary for the furtherance of surgery as for that of any other subdivision of greater Medicine. For it was on the Continent that the principles established by Lister took firm root and, particularly in Germany, were so ardently cultivated through experimentation and practice that modern surgery bears a more vigorous Teutonic than Gallic or Anglican graft. And is it not due solely to this legislative restraint upon the freedom of investigation that in these thirty years this country, which has continued to produce the greatest of names in the experimental sciences free from statutory restriction, has given us few instead of many notable successors to Hunter and Cheselden, to Cooper and Pott and Brodie, to Bell and Paget and Lister, who contributed to the science no less than to the craft of surgery? It would seem, indeed, that the restrictions placed upon animal experimentation have deterred the physician and surgeon from productive laboratory investigation far more than the physiologist, whose inclinations towards research the statutes of 1876 were expressly designed to hold in check ; for Great Britain's representatives of pure physiology and experimental neurology, despite the hampered conditions under which they work, stand out as the chief contributors of the generation to British medicine, though the public cannot appreciate, nor the profession for the moment fully understand, the significance of their labours.

Its consequences.

The spirit of investigation—all too rare—should be generously subsidized rather than taxed, encouraged rather than hindered. The outresult of the experiments of one participant in that Congress of 1881 is said to have saved enough for France to enable her to pay the heavy indemnity which the outcome of a war had imposed upon her. Nature is loth to give up her secrets ; discoveries do not come by chance ; they are made by those only who are industriously prepared to observe them,

and the inquisitorial methods of those who seek the light through experimentation, misjudged as cruel, are necessarily stern and persistent, whether the investigator deals with inanimate objects or with animate beings. Possibly Pasteur's greatest scientific triumph lay in a discovery in crystallography, but the same method of work was pursued in his studies of fermentation which inaugurated a new era in the brewing and wine-making industries ; in the saving of the silkworm from a destructive parasite, of poultry from cholera, of flocks from anthrax, and in the elaboration of an effective barrier against the most frightful of deaths—that from hydrophobia. From crystals under the microscope, to yeasts, to the silkworm, to chickens and sheep, to man—and when his studies reached man, who was henceforth to be saved from the poisonous bite of his friend and ally, the dog, Pasteur was accused of cruelty to animals. And the name of this simple and loving man, acknowledged the greatest of Frenchmen, but whom France alone cannot claim, is met with hisses when mentioned on certain platforms before gatherings of presumably intelligent people !

The London Congress of 1913 and the legislative inquiry of 1906.

This Congress meets again in London, under circumstances strangely similar to those of a generation ago. Again a legislative inquiry has just been forced upon British medicine by those who would abolish experimentation upon animals. But how different the testimony ! It bared to the public gaze a science of medicine which in thirty years had become transformed throughout the world as a result of the very activities the Commissioners were called to investigate. Medicine no longer, even as a practising profession, is looked upon as uncommunicative, consulting in the back room as though it had something to conceal from patient or family, for in the individual case the family, and in the general case the public, are not only awakened, but are sufficiently interested and informed to come into an open consultation and to share in the struggle against disease. Great funds are devoted to the purpose ; great corporations are held responsible for the health of their people ; formerly uninhabitable zones of the globe are being rid of their plagues ; governments, civic, state and national, not only employ the weapons of attack and defence forged in laboratories whose methods were in question a generation ago, but expect their medical officers to make progressive contributions by further researches.

A transformed medicine.

All these great movements, of which Huxley and Virchow and Pasteur must have had some foresight, are but expressions of the new alignments taking place in the profession of medicine—alignments which affect the surgeon no less than the physician.

Effect on the practice of medicine.

It has been a seeming paradox that the medical profession has so consistently endeavoured to make of the world a place where there is a constantly lessening need for the medical man. He is, indeed, already feeling the effect of these efforts, and as he becomes more and more a servant of the public health, and less and less a prescriber for individual ills, he philosophically

accepts and humanely welcomes this outcome of medical discoveries which the experimental method has already given and will increasingly continue to give.

Disorders which gave bread and butter to his predecessors are disappearing, as small-pox disappeared after Jenner. One injection robs diphtheria of its terrors, another, meningitis. Typhoid fever, which once replenished the doctor's autumnal pocket-book, is now looked upon as a civic disgrace, and where it exists or is likely to break out, protective vaccination is performed against it. Tuberculosis is everywhere fast coming under State care, and the widespread campaign against the disease, in which those outside the profession are taking no insignificant part, is gradually lessening its ravages. The health of children attending the public schools is supervised by appointed officers; district nurses and the social service worker visit the homes of the sick and assume duties which the hospital physician long neglected; a privately endowed Commission takes steps to eradicate hook-worm infection from a whole zone of the globe; an organized Red Cross Society, with Government affiliation, steps in in great emergencies; Governments see fit to pass Insurance Acts which make public officials of the profession at one sweep. Dr. Pound of Cure Lane is being superseded by his young disciple, Dr. Ounce of Prevention Street.

In matters of health the people as a whole have been largely permitted to shift for themselves, and when they have forgotten how large a percentage of the faces of the eighteenth century and before were pock-marked, many of them wish again to exercise their individual discretion regarding vaccination. 'Where there is no vision the people perish.' Governments, though interested in the welfare of the domesticated animals, are too often indifferent to the public health, and only in the face of emergencies, the greatest of which continues to be warfare, are they stirred to action, since freedom from disease is as essential as food and ammunition in the prosecution of a successful campaign. Hence have arisen the most striking examples of the effectiveness of preventive vaccination, at first against small-pox and latterly against typhoid.

Changed duties of the military surgeon.

The effect of the present realignments of medicine of which I speak is particularly apparent in the case of the military surgeon, for the Parés, the Wisemans and the Larreys, the Pirogoffs, the Dupuytrens and the Langenbecks of to-day are occupied with preventive inoculation, with the screening of the sick, the protection of water sources; more concerned with the first-aid package than with the extraction of bullets; more often called upon to use the microscope than the scalpel and ligature. Little wonder that from men of this type great contributions have come. It is a far cry from Guthrie's amputation through the hip-joint at Waterloo to Laveran and the discovery of the malarial plasmodium, or to Walter Reed and the mosquito and yellow fever.

In the search for biological truths the God-given privilege of becoming

an obvious benefactor to his species comes to but few men. No one of the countless inquisitors of nature knows whether the mantle of recognition will fall upon him—or, for the matter of that, especially cares—provided he be given the personal satisfaction of having taken a step forward in knowledge, no matter how trifling at the moment that step may appear to be.

Experimentation and tuberculosis.

To whom is chiefly due the expectancy of recovery for the consumptive? Is it to Leuwenhoek and the miscroscope; to the father of Lister and to Abbé, who perfected its lenses; to René Laënnec and his stethoscope; to Sir William Perkin, whose experiments with the coal-tar by-products furnished the necessary colour reagents; to Villemin, who demonstrated its communicability; to Koch, whose genius made possible the recognition and cultivation of the organism; or to Ernst von Leyden's propaganda in Germany, and in the United States to Trudeau, himself a victim of the disease, through whose precepts and example tuberculosis has been moved into the open, where it is being effectively attacked in a campaign of education shared by layman, legislator, and physician alike? Unquestionably through experimentation on animals the most important step in this progress was made—the discovery of the bacillus tuberculosis and its identification as the causal agent not only in pulmonary consumption but in the many 'strumous' and 'scrofulous' disorders which puzzled our forbears.

Experimentation and syphilis.

Where will lie the chief credit for the approaching victory over the great social scourge which since its outbreak in Columbian times has undermined the vigour of the race? Will it be with those earlier men who gave us the tools of research, the microscope, the culture media, and the colouring dyes; with Wassermann and the possibility of a chemical diagnosis; with Schaudinn, who discovered the spirochæte; with Metchnikoff and the successful transfer of syphilis to the ape; or with Ehrlich, who, by his original methods, has had the triumph of synthetizing a drug possessing a special affinity for the infecting organism but which leaves uninjured the tissues of the infected host? There has been nothing heretofore comparable to this relentless, persistent, certain elaboration of a chemical compound which at a single dose may destroy, within the living tissues, the organism of a dread disease. Before it all past discoveries of therapeutic agents must make obeisance, even Jenner and vaccinia, Pasteur and the attenuated virus, Kitasato and Behring and the elaboration of the antitoxins.

What would the opponents of animal experimentation of 1876 have thought could this have been foreseen, this which is only the beginning, with pneumonia and cancer still to be overcome? That the means do not justify the end? That it is unfair to the lower animals, some species of which with unquestioned cruelty man sacrifices for adornment, for sport, for covering, for food; some of which he deliberately mutilates in

the process of domestication or in preparation for his table; some of which, like the infected rats and squirrels of plague-ridden districts, he must attempt to exterminate for his self-protection? But in the search for knowledge the investigator does not exempt himself as a subject of so-called vivisection when lower animals do not suffice for the purpose;[1] nor will he ever hesitate even to endanger his life, whatever may be the ethics of the question, if thereby information is likely to be gained concerning some disease fatal to his kind.

Experimentation and parasitic diseases.

To whom is due the approaching transformation of the tropics into a possible habitat for civilized man? Again a series of investigators on investigators' shoulders, until Theobald Smith, in the case of the tick-infected cattle decimated by bovine malaria (Texas fever), in 1893 showed for the first time that a parasite might be an essential intermediary host in the transfer of a disease from animal to animal. Promptly all blood-sucking parasites of man became objects of suspicion, and ere long convictions followed—first in the case of filaria by Manson; then Ross's demonstration that the mosquito is also an essential link in the cycle of 'million-murdering' malaria[2]—a discovery which was made possible through his studies of infected birds. But when it became necessary to establish unequivocal proof in the case of man, the younger Manson, and Warren, of the London School of Tropical Medicine, did not hesitate to submit themselves to the bite of infected mosquitoes sent for the purpose to England from the Roman Campagna.

Texas fever.

Malaria.

It is hardly possible for one unfamiliar with the experimental method and its necessary tedious controls to conceive of the patient, unflagging, time-consuming labours on the part of many investigators the world over, often conducted in most unhealthy districts and in the very shadow of death, which step by step, unwavering, led to this final demonstration —labours which in review seem almost beyond the possibility of accomplishment. The discovery of the malarial plasmodium in the blood, the determination of its several varieties, the study of the sexual cycle of its development, the changes in the stomach wall of the mosquito and the lodgment of the sporozoites in the glands of the proboscis, the parti-

[1] Henry Head's important studies on the sensory disturbances produced by a purposeful division of a nerve in his own arm may be given as an example.

[2] These significant lines, too little known, were written by Ronald Ross on the eve of the discovery:

This day relenting God
Hath placed within my hand
A wondrous thing; and God
Be praised. At His command,

Seeking His secret deeds,
With tears and toiling breath,
I find thy cunning seeds,
O million-murdering Death.

I know this little thing
A myriad men will save.
O Death, where is thy sting!
Thy victory, O Grave!

cular species among the many genera of mosquito concerned, the fact that the female alone is the active agent, the finding of the length of time necessary for infectivity—each step, whether by Frenchman, by Italian, by Canadian, by Englishman, or by German, a triumph of the spirit of research! And no great objection is raised if, in the process, the mosquito, bird, and man are the subjects of experimentation.

Yellow fever.

Soon the participation of another species of mosquito in the transmission of a still more dreaded disease was demonstrated in Cuba by the United States Yellow Fever Commission, at which time Carroll, Lazear, and some brave volunteers from the ranks permitted themselves to be bitten by infected parasites—the lamented Lazear dying a victim of the disease he had invited. The imagination of the public is only beginning to respond to these acts of heroism constantly being performed by investigators the world over—performed for humanity's sake, and surely no less but even more heroic than the quickly applauded dangers risked in warfare for the sake of country alone. Far better could our proof of the transfer of yellow fever through the bite of the stegomyia have come through the sacrifice of lower animals, had animals susceptible to the disease been available for the purpose, but to the members of that Commission there seemed no alternative to experimentation upon human volunteers. And what have been the results? Pestilential districts become habitable; commerce with tropical ports, once annually quarantined because of the recurrence of a dread scourge, continues the year round; across an isthmus formerly plague-ridden two oceans are being joined, where under a former régime shovels alone, without drainage and screens, proved utterly unavailing.

And after the cattle-tick and the mosquito, all other biting parasites—the flea and the bed-bug, the fly and the louse—have risen in dignity from a disgrace to a menace.[1] Immediately the patient studies of the insect world by the entomologist, disturbed from his quiet seclusion as a systematist into active participation in medical problems, become of the greatest practical value, and under the wing of preventive medicine his specialty, like that of the helminthologist and protozoologist, becomes as essential to the medical curriculum as was that of the botanist a century ago. These are astonishing changes to have been brought about by the experiments of one modern scientist who had the training, the

[1] Parasitism comes to have a far broader significance in relation to medicine than is covered by bacteriology alone, and from its widespread existence in nature, among plants as well as animals, and from the fact that 'every parasite has near relatives that do not possess the parasitic habit', it may be assumed that these habits are acquired, and that consequently some diseases may be of recent occurrence and that still others may arise anew. Metchnikoff has even suggested that among parasites we may look for the latest products of evolution. It is a far cry in the history of parasitism from the first given example—the misletoe, by Pliny—to a hæmatozoon, one cycle of whose development must take place in the body of a blood-sucking intermediary host.

imagination, and the patience to investigate the ticks of cattle afflicted with a destructive plague, and after four years of laborious study to identify them as essential agents of the disease.

Experimentation and the diseases of animals.

Herein, it seems to me, lies the weakest point in the opposition to experimentation on the basis of cruelty--namely, that the animals whose preservation is desirable benefit from these investigations as greatly as man. There is no more notable example of this than the case of man's companion, the dog. Through the deserved attachment which has grown out of this companionship, a sentiment has arisen which would exempt the canine species from experimentation. But had such a law been put on the statutes, Copeman's discovery of the bacterial cause of distemper, and of a successful method of inoculation against this most fatal and distressing canine disease, would have been impossible; and the same is true of the fatal malignant jaundice—a parasitic blood disease conveyed by the bite of the dog-tick, so prevalent in some parts of the world as to make the rearing of dogs impossible, and for which Nuttall has found an effective remedy and means of prevention. What a credit to the societies for animal welfare could such discoveries have come through their own efforts rather than through the efforts of those whose methods of research they are prone to question!

Societies for animal welfare.

Large sums of money have been wisely devoted to the prevention of cruelty to animals, and much good has been done in the past, but by a strange process of evolution most of those now entrusted with these funds, instead of grasping the opportunity to advance knowledge of the diseases of animals, have devoted their energies to opposing such advance as may be undertaken for the sake of man at some expense to animal life. The future offers a great opportunity for these societies, whose capital is now largely expended, on the one hand, in the wholesale sacrifice of stray and diseased animals, and on the other in destructive criticism of the methods of those they call vivisectionists; for by constructive investigation and by the employment of the same methods which have been elaborated by those whose primary object is the study of the diseases of man, they may become as great benefactors to animals, and incidentally to man, as the medical scientists have been to man, and incidentally to animals.

Veterinary research hospitals.

There could be no better outlet for the present wasteful methods of many of these organizations than the establishment of veterinary hospitals, in which modern methods of treating disease could be employed, further investigations made, and students of veterinary medicine taught—the three great functions of any hospital; and there is promise in some communities that this wise step may be taken.

Though less true of many European countries, where veterinary institutes which are doing admirable progressive work are under govern-

ment control, in close association with universities, in the United States, and I think the same is true in a measure of Great Britain, most veterinarians have profited not at all by the advance in general medical knowledge of the past generation. This applies particularly to veterinary surgeons who still trust, in the operations which they venture to undertake, to the rough and casual methods of old, with scant if any pretence of the modern refinements of skilful anæsthetization and aseptic surgical technique. Little wonder that people who are aware of this difference prefer to have their pets, when in need of surgical care, operated upon in an experimental laboratory rather than in many of the established veterinary hospitals. Some years ago, in the surgical laboratory at Johns Hopkins, this work grew to such proportions that a course covering the technique of operative procedures was offered to a group of veterinarians, who there for the first time learned methods which would enable them to save valuable animal lives by procedures which they had considered impossible, and which for the general run of veterinary surgeons had been impossible.[1]

When himself the victim of an operable malady, man voluntarily submits to a surgical operation, which is nothing other than vivisection, aware that the momentary risk and temporary discomforts—happily far less than they were of old, despite the increasing magnitude of the procedures which are undertaken—are but a small payment for the possible and probable ultimate good.

And most experimental procedures in the laboratory to-day are conducted on the same principles and with the same precautions that are observed in the hospital operating-room, and the rewards of experimentation, largely for these reasons, are greater than ever before. This is one of the great contributions which the surgery of Lister has made to the advancement of the medical sciences—a contribution as great as that represented by the immediate benefit to the stricken patient no longer liable to the dangers of wound infection.

Difficulties of experimentation.

It does not seem to be realized by the opponents of such forms of research as entail experimentation upon animals, how few individuals undertake it, for the work requires elaborate preparation, expensive and delicate apparatus, is time-consuming and fraught with baffling disappointments. Ehrlich experimented with 605 synthetically prepared chemical compounds before salvarsan was attained. Painful experiments were necessary before anæsthesia, just as painful surgery was necessary; and the extraordinary

[1] It was during this laboratory course that a woman prominent in the local antivivisection circle, who owned a number of cherished and interesting dogs, brought one of them, a valuable bull-terrier, to be operated upon for an extreme vaginal prolapse—one of the complications of whelping incidental to these highly-bred pets. The owner was so gratified by the result that the animal and one of its mates were subsequently brought to the laboratory to have their ears pointed and tails docked—a request which was of course refused. Such are the inconsistencies of mankind!

thing is that the physiologists of the days before surgical anæsthesia and reactionless wound-healing ever learned from animals what they did of the functions of the human body, for the solution of the problems of digestion, circulation, secretion, and nervous action cannot be satisfactorily undertaken if the subject be emotionally disturbed or suffer pain or be not gently and properly cared for. Whatever the conditions may have been in the past, the opponent of research need have no apprehension to-day on the score of the elicitation of pain. And in this connexion is it not extraordinary to recall the protestation once raised by poets, divines, and even physicians themselves, against the banishment of pain by ether and chloroform—for had not man been fashioned to suffer?

Anæsthesia and pain.

Experimental therapeutics will in time doubtless give us the ideal anæsthetic, in the form of a drug possibly allied to chloretone or scopolamine, a single injection of which will induce a prolonged insensitive sleep, perchance of long enough duration for primary wound-healing to occur. Then will the surgeon's weight of responsibility, whether in laboratory or clinic, be greatly lightened, for undoubtedly to-day inhalation anæsthesia itself gives him his chief sense of anxiety, and his patients, whether animal or man, their chief discomforts. Still, in spite of its present defects, anæsthesia—and it is a pity that there is no one man to whom we can unqualifiedly attribute its discovery—shares with Lister the credit of having made of surgery the most important agent which we possess for the cure of disease. Its principles are so definite, its technique so easily acquired, its performances in the long run so safe, that as a therapeutic measure human vivisection—to use this cruel word in a sacred sense—has almost wholly lost its terrors. The triumphs of surgery stand beside those of hygiene and preventive medicine as the notable medical achievements of these thirty years.

Of the new alignments which have been the outcome of modern surgery I particularly wish to speak. Is it not a striking fact that the health officers comprising the medical corps of our armies and navies—men who have a medical degree, and who may be called upon in an emergency to practise any branch of greater medicine—are called surgeons? I have indicated in some of the preceding paragraphs how far their activities may be removed from handicraft, supposed to be the chief business of surgeons. Though formerly this may have been their sole occupation, they are now coming to use more the armament of the scientific physician and public health officer.[1] Thus the army medical officer, unhampered by tradition, more nearly fulfils the office of the Greek physician, who, as Sir Clifford Allbutt has so well said, had no

The army surgeon becomes physician and health officer.

[1] One should of course not forget in this connexion Sir John Pringle, the originator of the Red Cross idea, who may be regarded as the founder of 'military medicine', and whose volume, *Observations on the Diseases of the Army*, London, 1752, is one of the medical classics.

more scruple in using his hands in the service of his brains than had Phidias or Archimedes; whereas his brother in civil life still adheres to the traditions of the times when the manual and debasing craft of surgery was cast off from medicine.

Surgery, by force of circumstances, groped its way alone through the dark centuries—a craft largely devoted to the repair of wounds and injuries inflicted in warfare. But its principles were sound, its growth steady—until now, and particularly in the past generation, it has rapidly developed into the most generally useful therapeutic measure which the physician has at his command. Its principles, moreover, are as profitably employed by the investigator in his laboratory as by the clinician in the case of an individual the victim of disease. It has served to break down medical sects and systems, such as Hahnemannism—for what principle of homœopathy justifies the use of scalpel and forceps? Indeed, surgery has been one of the great factors in the present realignments of medicine, for from Lister and Pasteur as their fountain head, the great streams of progress have flowed, in the case of the individual, into the art of surgery, and in the case of the community, into prophylactic medicine. Even the physician, who for so long held himself aloof from anything savouring of handicraft, returns to it with that useful instrument, the hollow needle; and paracentesis, lumbar puncture, and the extraction of blood for diagnostic, or the administration of drugs and sera for therapeutic purposes, by a minor surgical act are an acknowledged part of his therapeutic resources. By a strange transformation, too, he has become the phlebotomist, and the venesections and cuppings, formerly the overworked province of the barber surgeon, are largely practised by him to-day.

The physician reverts to handicraft.

Billroth said some thirty years ago, 'Die innere Medicin muss mehr chirurgisch werden,' and this seems to be what is taking place. Before Billroth's time it was largely internal medicine versus external surgery, for operative measures were necessarily confined to the exposed parts of the body; the visceral surgery of to-day—the larger part of the present surgeon's work, in the inauguration of which Billroth himself played no inconspicuous part—was then inconceivable.

We still retain many of the traditions of those earlier days, days when a pure craftsman, steeled to act in the face of unanæsthetized suffering, amputated, removed surface tumours, ligated vessels, and cut for stone, the fistula, and a few other ills, with an unerring hand and unbelievable speed. The high-road to surgery was through the anatomical dissecting room, and in these surroundings the operations of surgery were then, and are still, largely taught, with the result that the rule-of-thumb operations of one generation are handed on to the next, who will never use them, owing to the extraordinary progress ever making in these measures, due to methods of asepsis and the increasing knowledge of surgical possibilities.

Changes in the character of surgery.

To-day an individual of wholly different temper from the surgeon of

old ventures to do operative work, and the essentials for his training are of an entirely different order. Descriptive anatomy, useful as it may be to him, is no longer a prime requisite in the terms of the past generation, but his knowledge of visceral physiology and pathology must be broad, his observations of asepsis unimpeachable, his skill in hæmostasis and handling of the various tissues delicate and painstaking. As a training ground the anatomical dissecting room fast gives way to the experimental laboratory.

An entire change in the character of operative workmanship largely explains the transformation in the surgery of the past decade or two. The accurate and detailed methods in the use of which Kocher and Halsted were for so long the notable examples have spread into all clinics—at least into those clinics where you or I would wish to entrust ourselves for an operation. Observers no longer expect to be thrilled in an operating room; the spectacular public performances of the past, no longer condoned, are replaced by the quiet, rather tedious procedures which few beyond the operator, his assistants, and the immediate bystanders can profitably see. The patient on the table, like the passenger in a car, runs greater risks if he have a loquacious driver, or one who takes close corners, exceeds the speed limit, or rides to applause.

Visceral surgery and specialization.

And in all of this new visceral surgery it seems to me that the physician has merely come to do his own operating, that internal medicine is merely becoming surgicalized, much as military surgery has become largely medicalized. In the breaking up of greater medicine into its clinical specialties, the primary cleavage gave us the physician and surgeon, with medicinal versus manipulative forms of treatment as their essential distinction. Chiefly on anatomical lines each of these major subjects has broken up into a second or even a third order, but in the case of these further subdivisions it is to be noted that there is an inevitable tendency for operative therapy to become attached thereto, so that we have the ophthalmic or aural or laryngological surgeon, the gynæcological, orthopædic, genito-urinary or neurological surgeon, according to the direction whither opportunity or training or inclination may have led. Even the obstetrician—the half-brother of the physician and surgeon—reaches out for the operative side of the diseases of women, with the complete *Frauenklinik* in mind.

Convergence of medicine and surgery.

And what is happening in these specialties is an indication of the tendency in the two major branches, for internal medicine and surgery, as the treatment of disease grows less empirical, unquestionably tend to converge. Having the same preliminary training, possessing the same diplomas, the graduates of our schools gravitate toward surgery if handicraft appeals, otherwise toward medicine, but in habit of thought and general attitude toward disease there is little difference to-day between the higher order of physician and surgeon. Though possessing a strong distaste for the act of operating itself, many physicians have admirable surgical judge-

ment and are perfectly capable of the surgeon's craft, just as a well-schooled surgeon should be capable of practising the physician's art. The basis of their training has been the same, and their divergence to-day comes about only through occupational likes or dislikes. Both have the same familiarity with the more common disorders—those of the abdominal cavity for example—and indeed the surgeon, provided he devotes the time he should to the preliminary study of his case, is apt to hold the advantage over his medical colleague in this regard, unless the latter industriously follows his patient to the operating table, where, as is known, he can often learn from living pathology much that was impossible from the pathology of the autopsy room.

A clinic, let us say on aneurism, is given in very much the same vein by both physician and surgeon, and their common road forks only at the post of treatment, where down one (and the longer) path beckon Valsalva, Tuffnell, and Balfour, to rest, a low diet and potassium iodide, and down the other (and shorter), Hunter, Corradi, and Matas, to some method requiring exposure of the lesion through an incision.

The surgical internist the physician of yesterday.

The internist has learned that effective operative work cannot be done well for him by one who merely represents his hands, and he would not venture to refer his patient with obstructive jaundice or hæmaturia or pyloric stenosis to a mere operator who did not have the same knowledge of and the same attitude toward the pathological lesion of liver or kidney or stomach that he himself has. This, in other words, is equivalent to the physician's doing his own surgery, for I think it is stupid to say, as has been said, that the surgeon has taken the appendix and gall-bladder, and now the stomach, thyroid and brain, and threatens to take the lungs and cardiovascular system and so on, away from the physician. It means merely that as the physician becomes more and more a craftsman and user of instruments of precision, internal medicine and surgery tend to draw together and to overlap, as they have not done since the thirteenth century, when the physician-priests were debarred by ordinance from undertaking any operation involving the shedding of blood.[1]

Neurological surgery.

In no subdivision of medicine has this been more apparent than in the difficult subject of neurology—a subject which has been conspicuously poor in therapeutic resources. Time was—and not long ago—when under the direction of the neurological diagnostician, who mapped out the field of operation on the scalp, blundering attempts were made to do his cranial surgery for him. The combination was an utter failure, for the same reason that similar combinations had always

[1] By no means should this idea be distorted into an encouragement for doctors with insufficient technical training to undertake their own operations; and I understand there is a widespread tendency in some districts for them to do this, brought about in all likelihood by the fact that with his diminishing practice many a lesser physician feels that he can no longer afford to 'turn over' his surgical work to another.

proved failures before. Not until the operative possibilities of neurology were taken up by those possessing some preliminary knowledge of and interest in the nervous system and its diseases, as was notably the case with Sir Victor Horsley—in other words, not until certain neurologists began to do their own surgery—did the recent rapid strides in this direction come about. To-day a school of operating neurologists is being trained to continue this work, for as promising a future lies before intracranial as lay before abdominal surgery; but the error must not be made of permitting surgical specialists to enter this or any other restrictive field of operative endeavour without a broad preliminary training in medicine and general surgery. This mistaken course, critics say, has been pursued in some of the established surgical specialties. That they may lose sight of the patient in their expert knowledge of the disorders of a certain portion of his body is a natural tendency of those who particularize in their work at too early a period.

The operating specialist and the general surgeon.

This undesirable attitude can be forestalled only by a long apprenticeship in general medicine and surgery before specialization is undertaken, and by subsequent contact and free exchange of ideas with those who continue in more general work. Indeed, the existence of the operating specialist, as contrasted with the general surgeon, is justified only if the former takes advantage of his opportunities to contribute to the knowledge of the disorders he specially treats. When progress ceases to be made, through the intensive studies which the smaller field of work permits, there is every reason why the vagrant specialty should be called back under the wing of its parent, general surgery, from whom under no circumstances should it ever be permitted to wander too far.

Place of the specialist.

The surgical specialties, to change the figure, should represent merely grafts on the parent stem, for in their cultivation as separate plants they may cease to blossom and bear fruit. Indeed, the establishment of sub-departments as offshoots of general surgery in a given hospital is justified solely by the promise of productivity of the individual around whom such sub-departments are permitted to grow, but with no anticipation that a successive line of equally competent individuals will be found to justify the continuance of this or that specialty in perpetuity. By such a process of inbreeding the stock is rapidly weakened. It has been said that ' the specialist should be a trained physician, a skilled surgeon, and something more, but he is often something else—and something less '.

One thing which has kept the so-called general surgeon from getting a distorted image of his patient's malady—a vice of which the specialist is often accused—is his wider experience with disease affecting the body as a whole ; and it would be rejuvenating to many of the surgical specialties if they could be periodically absorbed by general surgery, to be born again under the leadership of individuals who through fresh contributions could give a new impulse to a senescent subject. On this basis there are

any number of fields which under cultivation could be made to yield abundant crops ; for the blood-vessels, joints, thoracic viscera, ductless glands, and much else, are as deserving of intensive study from the operative standpoint as were the pelvic organs, the nose and throat, or the genito-urinary apparatus.

Rewards of special work.

And for those who on this basis prove worthy of the opportunities of specialization, and for whom every possible facility, in laboratory and clinic, should be given for intensive work, the most satisfactory rewards are open. In commenting on his thyroid investigations, the celebrated Pirogoff once said to Professor Kocher that ' the most perfect satisfaction which one can have in medicine comes through the thorough study of one special chapter in every direction and from every point of view—clinical, histological, physiological, and pathological '. No knowledge or experience which in fragmentary fashion may cover a multitude of ills can be comparable to this thorough familiarity with one subject from every possible aspect.

Institutional insularity.

Still the general surgeon is by no means exempt from the frailties of the hereditary specialist. He, no less than others, easily becomes the slave of local custom, a creature of habit, who accepts innovation with reluctance. The customs, good and bad, of a surgical department are easily handed on from chief to house officer, to clinical dresser, with little change from generation to generation. Customs which once were good may have staled, and those whose vision is limited to the walls of a single institution be unaware of it. At the beginning of the present century, twenty years after it was deemed unnecessary by its originator, the carbolic spray was actually to be seen in use in a famous hospital.

A certain loyalty to the methods of a locality, and even to those of an individual hospital, is apt to be cherished and suspicion cast upon the hearsay methods and results of other, and possibly rival, clinics. This, I am told, was formerly true of your great London hospitals no less than of others. New technical principles, unless they have been observed and their value appreciated, find their way slowly from institution to institution. Reading of methods will not suffice ; the objective exchange of ideas, which can come through demonstration alone, is necessary. The rubber glove was in general use in Halsted's clinic for twenty years before it was adopted elsewhere. Those who had not visited his operating room scoffed at the idea. What was to become of our prized *tactus eruditus*? But onlookers gradually became disciples, and now this useful safeguard is so generally employed that the very source of the idea is forgotten.

Aids to precision.

' Surgical instinct ' and the ' educated touch ' have been overworked shibboleths which have stood in the way of many innovations, particularly those which concerned the introduction into practice of instruments of precision. As told in rhyme by Oliver Wendell Holmes, fun was poked at Laënnec and his newfangled toy, the stethoscope. Probably Floyer heard a protest against the ' pulse watch ',

and Wunderlich against the thermometer; and only yesterday was one raised against the instrumental measurement of blood-pressure. Had not the hand been industriously trained to estimate the degree of fever by contact with the patient's brow, and the fingers the quality of the pulse at his wrist? Even the Röntgen ray brought misgivings to those skilled in the elicitation of crepitus from fractured bones.

The tendency to disparage the so-called accurate or scientific attitude in medicine and to make much of the practical doctor or surgeon who is superior to the microscope and other laboratory accessories is rapidly passing. It was one of Paré's canons that

> Science sans expérience
> N'apporte pas grand' assurance.

But the confidence which is bred of practice uncrossed with science is still less dependable. Abraham Flexner states that during his study of the North American medical schools he heard much talk of training the practical rather than the scientific doctor, and remarks that the chief difference between them, as far as he can see, is that the scientific doctor is less likely to lose his patient. Most progressive surgeons of late years have adopted the wise custom of frequent inter-institutional visitations, and this custom of attending the clinics and observing the methods and workmanship of others has done much to reconcile these opposed points of view.

Travel organizations.

To make this habit more effective than was possible when indulged in by an individual alone, just ten years ago a small group of surgeons in the United States formed a 'Travel Club', which at stated intervals has since visited, in rotation, the institutions of the six or eight important medical centres represented in their organization. At these exercises no papers are read, but a concerted effort is made by the local members of the society and their colleagues to open their clinics, laboratories, and class-rooms for inspection while under full operation. Thus, as in our student exercises the present educational tendency is less and less toward subjective, and more and more toward objective methods of teaching, so the success of the organization I mention has demonstrated that the same principle is equally applicable to medical societies. Similar travelling groups have come to be established in other divisions of medicine and in more specialized branches of surgery, and I believe, too, that the idea has taken root in England and other countries.

The Society of Clinical Surgery.

From its foundation, the object of our society, inasmuch as its members were mostly teachers and investigators as well as surgeons, was threefold—to observe the methods of instruction pursued by their colleagues in other cities, to follow at first hand the progress of their laboratory studies, and to attend their surgical procedures in the operation room. Our early peregrinations disclosed very obvious differences in surgical habits, thought, and workmanship among the various institutions represented in the society membership—

differences, however, which in the course of a few years largely disappeared. Unquestionably there is now a more nearly equal and a much higher level of workmanship throughout; and as the members of the organization in one capacity or another are in the position to pass along the instruction which they have received, this will not only have a broadening effect on the surgery of the present, but a still more marked influence on the surgery of the next generation. Such a clinic as that of the Mayo brothers, where may be seen a composite of the best in international surgery, is a good example of the effect of sympathetic, intelligent, and systematic inspection of the work of others.

Experimental pathology and surgical technique.

As I have said, the application of surgical principles, whether in laboratory or clinic, is constantly becoming more general. The slipper of surgery has been found to fit that Cinderella of Medicine, experimental pathology, whose coach now has devoted outriders representing all departments. Observations which even in the hands of a Bernard or a Cohnheim were impossible before the era of reactionless wound-healing now become possible, and not only may the counterfeit of conditions of disease be produced and studied without the complicating element of sepsis, but, with surgical methods, the Pawlows and Carrells of the laboratory bring about altered conditions which shed new and important light on physiological processes.

All this has led to the establishment of special laboratories of surgical research, where modern operative methods are particularly applied, but as a matter of fact they are nothing more than laboratories of experimental pathology, or, better still, of *experimental medicine*—as the Institute in St. Petersburg and the Hunterian Laboratory in Baltimore are called. The establishment of similar institutions elsewhere indicates that an increasing number of surgeons to-day have instincts which lead them into the broader aspects of disease and away from the mere operative tasks that formerly kept them in the dissecting room. Other than this there is no special significance in these laboratories, where investigations are undertaken which are as appropriate for anatomy, physiology, biochemistry, or pathology as for medicine or for surgery.

Departmental interdependence.

Indeed, the definite boundaries formerly separating these subdivisions of the curriculum have become so overgrown by surgicalized experimental pathology that it is sometimes difficult to tell when the investigations of one department are trespassing on the preserves of another, nor does it much matter if the common goal is the advancement of medical knowledge. This unquestionably is a development in the right direction, for it is a peculiarity of the medical sciences, as Huxley once remarked, that they are independent in proportion as they are imperfect. Even anatomy, long a purely descriptive science, is adopting the experimental method. At Johns Hopkins a professorship of *physiological* anatomy has recently been established, and the Carnegie Institution has founded an institute of experimental

embryology and has put a great anatomist at its head. These are straws of promise for the future anatomist, and he will be brought into closer contact than before with the physiologist, the pharmacologist, and the pathologist, who themselves already touch elbows so intimately that many of the problems which engage one of them might suitably be undertaken in the laboratory of one or the other of his colleagues.

In consequence of this lessened departmental insularity, all react far more quickly than heretofore to a ray of light disclosing some new aspect of disease, just as they do to some novel method of study which reopens a door long closed against profitable investigation. The glands of internal secretion and their interrelations, brought to the fore primarily through the clinic; the principle of intravital staining, with all its promising ramifications, introduced by the experimental therapeutist; the studies of cellular physiology which refined methods of tissue-staining promise to make so fruitful; and the growth of tissues *in vitro* by the experimental anatomist—these occur as immediate illustrations.

A closer contact between clinical and preclinical science.

Indeed, the possible prompt application by the clinician of many of the discoveries made by his colleagues in the medical sciences, as well as the fruitfulness in turn of the hospital clinic in problems suitable for the pure scientist, have served to realign them so that in some institutions the physiologist and pharmacologist have wisely been given posts comparable to that long held by the pathologist alone. Thus, through experimental or physiological pathology—or by whatever term one may wish to designate the present rejuvenation of a method which is having such a strong influence in the unification of their labours—biologist, morbid anatomist, therapeutist, hygienist and sanitarian, physician and surgeon alike, are brought into closer copartnership than has heretofore been thought necessary or advisable. And as the physician and surgeon develop under the influence of a closer contact with their brethren of the non-clinical medical sciences, the leaders of these two branches will of necessity come more and more to hold an academic attitude toward their posts.

Hospital practice versus home practice.

This inevitable tendency, so far as our hospitals and schools are concerned, will have a revolutionizing effect on clinical medicine and surgery. The change cannot be made in a moment, any more than it was possible in a moment to place anatomy and physiology and the other preclinical sciences on a full-time academic basis, but once established we would no more recede to the earlier conditions, where energies were divided between school and practice, often to the detriment of the school, than we would now in the case of the so-called scientific branches.

And this brings me to the final subject on which I wish to touch, one which of late has aroused much discussion and has been thoroughly ventilated here in London before the Commission on University Education —namely, the relation toward the hospital and medical school to be held

by the new order of physician and surgeon and their junior associates. Herein lies a matter of great concern to many and a problem which will not be solved by shunning it.

Our great public hospitals have ceased to be grievous and infected places, and people are rapidly losing their dread of them. Meanwhile, as disease becomes better understood, the complexities of its accurate recognition and successful treatment become ever greater and require the studies of an increasing number of co-workers—a situation which makes a most unsatisfactory matter of the office or home consultation. In consequence people, in greater and greater number, desire when ill to enter institutions for study, and this applies to the well-to-do as well as to the poor.

Increasing complexities of diagnosis and treatment.

Consider, for example, the present-day methods essential for the recognition and treatment of a single disease which with its varied complications is possibly not only the most prevalent one the doctor encounters, but which has been regarded as the one most easily prescribed for. Indeed, so definite and so effective were his therapeutic measures supposed to be that the shabby habit was acquired of making the administration of iodides and mercury serve as an aid to diagnosis—a habit which often led to added confusion. To-day the clinical acumen of the practitioner, experienced though he may be, has given way to a chemical test by a laboratory expert, and only when the disease is thus proved to exist is a diagnosis accepted. The discovery of the spirochæte and the study of its biological habits have taught us that in certain parts of the body, especially the central nervous system, the organism is not accessible to the former methods of treatment, or indeed, if reached, that it may have acquired an immunity and resistance toward the very drugs supposed to destroy it.

Let us suppose that the malady has taken the form of a cerebral gumma. The patient has headaches, and there is a suspicious clinical history. What is required? A lumbar puncture, a cytological study of the fluid, and the Wassermann test requiring one specialist; an ophthalmological and perimetric examination needing another; a neurologist certainly, for a careful study is necessary to determine if possible the situation of the lesion; probably, too, a röntgenologist; and in the end, perhaps, another to administer salvarsan; or, in the case of a non-absorbable fibrosyphiloma, as likely as not an operation will finally be needed. And this for what was yesterday a simple problem, easily faced by the practitioner single-handed! Thus, scientifically accurate diagnostic measures and a new form of therapy requiring blood-letting and manipulative skill make a craftsman even of the syphilographer. This is a far cry from a written prescription, a bottle and spoon.

Requiring a multiplicity of consultants.

No, the practitioner cannot be chemist, neurologist, bacteriologist, ophthalmologist, radiographer, surgeon, and what not rolled into one,

and do effective work. The Jonathan Hutchinsons of the profession are, alas! rare. And as the co-operation of those expert in special lines becomes more and more necessary the tendency will grow for these conjoint studies of individual cases of disease to be carried out in properly equipped hospitals, where the data essential for a diagnosis can be more quickly and effectively accumulated and the paraphernalia for treatment kept in smooth running order from constant use. This is particularly true in so far as it concerns surgery, for in proportion as this form of therapy is the more dangerous, so much the more need is there for surrounding it by every possible safeguard against misuse or accident.

The more difficult and complicated problems of disease will thus gravitate to large institutions where no longer 'visiting' appointees but directors of hospital units in continuous service, aided by a correspondingly adapted hospital administration, can uninterruptedly devote themselves to their work without entering into competition for practice beyond the walls of the institution. For when advantage is taken of a hospital position to make it a high-road to practice, the visiting physician or surgeon immediately becomes a less valuable institutional servant, if for no other reason than through the dissipation of his energies. When this abuse is widely corrected the family physician of old, so rapidly passing as a type of practitioner, and than whom no more useful member of the community ever existed, may again come into his own, with an even larger and better field than before, and quick to take advantage, in his more difficult and serious problems, of institutions where the chiefs of service are in no sense his competitors, but rather his coadjutors. Owing to the present dismemberment of medical practice into its many specialties the public has begun to feel that there is no balm and no physician in Gilead.

Academic clinical posts.

The family doctor.

Meanwhile it will be the *energetische Imperativ*, to use Ostwald's symbol, of the salaried hospital incumbents to take advantage of their opportunities to observe and investigate conditions of disease for other and higher reasons than the expectation of financial returns from competitive practice. They will also instruct and be instructed by their full-time junior associates, and these in turn by the student body, without whose actual aid and stimulus a properly modelled hospital organization can hardly survive, and who in return will receive, in addition to the advantages once attached to the old apprenticeship system, the added benefit of contact with the active work of co-operating departments.

Hospital patient, student, and teacher.

The cry has been raised that this evolution of the physician and surgeon will make teachers incompetent to instruct those who are to enter practice, but this same cry was once raised against the appointment of a full-time physiologist, anatomist, and pathologist, whose loss of contact with practical medicine, it was presupposed, would disqualify them on these same grounds. After all, is not the essential thing to inculcate in the

student's mind the habit of acquiring clinical knowledge for himself? And there is no possible way of doing this except by bringing him into immediate touch with the sick-bed. This, in the terms of Huxley, makes for centrifugal education, whereas our customary methods are centripetal in direction, with the effort to drive formulas into the student's head rather than to teach him how to produce ideas of his own. In an address on this subject Henry Fairfield Osborn aptly said that 'the measure of a teacher's success lies in the degree with which ideas come, not from him, but from his pupils'.

Summary.

These, then, are some of the transformations and realignments which are taking place in medicine, and which, directly or indirectly, have come through the great contributions made a generation ago by those with whose names this address was introduced: the public, not only awake to the great developments in medicine which animal experimentation has made possible, but ready to participate in their application for the general welfare; the physician at large, the valued family adviser, reaching out toward the important problems of hygiene and prevention of disease, while the individual patient, as the complexities of disease unravel, needing more and more specialized and more and more surgicalized treatment, gravitates toward the modern hospital; the preclinical science departments, which formerly existed in splendid isolation, with their fences down, owing to the levelling influence of experimental pathology; hospital organizations undergoing changes which will ultimately require the full time and continuous activity of those who serve them for the benefit alike of student, of science, and of patient.

The kaleidoscope of medicine is turning. These are merely some of the present rearrangements of the images as I see them—new patterns from original fragments—for 'Is there anything whereof it may be said: See, this is new? It hath been already of old time, which was before us'.

GENERAL ADDRESS ON

CHEMIO-THERAPY

By Seine Exzellenz Wirklicher Geheimer Rat
Professor Dr. PAUL EHRLICH

It must be a great pleasure and a special honour for all of us to meet here personally on British soil for a scientific purpose, in order to take part in the great work which will be of benefit to the whole world. Are we not here in a country that has produced two men who must be considered among the greatest men of all times, Jenner and Lord Lister? Like a star in the darkness of his age, Jenner's great achievement, which broke the power of such an awful public plague as small-pox, still shines with peerless splendour. And on the occasion of the last Congress which was held here we gathered with wondering admiration round Lord Lister, who through his introduction of antiseptics brought about a revolution in surgery which stands alone in the history of medicine. Here in England the first example of a modern Institute for Tropical Diseases, which is a model for all other institutes of this kind, was created under the direction of Sir Patrick Manson. Through Ross's excellent work, Laveran's discovery of the causes of malaria was so far advanced that entirely new lines were opened up for the hygienic struggle against tropical and subtropical diseases.

The proof by Castellani that a trypanosome is the cause of sleeping sickness, the classical work by Bruce on illnesses caused through trypanosomes, the specific cause of kala-azar (Dum-dum sickness) as proved by Leishman, are all well known to us. The therapeutic influence of atoxyl in the cases of trypanosome diseases was first established in the Liverpool Tropical Institute by Thomas and Breinl, and quite recently Plimmer has brought forward the use of tartar emetic as an effective weapon against protozoal diseases.

The life-work of Almroth Wright is also known to all of us, i. e. his work on opsonins and on the prophylactic treatment of typhoid fever, which has been carried out in a practical and most excellent manner. Even these few names, to which I might add many others, show what a high and leading position England has taken and still holds in the fight against infectious diseases. To prevent the spread of and to heal infectious diseases was at all times the highest aim of medical aspirations; however, a systematic pursuit of this purpose has only been possible in recent times, as through the labours of all civilized nations we have got an

insight into the nature of infections, the cause of diseases, and the means by which they are transmitted. Through these methods it has been possible to infect animals artificially and so obtain material on which to test the drugs in a systematic and rational manner. From the very first beginnings of therapeutics, chemio-therapy has indeed been in existence, as all the remedies which we employ are chemicals ; on the other hand, experimental chemio-therapy could only develop in modern times in a fruitful manner as a result of all this pioneer work. But here also it has been proved that the four most important factors are : *patience*, *skill*, *luck*, and, last but not least, *money*.

Now, gentlemen, I may perhaps take the liberty of giving you an insight into the workshop of the chemio-therapeutic work. The whole area is governed by a simple, I might even say natural, principle. If the law is true in chemistry that ' Corpora non agunt nisi liquida ', then for chemio-therapy the principle is true that ' Corpora non agunt nisi fixata '. When applied to the special case in point this means that parasites are only killed by those materials to which they have a certain relationship, by means of which they are fixed by them. I call such substances 'parasitotropic '. But I should like immediately to add that there are evident exceptions to this law. So, for instance, we are acquainted with a small series of cases in which the apparent therapeutic results are obtained although the allied substances in question do not possess parasite-destroying qualities. That is the case in the infiltration of the subcutaneous tissues which is caused by a kind of yeast (sporotrichosis). Here Block proved that the clinically highly therapeutic iodide of potassium first of all dissolves the cells of the infiltration, whilst the parasites, as such, are not in the first instance attacked.

But, in any case, it is safest and best for the development of chemio-therapy not to build on the basis of exceptional work, but it is better to start with such substances which produce the destruction of the parasites by fixation.

Now it has been assumed in different quarters that some of the more modern remedies are incorrectly regarded as parasiticides, but in reality they are not such. Thus, for example, salvarsan or mercury salts are not intended to act directly on the parasites, but indirectly, owing to the fact that they excite the organism to the formation of specific anti-substances. This view is based mainly upon the fact that if one mixes the substances in question, such as, for instance, neosalvarsan, with certain pathogenic agents, e.g. spirochætes, in test-tubes one cannot perceive any reduction in their mobility after observing them for hours together. From this fact, which was first discovered by Professor Hata, the conclusion has been drawn that salvarsan or neosalvarsan, as such, did not in any way directly influence the spirochætes. Now it can very easily be shown that this conclusion is quite incorrect. If, for instance, following Castelli, one suspends the spirochætes of relapsing fever in indifferent mixtures of serum which do not injure their vitality, and if

one fills two small tubes therewith and adds to one of the tubes a very small quantity of salvarsan or neosalvarsan, and if one then centrifugalizes and then draws off the liquid ; if one washes the remaining spirochætes again in a mixture of serum and again centrifugalizes it ; then one obtains in both tubes a deposit of spirochætes which on microscopic examination shows the same properties, i.e. equally good mobility of the spirochætes. If, however, the spirochætes obtained in this manner are injected into test mice, then one can very soon convince oneself that the spirilla treated with salvarsan do not give any infection to the animal, whilst the mice vaccinated with the contents of the control tube promptly show signs of infection. This proves that salvarsan or neosalvarsan, as the case may be, is absorbed by the spirochætes, and must have damaged them, and that this trace of salvarsan which is so exceedingly minute that it can scarcely be weighed, was sufficient to prevent the increase of the parasites in the animal body. Therefore, by this very simple and easily intelligible experiment, the direct effect of salvarsan and neosalvarsan on the spirochætes, and thereby the principle of fixation, is absolutely proved ; the objection of the indirect effect based upon anti-substances therefore falls to the ground.

It was necessary, however, to penetrate more deeply into the mechanism of this fixation of remedies, and it is only after long-continued efforts that success has been attained in obtaining a clear conception. In order to make practical progress it appeared to be necessary not to be satisfied with the primordial idea, but to see in what manner the drugs are fixed by the parasites or, as the case may be, by the cells. Only by taking a very roundabout way has it been possible to obtain clearness with respect to these complicated relations, and in this connexion it was especially the studies on trypanosomes, and especially the investigations of ' drug-fast ' strains of germs, which led to quite definite representations with regard to the process of fixation. There was no difficulty by continued treatment of the experimental mice with certain definite remedies, e.g. fuchsin, in finally obtaining a race of trypanosomes which had become immune against these remedies, i.e. ' drug-fast ', in the case mentioned above immune to fuchsin. There were especially three classes of different remedies which were very suited to this purpose :

1. The class of the arsenic compounds (in the following historical order : arsenious acid, arsenilic acid (atoxyl), arsenophenylglycin (salvarsan and neosalvarsan).

2. The class of the so-called azo-dyes (the trypan red manufactured by Weinberg, with which Shiga and I made experiments, and the trypan blue of Mesnil).

3. Certain basic triphenylmethan dyes (fuchsin, methyl violet, &c.).

When a race of trypanosomes has been rendered immune against fuchsin, then this race is immune against all the allies of fuchsin and methyl violet, &c., but is not immune against the two other classes.

Also a race immune against arsenic compounds is only immune against such, but not against the two other classes. We see, therefore, that the immunity is of a specific nature inasmuch as it is limited to a definite class of chemical substances.

It was just this specific character which indicated that it must be a question of purely chemical processes. Earlier studies relating to another subject, i.e. those relating to toxins and antitoxins, pointed to the nature of the said processes. In connexion with these it had been shown that the destructive toxins developed their injurious action on the cell by the fact that they are absorbed by certain specific component parts of the cell—side-chains—which I have characterized as ' receptors ', and that the anti-substances represent nothing else than the cell receptors produced in excess under the influence of the toxin and thrown off.

For many reasons I had hesitated about transferring these views relating to receptors to chemical bodies at all, and in this connexion it was especially the brilliant investigations by Langley relating to the effects of alkaloids which caused my doubts to disappear and made the existence of chemo-receptors seem probable to me.

From this point of view, the phenomena observed in connexion with the ' drug-fast ' strain of germs can be readily explained experimentally, owing to the fact that the chemo-receptors under the influence of drug-fastness suffer a reduction of their affinity for certain groupings connected with the remedy, which can only be regarded as purely chemical. This reduction in affinity explains in the simplest possible manner why continually increasing quantities of the arsenic compound become necessary for the destruction, e.g., of a race of arsenic-fast trypanosomes, for the smaller avidity can only be overcome by a corresponding surplus of the arsenic compound, if the quantity necessary for the destruction of the parasites is to be finally fixed.

We, therefore, come to the conclusion that in the parasites there are present *different specific chemo-receptors*; for instance, the *arseno-receptor*, which fixes the trivalent group of arsenic as such, and the *acetico-receptor*, which fastens to itself the acetic acid group; an *iodine-receptor*; an *ortho-amidophenol-receptor*, which conditions the fixation of the salvarsan; and many others in addition. A complete exhaustive knowledge of all the different chemo-receptors of a certain definite parasite is what I should like to characterize as the *therapeutic physiology of the parasite cell*, and this is a *sine qua non* of any successful chemio-therapeutic treatment. I should like to emphasize the fact that many observations indicate that certain chemo-receptors are due to several different kinds of parasites, not to a single one. The knowledge of these is of special practical importance, because remedies which are adjusted to these have a healing influence on a very large series of the most various pathogenic agents. *The larger the number of different chemo-receptors, therefore, which can be demonstrated the greater is the possibility of a successful chemio-therapy.*

Now if we seek for specific remedies, then the first condition is that they must possess a certain definite grouping, which is chemically allied to one of the chemo-receptors of the parasite. This is only a necessary prior condition of the toxic effect, but in general it is not a sufficient one in itself. Hundreds of substances may fix themselves on a parasite and only a few are capable of bringing about its destruction.

In the therapeutically suitable substance there must, therefore, in addition to the fixing group, which brings about the fixation of the *haptophoræ*, be another, which as such brings about the destruction, and which therefore is to be characterized as the ' poisoning ' or *toxophoric*. This representation exactly corresponds to the views which we have already long since obtained with respect to toxins, in which we distinguish the presence of a haptophoric group which conditions the cell fixation and also the formation of the antitoxins, and a toxophoric group which brings about the injurious action on the cell. In the case of the highly-complicated synthetic drugs the assumption will have to be made that the haptophoric group and the toxophoric group are not *directly* connected with one another, but as separate groups are linked with a chemical molecule in the character of side-chains. In this way we arrive in a natural manner at this, that chemio-therapeutic agents, built up in a complicated manner, may be compared to a poisoned arrow ; the fixing group of the drug which anchors itself to the chemo-receptor of the parasite corresponds to the point of the arrow, the binding member is the shaft, and the poison group is the arrow poison fixed to the shaft of the arrow. According to this scheme in the case of salvarsan (dioxydiamidoarsenobenzol) the benzol group would correspond to the shaft, the orthoamidophenol group to the point, and the trivalent arsenic group would correspond to the toxophoric group.

If we continue this comparison, then the substances which are used for poisoning the arrows are alkaloids and similar substances, which act injuriously on certain definite vital organs of the body ; and so we shall also have to assume that the toxophoric grouping of the synthetic drugs poisons the protoplasm of the bacterial cell, and this only appears to be possible when a chemical affinity exists between the toxophoric grouping and the constituents of the cell. The circumstance that all the derivatives of arsenic, which contain arsenic in the pentavalent form, i.e. in the fully saturated form, do not bring about any therapeutic action, but that this only commences when the arsenic group exists in the unsaturated condition corresponding to the trivalent radical, certainly points in the same direction. This difference between the saturated and unsaturated arsenic radical was discovered by the master mind of Bunsen, for in the year 1843, in his comparative studies relating to the non-poisonous cacodylic acid with the pentavalent arsenic and its poisonous reduction product the cacodyl with the trivalent arsenic, he came to the conclusion that ' the cacodylic acid had lost the power to form an attacking point, and at the same time it had lost its effect on the organism '. In the subsequent period

a very large series of analogous cases have become known corresponding to this truth, which point to the increased effectivity of the unsaturated radical. The best-known example is doubtless the high degree of toxic power of carbon monoxide as compared with the almost indifferent carbon dioxide.

Dyes act as bactericides only as such, but not in the form of their colourless products which correspond to the saturated type. *The fact is that all these unsaturated combinations contain unsatisfied avidities which render them capable of reacting additionally with other combinations.*

If, therefore, we poison a spirochæta with salvarsan, then there occur at least two different chemical fixations: first of all the fixation of the orthoamidophenol group, which primarily fixes the salvarsan to the parasite. It is only in consequence of this fixation that secondarily the trivalent arsenic group is given the opportunity of entering into chemical combination with the arseno-receptor of the cell, and so to exert its toxic effect. The avidity of the arseno-receptor can in itself be such that it can only react if favouring factors, which chemically must be regarded as a stereo-chemical facilitation, come into action.

Examples of such stereo-chemical facilitations are frequently found in chemistry, e.g. in the chemistry of the ortho-condensations. And so the haptophoric group of the arsenic molecule primarily brings the arsenic along to the cell and secondarily brings about its possibility of action.

Now, it is a frequent practice of many uncivilized peoples, in order to be certain of killing their enemies, that they not only rub over their arrow with one kind of poison but with two or three totally different kinds of poison. And so it also appeared advisable to imitate this procedure against the parasites, which is otherwise not very praiseworthy, and to poison our synthetically poisoned arrows not singly but doubly. In association with Dr. Karrer I succeeded in depositing the reduced arsenic compound, e.g. salvarsan, even on metals, and so in arriving at remedies which, used experimentally on animals, show an increased effect.

In the previous remarks I have described the conditions which are necessary in order that a certain substance may exert a parasiticidal effect, and indeed must effect such, if it operates directly on certain definite parasites in an aqueous solution, such as, for instance, is the case with the ordinary disinfectants. In the manner described above it is easily possible to arrive at a very large number of substances which will destroy bacteria and allied substances in aqueous solutions. But, of course, the problem is much more difficult when it is a question of internal disinfection or of the destruction of living parasites within the infected organism. If the problem is set before us of sterilizing a room, then indeed it is an easy matter to do so, owing to the present advancement of science; but the task becomes more difficult when the room is filled up with materials; and when these materials are of such a delicate sensitiveness as living cells, then the difficulty of the problem will be manifest without any further

explanation. As a matter of fact, it has proved that substances which bring about a colossal bactericidal effect in aqueous solutions even when they are highly diluted, are totally ineffective in therapeutics properly so called. For it has turned out that, generally speaking, these disinfectants are more or less powerful cell poisons, and seriously injure the organism; they are, therefore, not only parasitotropic but also *organotropic*.

Now, it depends exclusively on the relationship between parasitotropic and organotropic as to whether a certain disinfectant can be used as a remedy. In Robert Koch's celebrated experiment, in which even the largest doses of sublimate did not produce even a trace of therapeutic effect on anthrax infection, it is evident that the parasitotropic effect was reduced to nil by the organotropic effect. If the relationship of organotropic to parasitotropic is somewhat more favourable, then one may observe a peculiar phenomenon, consisting in the course of the infection being rendered worse to an extraordinary degree by the remedy, owing to the effect that the parasites increase to a much greater extent than is generally the case.

This phenomenon, discovered by Hata, is explained by the fact that the ratio of organotropic effect to parasitotropic effect is of such a nature, that almost the whole of the poison is absorbed by the organism, but only in infinitesimal quantities by the parasites. According to a biogenetic foundation principle it is quite a common thing for substances, which act destructively in *large* quantities, to bring about an *increase in the vital functions* in smaller doses. Only such substances, therefore, can be used as therapeutic agents in which the ratio between organotropic effect and parasitotropic effect is a favourable one, and that can be easily ascertained by experiment by a comparison between *dosis toxica* and *dosis tolerata*. *Only such substances can be considered therapeutic agents of which a fraction of the dosis tolerata is sufficient to bring about therapeutic effects.*

The organotropic effect of drugs is, of course, to be attributed, according to the views of Langley and myself, to this, that there are, in the most various cells of the body and its organs, quite different chemo-receptors, exactly in the same manner as we have postulated for the parasites. Apart from the pharmacological effect of the various remedies, this chemical difference of the organs appears clearly in the vital colouring.

I will mention here—in order only to indicate a few examples—the methyl blue colouring of the nerve trunks, the neutral red colouring of the cell granules, and the distribution of the isamin blue in the so-called pyrrol cells, so carefully and excellently investigated by Edwin Goldmann. The pathologico-anatomical findings point also to a chemical divergence on principle. When we see that after the introduction of paraphenylendiamine only the summit of the diaphragm assumes a black colouring; when we see that vinylamin in the case of all kinds of animals isolates and injures the renal papillæ and causes them to die; when after the introduction of cyanosin, as Hata and Goldmann have found, certain

definite regions in the hair of mice become coloured, and the colouring matter becomes stored to the greatest degree in the milk glands; when a colouring material of the pyronin series in the case of mice brings about a general dropsy amounting to 50–60 per cent. of the body without injuring the kidney, which doubtless is only to be referred to an alteration of the vessels of the subcutaneous connective tissue;—then all these phenomena can only be explained by the fact that at these particular spots definite chemical connexions of a specific nature must take place, which must be referred to the presence of certain definite chemo-receptors.

Now, according to the above representations, all these fixations are dependent on the haptophoric grouping of the drugs, and, therefore, it was a matter of great interest to observe how phenylarsenic acid, the mother-substance of the modern arsenic compounds, behaves when various different groups are attached thereto. In this connexion it has turned out that when we introduce different constituent fixation groups, e.g. chlorine, the oxygen group, the hydrocyanic acid group, the sulphuric acid group, the ammonia radical, we can manufacture, starting out from one substance, a series of combinations, the toxic effect of which may vary fifteen hundredfold. The combinations which are to the greatest extent free from poison—these are derivatives of sulphuric acid, especially the sulpho-phenylarsin acid and its salts—are less toxic than sodium chloride, and on the other hand there are substances the very smallest quantity of which bring about death. And in this connexion we can see that according to the nature of the substances, very different organs of the animal's body are injured. Sometimes it is the intestinal tract and the animals die of profuse diarrhœa; sometimes it is the liver, and the mice—a rare occurrence—become jaundiced and die of serious alterations in the liver; sometimes the red blood corpuscles become dissolved and the animals die of severe anæmia. Frequently also the central nervous system becomes injured, and in the case of mice this usually relates to the vestibular nerve of the inner ear. The interference with the equilibrium, produced in this way, causes the mice constantly to turn in circles just like the Japanese dancing mice. In the case of human beings the optic nerve is the point of attack for numerous derivatives of phenylarsin acid. The cases of blindness which have been observed after the use of very large doses of atoxyl, arsazetin, and other drugs are due to this injury.

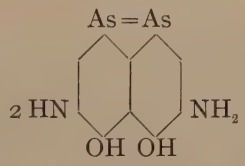
From this it is evident that according to the selection of the group annexed to the phenylarsin acid quite different organs will be affected. This is only explained according to the above views by the fact that there are, as already previously stated, in the various organs specific chemo-receptors which energetically attract certain fixation groups somewhat as the magnet attracts iron. And this view also provides us with the principle *according to which we have to construct our poisoned arrows.* We must attach to the phenylarsin acid group, or as the case may be to the

phenylarsenobenzol group, such grouping as is only related in a small degree to the organs of the sick body, but, on the other hand, is very closely allied to the receptors of the parasites.

I have explained above that the parasites possess a whole series of chemo-receptors which are specifically different from one another. Now if we can succeed in discovering among them a grouping which has no analogue in the organs of the body, then we should have the possibility of constructing an ideal remedy if we select a haptophoric group which is especially adjusted to the functions of the parasites.

A remedy provided with such a haptophoric group would be entirely innocuous in itself, as it is not fixed by the organs; it would, however, strike the parasites with full intensity, and in this sense it would correspond to the immune productions, the anti-substances discovered by Behring, and which after the manner of the bewitched balls fly in search of the enemy. Let us hope that it will be possible chemio-therapeutically to hit the bull's eye in this manner also. I do not consider this at all out of the question, as it may be proved in certain sicknesses, e.g. spirillosis in hens, that from the fiftieth to the hundredth part of the *dosis tolerata* of salvarsan entirely frees the animals from the parasites and leads to a cure. Such a dose truly represents a nil dose, as the hen cannot be damaged thereby in the slightest degree. But such favourable conditions have only very rarely been discovered up to the present; we shall have to be satisfied if we can succeed in obtaining good therapeutic results with the tenth or even the fifth or sixth portion of the *dosis tolerata*.

In the main the above are the principles which guided us in the construction of the new remedies. Among the numerous combinations which have been tested in experiments on animals in the case of trypanosomes and spirillar infection, and in the preparation of which I have been supported by the untiring co-operation of Dr. Benda, Dr. Bertheim, Dr. Kahn and Dr. Karrer, and which have been biologically tested, especially by my respected friend Professor Dr. Hata, and later by Dr. Castelli and Dr. Gonder and Frl. Leupold, salvarsan has proved to be the most efficient, the dioxydiamidoarsenobenzol of the formula



Here the orthoamidophenol group acts as the conducting and the arsenic group as the toxophoric group.

But now, gentlemen, the step from the laboratory to practice, i.e. to the bed-side, is an extraordinarily difficult and dangerous one—a step which can only be taken with the greatest care. Its difficulty and danger are in the main based upon two factors: (1) On the fact that in the case of men there exist so-called idiosyncrasies, forms of supersensitiveness which

do not occur in the case of animals. So, for instance, it is known that with a large number of thoroughly healthy persons, the use of harmless articles of food such as strawberries, crabs, &c., brings about unpleasant skin eruptions, and almost half the known remedies can incite such phenomena of supersensibility. It will not be a cause of surprise, therefore, that such phenomena may occur in a particularly serious form with the employment of therapeutic agencies which contain such powerful acting radicals as arsenic and mercury. I have already referred above to the disturbances of vision and cases of blindness which are produced by certain arsenic compounds.

Fortunately, it is proved that such primary supersensibility in the case of salvarsan is one of the very rarest phenomena, and that it was perhaps due to a number of other circumstances, sources of error of a hidden nature, which in many cases have led to the mistaken idea that supersensibility existed. In this place we must first of all mention Wechselmann, McIntosh and Fildes, Hort and Penfold, who have brought forward the very important proof that the destroyed bodies of bacteria which may occur in sterilized water are capable of bringing about a series of serious and unpleasant phenomena such as fever, vomiting, diarrhœa, &c. And even in the case of a pure unexceptionable water, technical mistakes in the manufacture of the salvarsan solutions may bring about injury ; too large an addition of alkali injures the veins used for the injection ; too small a degree of alkali brings about blood coagulation, and leads to thrombosis ; and a lengthy shaking of the solutions and standing in the air oxidizes the drug to a toxic product, the so-called oxide of arsenic, which is much more highly toxic than salvarsan.

(2) It has been shown that certain illnesses of a constitutional nature can cause a supersensibility. So, for instance, tuberculosis of the suprarenal region, the so-called Addison's disease, is an illness which, according to the observations of Wechselmann and myself, brings about a severe supersensitiveness of the patients to arsenic compounds, and the same applies also to the status lymphaticus, which, as has already long been known, must be regarded as a type of the constitutional lack of resistance and supersensitiveness.

Furthermore, the seat and location of the disease may also bring about supersensibility, a supersensibility which is excited by the so-called ' local reaction '. We are indebted to the master-mind of Robert Koch for the first knowledge of this peculiar phenomenon : the well-known tuberculin reaction of the tuberculous focus. Exactly similar reactions may, however, occur when, in a focus filled with parasites, the parasites are rapidly dissolved. Then under the influence of the liberated toxin an irritation of the tissues sets in which is connected with hyperæmia and swelling, and which is known in the case of the ' luetic ' illnesses as ' Jarisch-Herxheimer's reaction '. Such reactions are, of course, of no great importance in connexion with the skin tissues ; but if the reacting centres have their seat in the neighbourhood of vital organs (brain), then

the swelling may perhaps bring about injury of a nature serious to life, or even death itself. Indeed, it is well known that tuberculous meningitis may be influenced in an exceedingly dangerous manner by a careless tuberculin reaction ; in spite of this, however, no one will think of attributing the nervous disturbances to a neurotropia of tuberculin. And exactly the same phenomena may occur with salvarsan also if the spirochætæ of syphilis have localized themselves in the central nervous system.

As you will see, the treatment of patients is an exceedingly difficult and responsible task, and the clinical pioneers such as Schreiber, Wechselmann, Iversen, &c., deserve our warmest thanks. They have thrown the first light upon the most important questions (dosology, indications and counter-indications). From a series of observations, which is now so vast that it can hardly be surveyed, there has, however, resulted what I might call the 'therapeutic tactics', and which I should here like briefly to explain.

(1) The *Therapia sterilisans magna*, which consists in this, that by means of one or at most two injections the body is freed from the parasites. In experiments on animals, and also in the case of a series of important maladies, this principle can be carried through in a clear and pure manner. Here, therefore, the old therapeutic remedy is applicable : ' Frapper fort et frapper vite.' It is a matter of course that the necessary dose must be greater in proportion to the advance of the disease, for it is absolutely clear that if a definite number of parasites is destroyed by a certain definite dose of the remedy, the quantity must be multiplied if the number of parasites—as happens during the course of the infection—has likewise multiplied. Apart from this, by a rapid destruction of the parasites endotoxins are set at liberty which, with increasing numbers of parasites, reach the circulation of the blood in ever-increasing concentration. And then, frequently at the height of the disease, serious or irreparable pathological disturbances occur, such as suppuration or necrosis, which of course continue even when the pathogenic cause has been destroyed, and may lead to unpleasant complications.

I refer here to the typhoid ulcers and to the abscesses and necroses in the case of horse sickness.

Therefore, it is in my opinion necessary to ALLOW THE THERAPEUTIC TREATMENT TO COME INTO ACTION AS EARLY AS POSSIBLE, as under these circumstances the full success is most easily and most surely attainable. And it is just in these present times when, owing to the progress of diagnosis, and especially the modern assistance rendered by the microscope and serological investigations which are connected with the names of Widal, Rubner, and Wassermann, we are in a position to recognize the specific infections early, we must select as the first principle of medical treatment : ' Frapper vite.'

We shall now have to ask ourselves the question, what are the causes which make it possible for such a favourable result to be obtained, a result which may be taken as *Therapia magna sterilisans*, radical cure of the body by means of a single injection ? Typical antibodies can be shown

to be produced fairly rapidly by the destruction of parasites and especially of protozoa. Hence, it is quite evident that this assisting action of the organism ought to be eminently efficacious. For if the medicine has destroyed not the whole of the parasites, but only 95 per cent., and 5 per cent. have resisted its action, then these remaining 5 per cent. are sure to succumb to the influence of the antibodies which are rapidly formed. If this is the case, the *Therapia sterilisans magna* is attained. Unfortunately, it has been shown that this salutary process may frequently be minimized considerably owing to the biological properties of the parasites. For it may happen that a part of the parasites which survive the first injection escape destruction by the serum either wholly or in part, and subsequently change into new varieties which have become serum proof, and which are known as a relapsing crop. The possibility of forming a relapsing crop largely depends on the nature of the parasites. In the case of spirillosis of fowls, which, as is known, is a disease taking a typical course, relapsing crops do not seem to form, and therefore the chemio-therapeutic cure is an eminently easy one. In cases of recurrent or relapsing fever in human beings, the number of the relapsing crops is restricted to three to four. The number of relapses which we have observed clinically corresponds exactly to the existence of the various relapsing strains. On the other hand, other parasites can exist in an extraordinarily great variety of relapsing strains, and in this connexion I need only mention the trypanosomes and, in particular, the parasite of human syphilis. In particular, in collaboration with Dr. Röhl and Frl. Gulbransen, and lately with Dr. Ritz, I have been able in the case of mice to produce, and to transmit for any length of time, eight entirely different forms of growths. It is clear that parasites of this kind which are able to form such a large number of relapsing crops offer very great difficulties in their treatment, as in this case the auxiliary forces of the body fail to act, so that it is necessary to do one's utmost to destroy the whole of the parasites all at once by means of drugs, as owing to their great power of adaptation a single germ surviving may perhaps be the cause of the infection breaking out afresh.

At this juncture the question arises as to what are the causes of some of the germs being able to escape disinfection.

If a certain exactly definable quantity of an antiseptic is added to a liquid containing bacteria (suspension of bacteria), a complete disinfection takes place; not a single germ escapes the destructive influence. However, such ideal conditions do not obtain in living organisms. Even in disinfecting a room we sometimes find that in certain places, in the so-called 'dead corners' formed by piping, water-pipes, &c., the gases of the disinfectant do not act sufficiently. In like manner the parasites which have settled in such 'dead corners' of the organism are not reached by the medicine.

Practical tests have, however, quickly taught us where such 'dead corners' are to be found in the organism; the principal one is the hollow

situated between the spinal cord and the dura, which is filled with a liquid as clear as water and almost entirely free from cells and albumen, the cerebro-spinal fluid. This condition of the cerebro-spinal fluid can only be accounted for by the fact that the cells by which it is secreted are in a high degree impervious to most of the constituents of the organism, for example, albumen, and that they only permit a limited quantity of substances with small molecules to pass through. The drugs with more complex molecules are thus kept back, like albumen, and cannot get into the cerebro-spinal fluid. Should, therefore, parasites be lodged in these places, it is impossible for the drug to attack them. This localization of the parasites is of very special importance in connexion with the parasyphilitic diseases, tabes and paralysis.

Another possible explanation of the deficient sterilization is this, that among the large number of parasites there may be some which are unaffected by certain drugs, thus resisting sterilization. On the whole I am of opinion that this fact does not play a very great part in the course of fresh infection, but that it becomes prominent in connexion with those frequently recurring diseases which are characterized by innumerable relapsing crops, such as sleeping sickness, lues, &c.

Here two possibilities are conceivable: Firstly, if sleeping sickness, for example, is treated with atoxyl in the usual manner it is possible—as can be demonstrated easily by experiment—for an atoxyl-proof stock to form itself by adaption.

But secondly, as I have already mentioned, the mere continual formation of relapsing crops, likewise in the course of time and of several generations, alone can bring about a change in the chemo-receptors of the parasites, which, according to circumstances, may result either in an increased or a reduced power of resistance of the parasites. For instance, I have found that a trypanosome strain, which was not affected by trypan red, lost this peculiarity after having passed into a relapsing crop.

We should, of course, expect that certain relapsing crops, especially those showing a great tendency to relapse, should become much less sensitive than the original stock. And the serious influence of the usual specifics, mercurial and arsenical, upon parasyphilitic diseases, in connexion with which Noguchi has lately proved the presence of living spirilla, tend to show that this is the case here.

These few facts and considerations suffice to indicate in which cases we may rely upon the result of the chemotherapeutic treatment being entirely successful, besides being quick, and in which cases such a result is to be obtained less easily and only by a circuitous road. The latter is especially the case with chronically recurring diseases accompanied by localizations not easily reached. If we compare the fight against parasitic diseases with a state of warfare, we find that, on the one hand, great battles are fought which may lead to victory in the course of one or a few days. In combating bacteria such a victory would compare with *Therapia magna sterilisata*. If, on the other hand, a fortress has to be taken, the

goal cannot be reached with one single stroke, but it may take months and even years.

May I be permitted at this juncture briefly to point out the aids which are employed in connexion with a bacteriological siege :

1. When parasites are lodged in a dead corner and are in consequence difficult to reach, it has frequently been found advisable to employ, instead of one single injection, a long series of injections extending over several weeks, the so-called serial treatment, in accordance with the rule : ' Gutta Cavat Lapidem ', and in this connexion I would specially point to the results which Leredde of Paris and Dreyfus of Frankfort have obtained by these means.

2. In this connexion it is desirable to employ a therapeutic agent of as small a molecular volume as possible, such as urotropin, which has been successfully employed by S. Flexner in cases of infant paralysis.

3. Moreover, various writers (Touton, Duhot, &c.) have suggested rendering the plexus epithelium more pervious to the therapeutic agent by means of certain chemicals, thus causing more of the therapeutic agent to get into the cerebro-spinal fluid. Unfortunately this method so far has not led to any tangible results.

4. The possibility of making direct injections into the cerebro-spinal canal, in order to let more of the therapeutic agent get directly to the parasites contained in the fluid, has also suggested itself. Thus Ayres Kopke of Lisbon has injected suitable disinfectants into the cerebro-spinal canal, while Swift and Moore of the Rockefeller Institute have quite recently adopted a novel and interesting method in the treatment of tabes, which promises to be very valuable. They treat the patient by first injecting salvarsan ; shortly afterwards they draw blood from him and inject considerable quantities of the serum obtained from this into the spinal canal. This method is not only novel but suitable in so far as it obviates all possible ill effects on the sensitive central nervous system by employing serum obtained from the patient himself, while at the same time it is possible to apply the curative in sufficient quantities. The results obtained in this connexion were entirely satisfactory.

It is fairly obvious that all these measures aim on the whole at rendering the places which are not easily accessible more accessible for the therapeutic agent than is the case under ordinary conditions. On the other hand, however, the greater power of resistance of certain parasites has to be taken into account, and this is a purely chemical question which can only be solved by chemical means. The road leading to its solution which promises the best results is that of combined therapy.

From what has been said it will be seen that combined therapy is best carried out with therapeutic agents which attack entirely different chemoreceptors in the parasites. For instance, it is useless to combine euchsin with its nearest relative, methyl violet ; and it is useless to combine therapeutically trypan blue and trypan red, for both attack the same spots in the parasites, but it is necessary to select from each group the

most effective substance and then to combine the most suitable representatives of the various types. It is clear that in this manner a simultaneous and varied attack is directed on the parasites, in accordance with the military maxim, ' march apart but fight combined '.

In this connexion the interesting fact has been brought out that, when such combinations are used *quoad* organism, summation of the toxic properties of the various substances need not occur, whilst, *quoad* parasite, the therapeutic properties can be increased. To quote a case in point, it has been found that a sulphite of *p*-oxyphenylarsenic acid, which is ten times as poisonous as salvarsan, when added to salvarsan up to a certain percentage, did not render the mixture more poisonous. But the healing properties of this mixture were three to four times greater than those of salvarsan in connexion with trypanosome infection in mice. Such phenomena are quite common in connexion with combined therapy. If one injects a substance, as for instance (a) parafuchsine and (b) salvarsan simultaneously, the colouring matter is stored up by other receptors, and finds its way into other organs than does the salvarsan, so that by this method an increase in the toxicity does not take place, provided the components have been properly selected. In this case $\frac{1}{2}$ plus $\frac{1}{2}$ is not equal to 1, *quoad* affection of the organs, but smaller than 1. On the other hand, both substances are concentrated in the parasites, and their effects can consequently be added or, in favourable cases, can be multiplied. We thus obtain the therapeutical inequality $\frac{1}{4}$ plus $\frac{1}{4}$ plus $\frac{1}{4}$ equal to 1.

I have maintained this point for quite a long time, and some very remarkable studies upon the same subject have recently appeared from Morgenroth's laboratory, in which stress is laid on the suitability of multiple, ternary, and quarternary combinations.

Moreover, according to more recent experiments it seems advisable, in all cases where anti-substances are produced, systematically to incorporate these same in the combination, because the anti-substances require a receptor apparatus entirely different in nature from the chemo-receptors. Thus the combination of pneumococcal serum with methylhydrocuprein, as tried by Morgenroth, has proved very efficient in connexion with tests on animals executed by Neufeld, while Dr. Bierbaum discovered simultaneously at my institute that exactly the same holds good in connexion with a combination of dysentery serum and salvarsan.

Thus the value of the combinations is clearly shown, as they make it possible, always assuming that suitable substances are used, to effect a cure with the smallest possible doses and in the least harmful manner, and to eliminate any danger resulting from a maximum dose of one of the ingredients. A further advantage of combined therapy is this, that under the influence of two different medicines, the danger of rendering the parasites immune to arsenic, which naturally would be a very great obstacle in connexion with further treatment, is apparently greatly minimized. Thus it has been shown that, in the course of a prolonged quinine treatment, it may happen—the cases are fortunately not very

frequent—that the malaria parasites become quinine proof, so that therapeutical doses of quinine are no longer able to remove the parasites. If, however, such a patient receives an injection of salvarsan, the malaria parasites are destroyed at once, for although they may be quinine proof, they are not arsenic proof. Should the dose of salvarsan have been too small to prevent a relapse, and provided further quinine is given to the patient on the parasites again appearing in the blood, a curative effect may be obtained by means of quinine. Consequently the combination of quinine and salvarsan has the effect of neutralizing or minimizing the quinine-proof qualities of the parasites.

For all these reasons I think that combined therapy will in the future conquer an ever-increasing field of action. Thus, for instance, Broden, in the Congo, succeeded in connexion with sleeping sickness in the human subject—it is true only in the early stage of this infection, which is so difficult to fight against—in obtaining good results by the combination of salvarsan and two basic colouring matters (trypallavin and tryparosan) by treatment lasting about a week.

It is precisely in the manifold character of the possibilities of combination that I see a special advantage, and peculiar possibilities of development. When once we are acquainted with the majority of the chemoreceptors of a particular kind of parasite, which will be a long piece of work, occupying many hands and heads, we shall have the most far-reaching possibilities of simultaneous attack by various agencies. And on this account combination therapeutics are also absolutely pluralistic in contrast to antitoxins, which may be said to act rather in one single direction.

And now, gentlemen, may I be permitted to refer to a few practical results. You are all aware that with a number of spirillar diseases the principle of *Therapia sterilisans magna* has proved most successful. You are aware that it is possible by one single injection of salvarsan to cure framboesia (yaws), which is also caused by spirochætes, and is a scourge of the tropics, curing it completely except in rare cases where unimportant relapses occur ; this has been shown by the work of Strong, Koch, and Castellani. Thus, in Surinam, a hospital, in which over 300 patients with framboesia were constantly under treatment, was closed and turned to other uses after the introduction of the salvarsan treatment, as one single injection sufficed to cure the disease, and the patients could all be discharged but two. It is to be hoped that in this way it will be possible to altogether extirpate framboesia.

Exactly the same favourable results have been attained with recurring fever in the human subject, the fever immediately subsiding after the injection of salvarsan, and the patients being cured by one injection. The very rare cases of relapse occasionally occurring are also readily curable.

To continue dealing with salvarsan, in syphilis, which is so closely related to framboesia, a fair percentage of cures has been obtained in the

very first stage of the disease by a single injection of a large dose, but of course the abortive cure by intensive treatment is far more certain.

With Vincent's angina and the diseases of the mucous membrane of the mouth, which are caused by spirochætes of the mouth, *Therapia sterilisans magna* is possible ; in fact in many cases a mere local application of salvarsan suffices. I may here further mention tertian malaria, in which form, but in this form alone, salvarsan has proved successful, and blastomycosis (Petersen) and the Aleppo boil. As regards diseases of animals, which can be cured by one single injection of salvarsan, I might specially mention breast diseases of horses, which is of such enormous importance to the military authorities, and lymphangitis epizootica, the African glanders in horses.

Most important are the recent observations of Rogers, who found emetin to be a specific against the very serious amœbic dysentery. And if here it is indeed advisable and necessary to repeat the injections, yet the triumph of therapeutics remains unassailed ; it is all one to the patient as to whether *Therapia sterilisans* or *Sterilisans fractionata* is employed, provided only he is relieved of his sufferings in a harmless manner.

Piroplasmosis also, which exerts a disastrous action, causing serious diseases in cattle and dogs, may according to the observations of Nuttall be favourably influenced by a pigment belonging to the class of trypan colouring matters, viz. by trypan blue, which was first composed by Mesnil. As I am informed the fight against this disease has been taken up in a general manner at Pretoria under the auspices of Theiler. The injections are there performed not by veterinary surgeons, but by the farmers themselves, and they are glad to save their valuable animals scot-free from this serious disease.

It is indeed easy to understand that the schizomycetes, which in themselves are so much hardier than the tender protozoa and spirochætes, will offer an increased resistance to the attack of drugs. Naturally here too there are fine differences, and it is perhaps no accident that the pneumococcus, the protoplasm of which is, of course, most sensitive, should in the course of treatment also have shown itself to be particularly sensitive. (I refer here to the fine researches of Morgenroth in the treatment of laboratory animals infected with the pneumococcus by means of derivatives of quinine, especially ethylhydrocuprin.) But in the case of hardier bacteria, too, such as the Bacillus typhosus, the possibility of sterilization is not beyond hope. The first successful experiments in this sphere were carried out by Conrad on rabbits and later confirmed and extended by Uhlenhuth and his fellow workers on this species of animal.

If I briefly allude to the very hopeful experiments of Gräfin Linden, who has endeavoured to influence tuberculous infections favourably by means of combinations of copper and lecithin, and if I add that salvarsan also has been shown to have a beneficial action upon the malignant anthrax-bacillus, and upon that of glanders and, possibly, upon that of erysipelas, both in animal experiments and occasionally too in human

cases, then all that we know about the chemio-therapeutics of the specific bacterial diseases has been told, so that it is just in this direction that there lies a wide field still to be worked. This field, important as it is, is still in the very first stages of experimentation.

And if after what has been said we cast a glance over the development of medicine and especially of the fight against infectious diseases, we must recognize that in the last fifty years the most important advances have been made in every direction, advances connected above all with the names of Pasteur, Robert Koch, and von Behring.

On the one hand we have the isolation of the pathogenic bacteria, which was made possible really by the Koch method of the solid culture medium and in which Robert Koch's pupils and fellow workers, Löffler, Gafftey, Pfeiffer, in the first order, participated ; the study of protozoa, which started from Laveran's discovery of the germ of malaria ; the discovery by Löffler and Frosch, Roux and Nocard, of the viruses which pass through filters ; and the recognition of insects as intermediate hosts and transmitters of infectious diseases, which is connected with the name of Theobald Smith and which has led to the most important consequences.

On the other hand we have the study of the immunity theory which was first inaugurated so brilliantly by Metchnikoff and which received a new impetus from the wonderful discovery of antitoxin by von Behring, through which a wide new field, that of the science of immunity and the investigation of serums, was opened up, on which Pfeiffer, Bordet, Widal, Wassermann and many others, including myself, have worked with successful result. Some of the most valuable fruits of these labours from a practical point of view have been the diagnosis of diseases first in the form of the Widal-Grüber reaction, and later the Wassermann syphilis reaction, the importance of which for diagnosis and therapeutics cannot be estimated.

All these discoveries, especially in regard to the ways of spreading diseases on the part of the infecting agencies, have, in accordance with the principle that ' Prevention is better than cure ', been made good use of in the fight against epidemics and for prophylactic measures, and have brought about an improvement surpassing expectation. In the second place, the struggle with diseases which have already broken out has been able to derive advantages from these discoveries, the most wonderful example being the diphtheria serum.

Now that the liability to, and danger of, disease are to a great extent circumscribed, so far as epidemics and many other diseases are concerned, the efforts of chemio-therapeutics are directed as far as possible to fill up the gaps left in this ring, more especially to bring healing to diseases in which the natural powers of the organism are insufficient. And I believe that now when definite and sure foundations have been laid for the scientific principles and the method of chemio-therapeutics, the way is visible before us ; not always an easy but yet a practicable way. In the diseases involving protozoa and spirilla extraordinarily

favourable results, as I have shown, have already been gained, which can also satisfy far-reaching tests. There are many valuable indications that in a series of other diseases—small-pox, scarlatina, typhus exanthematicus, perhaps also yellow fever, and, above all, infectious diseases caused by invisible germs—the prospects of success are brightening. But in contra-distinction to these super-parasites the ordinary or common bacterial diseases (diseases due to the streptococcus and the staphylococcus, coli, typhoid and dysentery, but above all tuberculosis) will still require a hard struggle. Nevertheless, I look forward with full confidence to this development also, and might, without being set down as an optimist, put forward the view that in the next five years we shall have advances of the highest importance to record in this field of research. There are indeed problems which often prove too great for the powers of individuals, and can only be solved by a many-sided effort. Considering the enormous number of chemical combinations which are taken into consideration in a struggle with diseases, it will always be a caprice of chance or fortune or of intuition that decides which investigator gets into his hands the substances which turn out to be the very best materials for fighting the diseases, or the basal substances for the discovery of such. But the chances in favour of finding a real cure, and so of winning the big prize, will naturally rise with the number of those who occupy themselves with the definite problem. It is just at this point above all that necessity arises to gather and unite all powers, and here special force attaches to that motto ' Viribus unitis ', which gives guidance in so many other fields : which in so exemplary and fine a way is the foundation of this great International Congress, to which thousands have been drawn from all lands, to give their testimony that in the world of science all national barriers have disappeared.

GENERAL ADDRESS ON

HEREDITY

By Professor W. BATESON, M.A., F.R.S., Director of the John Innes Horticultural Institution; late Professor of Biology in the University of Cambridge

Gentlemen,—Let me on behalf of my colleagues, the students of genetics, express our deep sense of the honour conferred upon us by the organization of your Congress in choosing our science as the subject of one of the general addresses. It is scarcely necessary that I should say on my own behalf that I feel it an extraordinary privilege to be permitted to represent that science on such an occasion.

Introductory Remarks

This is a great privilege, but it entails also a grave responsibility. Conscious as we are of the exceptional significance that the study of heredity must before long assume, it would be affectation were I to suggest any misgiving as to the propriety of allotting to this subject a conspicuous position in your deliberations. To the penetrative foresight of Francis Galton it was evident long ago that these aspects of physiology must one day become one of the chief preoccupations of reflecting minds. That inference he drew from a broad contemplation of the facts of descent. Traces of order among those phenomena he did indeed perceive, and by great labour and ingenuity he even gave numerical expression to his conclusions. But great as his services were in attracting attention to the problem, no one before the rediscovery of Mendel's work had ventured to imagine that the confusion, the paradoxes, the capricious disorder of the phenomena of descent were, in great measure, capable of a simple and ready analysis. It is this knowledge which has given to genetic science a position paramount among the branches of physiology, showing that in accurate genetic analysis a means is given not merely of elucidating the inter-relations of parent and offspring—the immediate subject of our investigations—but of contributing also to a right interpretation of various special problems of pathology and of anthropology, perhaps, also, to a true understanding of the course of human history, and certainly to the direction and control of the destinies of mankind. Knowing, as I must, that the manner in which these issues are presented to you to-day cannot fail to influence in some measure the development of genetic study in the medical world, I should be insensitive did I not feel how responsible is the position I now occupy. Most of all I fear that I may seem in some degree to exaggerate, not the importance of the principles

already ascertained, but the precision with which they can be shown to apply in the special cases of human physiology. In all the arts, perhaps most in the art of exposition, the hardest thing is to simplify the presentation by the omission of detail, and at the same time to leave the picture true to natural fact. So let me, therefore, at the outset say that in regard to almost every instance of principle I shall bring before you, though rule may be perceived, we have evidence of exceptions also. This is true even of the rules traced by minute analysis in the plants and animals amenable to experiment, and necessarily far more true of the human phenomena, which are brought within our reach for the most part by imperfect records. As in every department of physiological advance, we have first to trace the course of those great sweeping curves which lead to the recognition of ascertained law, and later those minor deviations, perhaps equally significant, which must hereafter be the subject of a further analysis.

The Mendelian Principle

Now the essence of Mendelian principle is very easily expressed. It is, first, that in great measure the properties of organisms are due to the presence of distinct, detachable elements, separately transmitted in heredity ; and secondly, that the parent cannot pass on to offspring an element—and consequently the corresponding property—which it does not itself possess. The determination or recognition of these elements by analytical breeding is one of the main objects of present-day genetic research. Each germ cell, ovum, or sperm may contain or be devoid of any of these elements ; and since all ordinary animals and plants arise by the union of two germ cells in fertilization, each resulting individual may obviously receive in fertilization similar elements from both parents or from neither. In these cases the offspring is ' pure '-bred for the presence of the character in question, or for its absence. But it may be formed by the union of dissimilar germs, one containing the element, the other devoid of it, and in this case we call the individual cross-bred, or heterozygous in that respect.

A population thus consists of three classes of individuals, those pure for the presence, having received two doses of an element ; those pure for the absence of the element, having received none of it ; and the cross-breds, which have received one dose only. From the physiological standpoint it is of great importance to distinguish the positive from the negative characteristic. But since it often happens that the full effect is produced in the organism by one dose only of a character, we have felt justified in regarding this positive effect as the sign or proof that it is caused by something present. A plant, for example, though cross-bred for tallness, may be as tall as one pure-bred for tallness. Each dwarf plant, whatever be its parentage, can only produce dwarf offspring. Not having tallness it cannot transmit that property. A cross-bred tall plant can, by self-fertilization, produce both tall and dwarf offspring.

Fowls with silky feathers cannot, if bred together, have offspring with normal feathers, but two birds, normal to all appearance, can, if they be cross-bred in that respect, produce silky offspring.

At a later stage we will consider to what extent we are justified in thus discriminating between the positive and the negative characters, but for the present we will note that, as a matter of symbolical expression, the distinction is immaterial. Now, the organism produces germ cells representing in equal number the characteristics of the germ cells whence it arose. There is, as we call it, segregation between the elements introduced from the parent. The double structure of the soma is, when the germ cells are formed, resolved into the single structures of the germ cells. If the organism is pure-bred in any respect, all its germ cells are alike in that respect. If it is cross-bred, its germ cells represent in equal numbers the positive or negative elements which were brought together in fertilization. In consequence, the numbers of individuals resulting from the various possible forms of union follow regular arithmetical rules, and the proportions in which the several classes appear give us the means of tracing and identifying the various systems of descent.

Conditions showing Dominant Descent

It was naturally with great interest that we began the examination of human pedigrees, and the result of that search soon showed that many of the more definite hereditary diseases and malformations follow one or other of the systems with which Mendelian analysis has familiarized us. The first condition thus recognized to have a simple Mendelian inheritance was the peculiar malformation known as brachydactyly described in America by Farabee. Another example is congenital cataract studied by Nettleship, to whose admirable researches we owe many of our most valuable and instructive pedigrees. Both these conditions descend as dominants. It is characteristic of them that unaffected members of the families do not transmit. Not having in them the critical element which causes the condition, they cannot pass it on to their posterity. In the human examples the individuals affected are almost always heterozygous, and hence among the children which are born to their marriages with normal persons we expect the affected and unaffected to be in equal numbers.[1]

Since then such pedigrees have been recognized in abundance. The collection of such evidence we owe especially to Nettleship, Gossage, Drinkwater, Lundborg, Weil, Bulloch, and to many more also. Some of the conditions, which may be stated with fair confidence to descend commonly as dominants, are the following: Brachydactyly, claw-shaped extremities, cartilaginous exostoses, membranous craniocleido-dysostosis,

[1] Marriages of such affected individuals are necessarily rare. For cases see Nettleship, *Trans. Ophth. Soc.*, XXVIII. 1908, p. 220, and Pick, *Deut. Med. Wochenschr.*, 1912, No. 11.

hypoplasia of the teeth, diabetes insipidus, chronic trophœdema (generally).

Skin affections: Tylosis, epidermolysis bullosa, teleangiectasis, hypotrichosis, porokeratosis, and xanthoma.

Ophthalmic diseases: Pre-senile cataract, ectopia lentis (sometimes), coloboma (sometimes), distichiasis, night-blindness (certain forms), and retinitis pigmentosa (sometimes).

Nervous diseases: Angio-neurotic œdema, tremor hereditarius, Huntington's chorea, ptosis, and probably several of the forms of spastic paralysis and of myo-atrophy.

Respecting nearly all these it may be remarked that exceptional cases, many undoubtedly authentic, are on record in which affected children have been born to parents actually or apparently normal. Many of these exceptions are genuine and serious, but others may be ascribed to irregularities in the age of incidence, and to mistakes as to slight cases.

Another difficulty arises from the fact that the numbers are frequently irregular. This is in part caused by imperfection in the records. In part, however, I think these aberrations point to real peculiarities in the process of segregation. We know well that in certain plants like wheat and peas the expected numbers appear with great constancy, but in others, as stocks and the primulas, long 'runs' are so common that smooth results are only obtained by the summation of large series of observations. We are perhaps right in conceiving the germinal tissue of a heterozygote as an emulsion of the two types, sometimes coarse, sometimes fine, and the degree of smoothness of the results may be an indication of its state in this respect.

In regard to a large group of diseases of nervous origin, such as Friedreich's disease, Thomsen's disease, and others, these sources of difficulty are especially serious, and though from study of many genealogies I have come to the conclusion that most of them are essentially dominants, I offer this opinion with reserve.

Recessive Conditions

Of recessive conditions in man we have less abundant evidence. Inasmuch as they usually appear from the unions of parents both apparently normal, though heterozygous for the condition, their occurrence is rare and sporadic. Lundborg has shown with great probability that paralysis agitans is one of these. The constant intermarriage of families in the valleys of Sweden, Norway, and the Alps gives the best opportunity for the study of this form of descent. Lately, also, the American students of genetics have produced evidence making it clear that feeble-mindedness has at least one of the marked features of a recessive condition. When both parents are feeble-minded they have no normal children. It is nevertheless difficult to regard this condition as a simple recessive, for unions of the feeble-minded with normal parents almost always produce some feeble-minded children. Among our experimental families of plants

and animals there are mysterious examples somewhat comparable with those, but the exact nature of the complication is still obscure.

Other conditions which exhibit a behaviour characteristic of recessives are albinism, myoclonus, epilepsy, and alkaptonuria. They may all appear in the children of normal persons, with special frequency as the result of marriages of related parents, and there can be no reasonable doubt that these conditions are due to the loss of some factor present in normal persons.

At this stage attention should be called to a remarkable group of cases in which the appearance of abnormality may almost without doubt be regarded as partially dependent on the conditions to which the soma is subjected. Of these I may mention in particular some of the congenital abnormalities, such as hallux valgus, and some of the congenital deformities of the joints, as, for example, dislocation of the hip or of the radius. In these cases we meet the curious fact that whereas the condition most usually appears in the offspring of normal parents, it may, in certain families, be transmitted from parent to offspring as a dominant. I am inclined to compare these phenomena with those so often witnessed in incubating eggs. When the incubators are mismanaged many of the chickens are born with deformed feet. Such abnormality, however, is found with especial frequency in particular strains of birds, though eggs from other strains exposed to the same conditions may give perfectly normal results. The liability is the thing transmitted, but without the appropriate conditions the effect is not produced.

Descent of Sex-limited Type

The next group of cases to which I would call your attention is one in which the descent of the abnormality is limited by sex. Of these the best known examples are those of the descent of colour-blindness, hæmophilia, and one of the forms of nystagmus.

A large pedigree of peroneal atrophy collected by Herringham shows a similar descent, and it has also been several times recorded for pseudo-hypertrophic muscular paralysis.

In regard to colour-blindness, the fact has long been known that males are affected with exceptional frequency. Colour-blindness among women is decidedly rare. Horner was, I think, the first to call attention to the fact that in the pedigrees of this condition there is remarkable regularity. Normal women transmit the affection to their sons, whereas colour-blind males with rare exceptions have only normal offspring. It is possible to interpret these phenomena by the suggestion to which several of us inclined some years ago, that colour-blindness is a dominant, inasmuch as the normal males cannot transmit it. We were led to regard the condition as due to the presence of something inhibiting normal colour sense rather than to the absence of the factor to which that sense is due. Further investigation has, however, thrown doubt on this simple account. As to the descent, however, there is no doubt

that the sons of colour-blind males do not inherit the peculiarity, and therefore cannot transmit it. The daughters of colour-blind fathers inherit it, and though it does not appear in them, probably all of them have the power of transmitting it to their sons. Knowledge derived from direct experimental breeding of various animals has provided a very remarkable scheme by which the essential feature in this course of descent can be represented factorially. One of the simplest of such examples is that of the inheritance of colour in Sebright bantams. When a cross is made between a golden hen and a silver cock, all the chickens come silver, but when a reciprocal cross is made, a silver hen being mated with a golden cock, the pullets come golden but the cockerels come silver. In this case we must regard the silver as dependent on the presence of a dominant factor, and the golden as due to its absence. The silver male is pure for silver SS, having received that factor from both parents, but no pure silver hen exists. The hen is heterozygous for silver and may be represented as Ss. We must further regard the hen as also heterozygous for femaleness, Ff. In the formation of the eggs there is what we call repulsion between the factor F and the factor S; the two are not present together in any egg. Those eggs which are destined to become males carry factor S, and those eggs which are destined to become females carry factor F. Now in colour-blindness the repulsion that has been so clearly proved between these factors may be represented as acting in the germ cells of the male between the factor for maleness and the factor for colour-blindness; such that the sperms destined to become males carry factor M but do not carry the factor for colour-blindness, which passes entirely into those sperms which are destined to produce females.

It is evident that females heterozygous for colour-blindness are not colour-blind, and that colour-blind females can only be produced by the meeting of two germ cells both bearing the affection. It follows from the hypothesis that the sons of colour-blind women will all be colour-blind, and all the records of such families with which I am acquainted, with a single doubtful exception, are in agreement, in that the sons of such women were all colour-blind. It is practically certain that the same system of descent holds for a form of nystagmus, respecting which Nettleship has recently collected some important genealogies. As to the other sex-limited conditions in man I can only say a few words. Hæmophilia is the most familiarly known of these. It is likely that the condition in men follows the same descent as that established in the cases of colour-blindness, but inasmuch as males affected with the condition commonly do not live to the age of puberty, little is known of their powers of transmission.

I have already spoken of night-blindness as a condition which descends as a dominant, but it is very interesting that Nettleship has lately published certain pedigrees of night-blindness in which the descent is of the sex-limited type. This particular night-blindness is apparently

associated always with a high degree of myopia, and I suppose there can be little doubt that the recognition of the genetic distinction will lead to the discrimination of pathological differences also.

The Question of Inhibiting Factors and the Origin of Dominants

At an earlier stage of this lecture I mentioned a doubt as to the justice of the inference that the characteristics which behave as dominants are due to the presence of factors, and to that point I must now return. In our experimental studies of animals and plants we are aware that certain negative characteristics may be produced not by the absence of a quality, but by the presence of some factor which inhibits its expression. In illustration I may give as an example the Chinese primrose. These plants may have their flowers coloured or white, and we are aware that the whites may owe their whiteness in certain cases to the absence of colour—in which case, on a cross with the coloured form, the offspring are all coloured ; whereas other whites contain something which destroys or inhibits the development of pigment factors contained in them.

In view of these examples the question fairly arises whether some of the dominants whose descent I have traced in human pedigrees may not in reality be due to the absence of some factor which in normal persons prevents such development. This suggestion was prominently brought forward by Doncaster in regard to colour-blindness. He lately suggested a scheme according to which we should regard all normal males of the population as heterozygous, Nn, for the factor N normality, and all ordinary females as homozygous, NN, for the same factor. In either case the course of the descent would, both qualitatively and numerically, be the same. Between these two methods of representation we cannot as yet distinguish. The point is of great importance in another respect. The question will be prominent in most of your minds, whence do these dominant factors come ? Whereas it is comparatively easy to conceive some process by which some elements of the composition may be lost, we are quite unable to suggest any source from which a factor may be derived, or any mode by which it may be taken into the genetic system of the organism. We have been accustomed to speak of factors as being added to, or subtracted from, the sum total of an individual composition, but as to the actual origin of new dominants there is very little contemporary evidence.

In the case of those animals, such as the fowl, the wild original of which we think we know, many factors exist in our domesticated breeds which are not possessed by any of the wild species. No wild species, for example, has the complicated combs that we know in our domesticated breeds. None has the dominant white colour of the Leghorn ; and many other such instances might be given. How have these elements been added to the composition of the fowl ?

The problem is exactly parallel to that constituted by the occurrence of brachydactyly or night-blindness in man. We can only trace back

these peculiarities to a single individual of whose origin nothing is known, but whence did he derive the factor to which he owes his peculiarity? We may compare these cases with those of the origin of an infectious disease, such as typhoid fever. When a medical officer of health recognizes typhoid in his district he knows that it must have come from some definite source of infection, and but for one feature in the descent of dominants we might be tempted to suggest that they also arose through the introduction of some element from without into the system. I see no *a priori* impossibility in such a belief, but in these dominants we know that the distribution among the germ cells is approximately symmetrical, and nothing that we know of animals and plants justifies the suggestion that a foreign element can be so treated in gametogenesis. Perverse as such a suggestion may appear, I do not think we should close our minds to the possibility that these dominants arise by a process of loss of some inhibiting factor. Until we have some far more direct method of recognizing the presence of factors this suggestion cannot be positively gainsaid.

Let me call your attention also to the influence which this suggestion would have on our conceptions of evolution. We might extend the same reasoning to all cases of genetic variation, and thus conceive of all alike as due to loss of elements present in the original complex. For all practical purposes of symbolic expression we can still continue to use in our analyses the modes of representation hitherto adopted, but we must not, merely on the ground of apparent perversity, refuse to admit that the lines of argument here indicated may prove sound.

Inheritance of Normal Characteristics

Much as we now know of the inheritance of these abnormal characters, some common, others rare, we know little of the descent of the normal characteristics of an ordinary human population. Indeed, the only characteristics as to the genetic nature of which we have clear evidence are the colour of the iris and, to some extent, the colour of the hair. Our knowledge of these is derived from the work of Hurst; he it was who first succeeded in determining the inheritance of eye colour, which had long been a puzzle to anthropologists. The facts which he collected clearly showed that the presence of pigment on the front of the iris—the condition commonly known as brown or black eyes—is a dominant, whereas the absence of this pigment—the eyes we variously call grey or blue—is a recessive. And so far, though I anticipate the possibility of exceptions, no example has yet been produced of two parents critically known to have blue eyes producing offspring with brown or black eyes. As similarly shown, red hair behaves as a recessive, and I understand that at the forthcoming meeting of the British Association in Birmingham he will arrange a demonstration of this as exemplified by the inhabitants of a Midland village.

From the records of portraits of the Royal House of Hapsburg it is clear that the familiar facial peculiarities of the family behave with fair regularity as a dominant, and there are indications that several of the artistic gifts follow with some accuracy the course characteristic of recessives. By careful observation and record it would undoubtedly be easy to extend the list.

Descent of Insanity and other Conditions

Referring to the descent of various forms of insanity nothing has been made out with certainty with the exceptions that I have mentioned: hereditary chorea, which behaves as a dominant, and the condition somewhat vaguely described as feeble-mindedness, which behaves as a recessive. The genetic analysis of insanity is at the present time practically impossible from the fact that the diagnosis of the various forms of insanity is by no means clear, and also from the fact that the conditions of life have obviously much to do with the development or suppression of such weaknesses. Similar difficulties arise in regard to deaf-mutism. It is not in question that this condition is very often transmitted, but forms of deafness are so various, and there is such uncertainty as to the nature of the condition in any given case, that we cannot expect to perceive the operation of any regular system here. It is not impossible, also, that in regard to the descent of liability to infectious disease we may hereafter trace in man rules similar to those which have now been established in certain plants, especially by Biffen.

The Nature and Time of Segregation

I propose now briefly to allude to some other significant aspects of these results. First, as to the nature of segregation. This, I think, we must regard as a process comparable with the mechanical separation of substances which will not mix, or mix imperfectly; whereas some factors are continually transmitted in their entirety, others are liable to be broken up by what I regard as a process of quantitative fractionation occurring in the mechanical dissociation of the elements at certain critical cell divisions. As to which are the critical cell divisions we have no clear indication. I cannot agree with those of my colleagues who think segregation must occur exclusively in the maturation processes. The case of the single stocks which throw doubles, where the whole male side of the plant differs genetically from the female side, as proved by Miss Saunders, shows almost conclusively that segregation may occur in somatic divisions. It is also difficult otherwise to interpret the fact that in certain cases the parental combination influences the distribution of factors among the gametes, so that the distribution among the grandchildren is different according to the way in which the characters were combined in their grandparents. Into these details I cannot now enter.

The Definiteness and Fixity of the Laws of Descent

The tendency of our work, as you will perceive, is more and more to exhibit the definiteness and fixity of the laws of descent. The medical man, justly proud of what he can do to make good bodily shortcomings, looks naturally to the conditions of life as the influences which chiefly determine destiny. The statesman, to whom physiological fact is a mystery that he rarely feels any desire to explore, inclines naturally to a similar conception of life. There are signs that more true views are beginning to prevail. It is impossible to study such pedigrees as that of night-blindness, first compiled by Cunier and lately extended by Nettleship, in which a condition introduced by one individual into a community hundreds of years ago continues to be perpetuated according to definite arithmetical rules among a remote posterity, without being impressed with the fact that whatever influences may be brought to bear by hygiene or by education the ultimate decision rests with the germ cells. Evolutionary change is effected not so much by gradual transformation of masses under ameliorating or detrimental conditions, but in the main by the occurrence of individual and sporadic variation.

Eugenics and Legislation

It is beyond my province to discuss in detail the practical question denoted by the word eugenics. Those who are engaged in the work of physiological analysis probably do well to keep clear of those distractions. The direction, however, in which genetic research points is not difficult to determine. Sir James Crichton-Browne has discussed at this Congress the striking percentage increase of lunacy in recent decades, and, as always when such topics are under consideration, there are persons disposed to lay the blame on the conditions of life, the severity of the modern struggle, a greater or less consumption of alcohol or other drugs, and so forth; those, however, who have some knowledge of genetic physiology are aware that the whole force of modern science and legislation has hitherto been exercised in the preservation of defective strains in our midst, and will not feel serious hesitation as to the true cause of the increase.

The Mental Deficiency Bill we recognize as, in principle, a wise beginning of reform, but, on the other hand, we cannot hear without disquietude of the violent measures that are being adopted in certain parts of the United States with similar objects. It is one thing to check reproduction of hopeless defectives, but another to organize a wholesale tampering with the structure of the population, such as will follow if any marriage not regarded by officials as eugenic is liable to prohibition. This measure, we are told, is actually proposed in some of the States in America. Nothing yet ascertained by genetic science justifies such a course, and we may well wonder how genius and the arts will fare in a community constructed according to the ideals of State Legislatures.

Philologists tell us that by an irony of development the word ' dull ' comes from the same original which in Dutch has become ' dol '—mad, and is better known to most of us in the German equivalent ' toll '. But I anticipate that, connected as the ideas may be, we might by ridding our community of mania leave it gravely infected with dullness. There are other no less obvious considerations which I might develop, but I trust I have said enough to show that in the experimental analysis of genetics we have an instrument of novel and extraordinary power, and I am not alone in foreseeing that genetic science must profoundly influence the course of human thought and ultimately the conduct of society.

GENERAL ADDRESS ON

PUBLIC HEALTH

By The Right Honourable JOHN BURNS, M.P., President of the Local Government Board

THE RELATIONSHIP BETWEEN MEDICINE AND PUBLIC HEALTH

The past saving of life.
The expenditure of local authorities.
Periods of progress.
Motives of progress.
The doctrine that dirt causes disease and its triumphs.
    Typhus fever.
    Enteric fever.
Stages in the registration of disease.
The scientific period of disease prevention.
    Pasteur.
    Lister and surgery.
    Semmelweis and puerperal fever.
    Koch and tuberculosis.
    Factors in reduction of tuberculosis.
    A partial antidote to urbanization.
    Further possibilities of progress.
    Alcoholism and disease.
The application of science in medical practice.
The ever increasing institutional treatment of disease.
The rôle of the nurse.
National interdependence in relation to health.

I much appreciate this opportunity of addressing an audience which is representative of the medical culture of the entire civilized world: an audience of men who have assembled to interchange experience, to report progress, and to give and receive mutual enlightenment in their glorious work for the benefit of suffering humanity.

My intention in addressing you to-day is in the first place to bear my testimony to the immense debt of gratitude owing to you by the whole of humanity. Arising out of this, my remarks will in the main be concerned with the intimate relationship which exists between medicine and public health.

When speaking of the marvellous reduction that has taken place in the death-rate in this country, one is perhaps too apt to remember only

social and sanitary progress as the explanation of this great change. My address to you to-day may, I trust, serve to show the appreciation by the public of the fact that to a very large extent humanity is indebted for the saving of life and of suffering that has occurred to the vast improvements in the science of cure as well as of prevention of disease.

As a layman, I cannot attempt to set out the details of the marvellous progress of medicine, especially in its relation to public health, since you last met in London in 1881, and in the years preceding that meeting. Were I to attempt that task not a single nationality represented here would fail to claim names of honour in the record. My imperfect list can give only a few of the names that will be familiar to you; Norway has had its Hansen, Russia its Pawlov and Metschnikoff (lent to France), Austro-Hungary its Semmelweis, France its Pasteur, Budin, and Roux, Italy its Celli and Machiafava, Belgium its Bordet, Holland its Eijkman, Japan its Takaki and Kitasato, Germany has Ehrlich and Koch, America its Oliver Wendell Holmes, Morton, Read, Carroll, and Flexner, and the United Kingdom its Farr, Simpson, Jenner, Lister, Manson, and Ross.

I do not forget that I am here as the representative of public health in this country, and my remarks will be made chiefly from that standpoint. Although no impending triumphs of preventive medicine are likely to cause your glorious work in assuaging suffering to cease, I look forward to the time, which has indeed already partially come, when the family doctor will be engaged in advising as to means for preventing disease, in dissuading from habits inimical to health, in preventing overwork or laziness—both serious enemies of mankind,—in reporting external conditions, whether of work or leisure, needing to be amended, and still more often in discovering the early symptoms of illness which if neglected may produce serious disease, and in securing the removal of their cause.

We have, already, glimpses of important directions in which this ideal of future work is being realized. I refer to the modern campaigns against tuberculosis, in aid of child welfare, and in connexion with school medical inspection, which are rapidly becoming part of the national life in this and in other countries.

The national system of medical inspection of scholars, and the 'following up' of disease or disorder discovered at these inspections, is a work of the highest national importance. It is not only important in itself, but also in discovering a large amount of neglected illness, which should have received attention before school-life, and to which child-welfare work is now being increasingly directed. In all these relationships we are realizing to an increasing extent the essential continuity and interchangeability between treatment and prevention, in our efforts to reduce disabling disease.

The past saving of life. Some conception of the progress already secured by the application of science, especially medical and sanitary science, to the problem of healthy living—and I trust at the same time

some impetus to further triumphs on the part of your profession—may be given by the comparison to which I invite your consideration of the average experience of England and Wales during the three years 1909–11 as compared with its experience during three years based on the average experience of 1871–80.

In the three years 1909–11, 1,529,060 deaths occurred in England and Wales. This number is 772,811 fewer than would have occurred had the average death-rate of 1871–80 held good for these three years. As shown in the following figures, the largest share of this saving of life during three years has occurred in the working years of life, and the gain thus secured to the economic capacity of the nation has been on a gigantic scale. This saving of nearly 800,000 lives in three years is about equivalent to the population of New Zealand.

Comparing the aggregate experience of the three years 1909–11 in England and Wales with the experience of three years based on the average death-rate for the decennium 1871–80 :

The saving of life in these 3 years at ages				0–1	was over	98,000	persons
,,	,,	,,	,,	1–5	,,	126,000	,,
,,	,,	,,	,,	5–15	,,	44,000	,,
,,	,,	,,	,,	15–25	,,	58,000	,,
,,	,,	,,	,,	25–35	,,	78,000	,,
,,	,,	,,	,,	35–45	,,	77,000	,,
,,	,,	,,	,,	45–55	,,	50,000	,,
,,	,,	,,	,,	55 and over	,,	114,000	,,

The saving under certain special diseases is set out below. Comparing the aggregate experience of the three years 1909–11 with the experience of three years of the decennium 1871–80 :

The saving of life in 3 years in respect of				small-pox	was	25,463
,,	,,	,,	,,	measles	,,	7,824
,,	,,	,,	,,	scarlet fever	,,	69,974
,,	,,	,,	,,	whooping-cough	,,	30,884
,,	,,	,,	,,	fevers (typhus, enteric, simple continued fever)	,,	45,339
,,	,,	,,	,,	puerperal fever	,,	3,941
,,	,,	,,	,,	diarrhœa, dysentery, and cholera	,,	32,996
,,	,,	,,	,,	pulmonary tuberculosis	,,	114,799
,,	,,	,,	,,	other tuberculous diseases	,,	36,338
				Total saving on these diseases		367,558

Nearly half the total saving has occurred under the heading of the diseases here enumerated.

If we take the whole of the thirty-two years 1881 to 1912, and consider the saving of life during this period, the figures are truly colossal. During these thirty-two years, in England and Wales 17,083,751 deaths occurred.

Had the relatively high death-rates of 1871–80 continued during each of these thirty-two years with the populations of each of these years, 3,942,000 more deaths would have occurred than were actually experienced. This saving of life represents a population which is nearly

equal to the total population of London, or Australia, or of Ireland, and more than that of Switzerland.

Expenditure of local authorities. It is interesting to contrast the saving of life actually experienced during the last thirty-two years with the expenditure incurred by local authorities (sanitary and poor-law authorities, county councils, joint hospital boards, &c.) during the forty years 1870 to 1909–10. During this period the total expenditure of all local authorities in England and Wales exceeded 3,000 millions sterling, of which amount over 500 millions was obtained on loan since the year 1882–3. At the end of 1909–10 the total indebtedness of local authorities exceeded 536 millions sterling, or about seven-tenths of the amount of the National Debt.

In the following table an analysis is made of the main items of loans by local authorities which may be regarded as directly or indirectly concerns of public health, and a comparison is made with the corresponding items for 1883–4. The figures are stated in round numbers.

	1883–4	1909–10
	£	£
Total outstanding debt for all purposes	165,000,000	536,000,000
Outstanding debt incurred in respect of schools	14,000,000	46,000,000
,, ,, ,, highways, &c.	17,000,000	57,000,000
,, ,, ,, waterworks	29,000,000	127,000,000 [1]
,, ,, ,, sewerage and sewage disposal	15,000,000	42,000,000
,, ,, ,, housing	3,500,000	10,800,000
,, ,, ,, hospitals	182,000	4,600,000 [2]

The above statement does not include municipal and poor-law expenditure on the supply of gas, electricity, tramways, harbours, on asylums, police, markets, workhouses, &c.

Since 1882–3 local authorities have expended out of rates and by means of loans the following sums:

	£
Education	320,000,000
Highways, bridges, and ferries	289,000,000
Waterworks	131,000,000
Sewerage and sewage disposal	89,000,000
Hospitals (not including Metropolitan Asylums Board and Workhouse Infirmaries)	20,000,000
Housing of working classes	11,000,000
	£860,000,000

A large proportion of the total sums mentioned above has been expended by local authorities on objects which are unrelated to the public health, though they may have increased the public comfort. The expenditure on education has doubtless helped the public health as well as national efficiency, though only a share of this expenditure should be allotted to the former heading. If we assume that 500 millions sterling have been expended which can be rightly associated with the saving of

[1] Including loans of Metropolitan Water Board.

[2] Does not include Poor Law Institutions or Metropolitan Asylums Board Institutions.

nearly four million lives which has occurred during the last thirty-two years, and if we take the average economic value of a life saved, i.e. prolonged to an older age, to be £150, a very modest estimate, evidently even this large sum can be regarded as having been a profitable investment. It has to be remembered that the benefit of many of the large capital expenditures on water-supplies, sewerage, housing, hospitals, &c., will be reaped even more in the future than in the record of the past.

Periods of progress. How has the saving of life already achieved been secured? No complete answer can be given in a few sentences, and perhaps my best plan is to proceed by examples, drawing inferences from the historical facts of medicine, which are open to the layman as well as to the doctor.

The progress that has been made in this country during the nineteenth century comes within two overlapping periods. The first of these is the period of what may be called crude generalizations; the second is the period in which progress as to each disease has been based on the actual results of scientific investigation.

Motives of progress. In the progress of the earlier period—and happily the same motives continue to be potent now—motives of philanthropy, panic, and economic necessity combined to secure, though too slowly, the needed sanitary reform. I cannot stay to discuss the high value attaching to the devoted efforts of such men and women as John Howard and Elizabeth Fry, of Sadler, Oastler, and their collaborators, of Lord Shaftesbury, of Charles Dickens, of Robert Owen, of Charles Kingsley, and in official life of Edwin Chadwick, of the Poor-Law Commissioners, who succeeded in arousing England and securing improvement in the conditions of work and life, and even of imprisonment; nor of such men as David Livingstone, who did so much to 'heal the open sore of the world' constituted by slavery, to open up tropical countries and thus to prepare the way for the recent triumphs of tropical medicine. It is not without interest to note that the earliest successful attempts to enforce sanitation and to secure healthy conditions of work were in connexion with factory work. This involved the appointment of paid inspectors, introduced by the Act of 1833. Factory inspectors preceded medical officers of health and sanitary inspectors; and under the Home Office their beneficent work in industrial hygiene has, year by year, become more far-reaching and effective.

Next to philanthropy, panic has been, perhaps, the most efficient factor in early sanitary progress. So far as this country is concerned, cholera has been far from an unmixed evil. In 1831–2 it caused 41,544 deaths in the United Kingdom; and this led to spasmodic efforts on the part of many local boards of health to sweep the Augean stables of insalubrity. The lesson was not, however, sufficiently learnt until later epidemics of the same disease occurred, followed by the creation of local sanitary authorities, and the expenditure of large sums on schemes of water-supply and sewerage.

The economic value of health and the wastefulness of premature sickness and death are not even now sufficiently realized; but the steadily increasing readiness of local authorities to expend money on sanitary measures indicates our increasing realization of the truth that national health is national wealth.

The doctrine that dirt makes disease. It was the great merit of the work of Edwin Chadwick that he added to philanthropy the important economic motive, showing through numerous local investigations the regular association between unhealthy conditions and disease, and the production of pauperism by means of disease. Of the value of this generalization we are now fully advised. At least one-half of the total cost of pauperism in recent years is stated to be swallowed up in direct dealing with sickness, and to this must be added the indirect contributions of sickness, through deaths of bread-winners, and the host of by-products of sickness with which disease helps to populate our workhouses.

Chadwick was largely influenced by the teaching of Dr. Southwood Smith and his collaborators, that epidemic diseases as a whole are the direct consequence of local insanitary conditions. This valuable generalization, as we now know, needs a modified and more accurate statement, specialized for each individual disease. In its original form, however, it embodied a realization of the immense power of the environment to make or mar the national life; it secured the beginning of our national improvements, and it laid the foundation of the house of health which as a nation we are building.

Typhus fever. The value of the teaching of this doctrine is shown in the almost complete annihilation of typhus fever and the rapid decline in amount of enteric fever. The history of typhus is a romance in sanitation, from which the principles and practice of preventive medicine could be adequately taught. It occurred under conditions of dirt and overcrowding; but, apparently, it arose only by direct infection from person to person. It was spread, like small-pox, by vagrants from parish to parish, repeatedly brought by them from Ireland to England, and it was not brought under control until the migrations of vagrants had been limited, the sick had been segregated in hospitals from the healthy, 'contacts' had been kept under adequate supervision, and the houses harbouring the disease had been disinfected and the vermin therein destroyed. In many instances the courts and alleys, the favourite lurking-places of the disease, were also swept away. Consider the following figures: In the ten years 1871–80, in Ireland, 7,495 deaths were returned as due to typhus fever; in the three years 1909–11 the number had fallen to 143. In England and Wales, in the ten years 1871–80, 13,975 deaths were caused by typhus fever; in the three years 1909–11, with a much larger population, the number thus caused was 30.

Enteric fever. The history of enteric fever is also very striking. In England and Wales, in the ten years 1871–80, 78,421 deaths were ascribed

to this disease; in the three years 1909–11, its greater population had 6,447 deaths from the same disease—a number far too large, and in some measure due to the fact that some local sanitary authorities still do not realize to the full extent the importance of the generalization that filth implies disease. The importance of this generalization is shown by the excess of this disease in towns in which privy and ashpit systems continue, and the water-carriage system of sewerage has not been installed.

When our medical officers of health—the advance-guard in our army of sanitation, and our advisers as to the best line of action—came to investigate the facts, they ascertained that explosive epidemics of this disease are nearly always caused by specific excremental contamination of a public water-supply or of a milk-supply; and the knowledge of these facts conveyed to local sanitary authorities throughout the country has induced them to expend enormous sums on public water-supplies and sewerage, and to the adoption of precautions in connexion with our milk-supplies. With more accurate knowledge of the methods of spread of enteric fever, it became clear that the nursing of enteric fever under the home conditions of the poor involved further spread of the disease: and to the use of isolation hospitals must be ascribed a large share in the realized reduction of enteric fever, as also of typhus fever. In recent years it has been shown that there is a residuum of enteric fever, not explicable by origin from the above-mentioned sources; and other smaller sources of infection have been traced, including chronic 'carrier cases'. Contaminated shell-fish, especially mussels and oysters, have been shown to be responsible for a considerable amount of this disease, and I have at the present time under consideration the issue of regulations which will give sanitary authorities additional control over this source of infection. There is reason to expect that enteric fever will ere long be known, in fully civilized countries, like typhus fever, scurvy, and leprosy, as in the main an historical curiosity.

Stages in registration of disease. The history of registration of disease is inseparably associated with that of public health, and it is convenient at this point to refer to this important subject. Until the ugly facts as to excessive mortality under conditions of filth and overcrowding could be persistently obtruded upon the public mind, reform was difficult, sometimes impossible. The need of registration of deaths was realized as an instrument of sanitary progress—the epidemic of cholera in 1831–2 giving a great impetus in this direction—and political considerations aided its enactment.

It is not the least of Chadwick's great services to the State that he discovered William Farr, the humble apothecary, and placed him in charge of our national vital statistics. The series of reports issued by Farr have had an immense influence in promoting the public health. They threw the light of publicity on the towns and parts of towns in which fever and consumption throve; they enabled the moral of great epidemics to be pointed, and painstaking investigations into their causa-

tion to be made by medical officers of health and others. The later reports by Drs. Ogle, Tatham, and Stevenson have worthily continued the series initiated by Farr. In particular the decennial supplements have thrown valuable light on the incidence of industrial mortality.

As time went on it became clear that registration of deaths gave a very imperfect view of the prevalence of disease, and that so far as infectious diseases were concerned, valuable time was lost when preventive action could only be taken after the patient's death. Death registration told of the total wrecks which had occurred during the storm; it gave no information as to early mishaps enabling others to trim their vessels and thus weather through. It gave a list of killed in battle, not of the wounded also.

Progress occurred step by step, in our characteristic British fashion. Pioneer attempts were made by medical men in a number of districts to secure the voluntary notification of different forms of illness. The gradual failure of these attempts led to agitation in favour of the compulsory notification of the chief acute infectious diseases. Local powers for this purpose were secured by a few enterprising towns, in 1889 an Adoptive Act for the whole country was passed, and in 1899, by a further Act, the notification of these diseases was made compulsory throughout England and Wales. From time to time extensions of the list have been made by local authorities and by the Local Government Board; among the most recent additions have been the Orders by which the notification of poliomyelitis and cerebro-spinal fever has been made generally obligatory. It may be hoped that the more precise information thus obtained will throw new light on the conditions governing the spread of these serious diseases.

In a large number of districts ophthalmia of the newly born is already notifiable: and I am about to issue an Order making this disease notifiable in every sanitary area, and enabling sanitary authorities to provide the prompt medical aid and nursing which are required to prevent this serious cause of blindness in childhood.

But you will, I think, agree with me that the most important extension of the principle of notification has been in regard to tuberculosis. The knowledge that this disease is communicable, and the importance, even apart from the fact of communicability, of having exact knowledge of its special haunts, and of its prevalence in different industries, and among the poor living in crowded streets and courts, have enabled me step by step, by means of Departmental Orders, to apply the principle of compulsory notification to all forms of this disease. In order that public opinion might confirm the proposals, and to ensure that notification would be effective as well as merely prescribed, action was deliberately taken in stages.

On December 18, 1908, an Order was issued, making obligatory from January 1, 1909, the notification of cases of pulmonary tuberculosis in poor-law practice. This was followed on March 22, 1911, by an Order

making the notification of cases of pulmonary tuberculosis in hospital practice obligatory from May 1, 1911.

On November 15, 1911, an Order was issued making it obligatory from January 1, 1912, on all medical practitioners throughout England and Wales to notify cases of pulmonary tuberculosis occurring in their practice, whether public or private: and on December 19, 1912, this duty was extended from February 1, 1913, to all forms of tuberculosis.

I have made careful inquiries, both from private practitioners and from hospital physicians, and have been much gratified to find that each step in the extension of notification has been welcomed by them, and that instances in which hardships to patients have occurred *as the result of notification* are very few indeed.

This is due in part to the fact that notification is confidential in character, but also, and I believe chiefly, because the notification has not only enabled investigation of sources of infection to be made and precautionary measures to be adopted, but has also put the patient in the way to receive more efficient treatment. Tuberculosis is the one disease in which the fact that measures of treatment and of prevention are to a large extent identical is becoming fully realized; and the important endowment of the treatment of this disease in connexion with the National Insurance Act will enable local authorities and their officers to take active measures both of prevention and treatment on a larger scale than had been previously practicable.

During the last few years a weekly list of cases of certain important diseases in foreign ports has been distributed by the Local Government Board to port medical officers of health, thus giving them warning of the points from which the importation of disease is particularly to be feared.

By an Order of the Local Government Board, dated December 13, 1910, every medical officer of health has been required to send to the Board at the end of each week a list of the cases of the different infectious diseases notified in his area during the week; and during the following week a complete statement of these cases in each and all the 1,800 sanitary areas in England and Wales is distributed by the Board of which I am the head to each of the medical officers of health of these areas for their information.

For 1911 the first statistical return was prepared in my Medical Department showing the rate of sickness for each notifiable disease in every sanitary area; and by this means there is now accumulating a more accurate record of the sickness of each district than was previously possible. The influence of such information in securing investigation into the sources of disease, and in enabling pressure to be brought to bear on reluctant or obstructive authorities will be very great.

No mention of notification would be complete which omitted reference to the Notification of Births Act. This Act has enabled medical officers of health to obtain the information as to births within thirty-six hours of their occurrence, whereas registration may be delayed for six weeks;

and by this means early visits can be made in suitable cases by means of nurses or health visitors, competent to give useful advice. I am hopeful that this Act will ere long be adopted in every sanitary area. Already it applies to more than half of the total population of England and Wales.

I must hasten on to make a few remarks on what I have called the scientific period of disease prevention. Historically, this cannot be separated from the earlier period of cruder, though most valuable, generalizations, in which already valuable work was being done by local sanitary authorities, under the guidance of their medical officers of health. The old generalizations, embodied in our warfare against overcrowding, uncleanliness, the herding of families in unsatisfactory houses, against impure water-supplies, against accumulated filth in and about the dwelling, against dusty occupations, retain their full value, and the warfare against these evils still needs to be pursued with unabated energy. Each year, however, our knowledge of the causation of individual diseases is becoming more exact ; and our warfare against disease can be pursued not only with what may be described as the bludgeon, but also with arms of exactitude not previously available.

Louis Pasteur. The pioneer of this period, who showed the way along which you are now so successfully travelling, was one who was on the platform of the meeting of this International Congress in August 1881, when last it met in London. He was then your most honoured member, and the immense possibilities of future prevention of disease opened out by him were already being realized. Need I say that I refer to Louis Pasteur, a name to be ever held in reverence as that of the father of scientific preventive medicine ? Although himself originally a chemist, he has exercised a greater influence on medicine than any other man since Harvey. He succeeded in destroying the idea of spontaneous generation, and in establishing on a firm basis the germ theory of disease. As has been well said, he associated the faith of an apostle with the inquiring patience of a scientist. With indomitable courage he pursued his investigations, undeterred by obstacles and opposition. When his friend said to him, ' Studies of that sort require much courage,' his simple answer was, ' What about duty ? '

It would ill beseem me as a layman to attempt before an audience of medical men, including our distinguished French *confrères*, to describe the work of Pasteur. I like to think of it especially as leading up to and inspiring that supremely important work of Lister, which has revolutionized surgery and saved an untold number of lives.

Lister. May I be excused for dwelling for a moment or two on Lister's work and its results ? Born in 1827, in 1860 he became surgeon at the Glasgow General Infirmary, where, as elsewhere, the surgical wards were ravaged by erysipelas, pyæmia, and hospital gangrene. The introduction of ether as an anæsthetic by Morton in America, and the later introduction of chloroform by Simpson in 1847—although they prevented an enormous amount of suffering—had temporarily increased

the mortality of surgery, by rendering operations painless, and therefore more frequent, in wards in which septic infection prevailed. In those days suppuration was regarded as a normal process, now when it occurs as the result of an operation it is regarded as a grave reflection on the surgeon in whose practice it appears. Applying Pasteur's discovery that putrefactive fermentation is due to microbes introduced from without, Lister, by antiseptic methods, without altered sanitation of his wards, so improved their record that in 1867 he was able to report that during the preceding nine months not a single case of erysipelas, pyæmia, or gangrene had occurred among his patients.

Listerism. It is impossible to express statistically the saving of life and suffering effected through Lister's work, but its magnitude is well known to all of you. Operations which in former times were needed to save life, were commonly followed by sepsis and death. A major operation for a compound fracture or other serious injury, often meant merely the postponement for a few days of the fatal result. In many hospitals, and sometimes for long periods, each surgical patient in succession was liable to be attacked by erysipelas, pyæmia, or gangrene. Little wonder, then, that hospitals were regarded as death-traps, and an operation in the poorest home often had a greater prospect of success than a similar operation by a distinguished surgeon in our largest and best hospitals. The hospital and its architect were commonly blamed for this, and the fact that death lurked in the skin of the patient himself, in the finger-nails of the surgeon, and in the sponges of the nurse, was unrealized. Listerism has changed all this. It has done more. It has made possible triumphs of cure not previously dreamt of. Abdominal diseases and diseases of the brain and its coverings, formerly inevitably fatal, have come within the range of treatment, and the possibilities of saving and prolonging life have been immensely increased. The following comparison of the experience of two of the largest metropolitan hospitals shows the greater extent to which operative assistance can now be given to patients. In 1878, 15·2 per cent. of the total surgical patients in these hospitals (4,531 in number), and in 1911, 79·1 per cent. of the total surgical patients in these hospitals (6,490 in number), received operative treatment.

As a by-product of Listerism there has been an almost incalculable saving of pain, directly in connexion with the progress of injuries and operations, and indirectly in consequence of the number of painful conditions which can now be attacked by operative measures. We may therefore add the name of Lister to those of pain-destroyers, which include also Morton and Simpson, the introducers of ether and chloroform respectively as anæsthetics, and the modern chemists who have introduced the wonderful series of drugs producing local anæsthesia.

Semmelweis. From Listerism we may pass on to mention puerperal fever, a specially urgent case for the application of Listerism, an application which—if the teaching of Semmelweis had been appreciated—would have been adopted before Lister. As early as 1847, Semmelweis demon-

strated that puerperal fever was caused in the lying-in wards in his clinic at Vienna by contact infection, brought by students who were also engaged in the post-mortem or dissecting room, or in surgical wards in which there were open wounds. In 1849 Semmelweis's work was brought before a London Medical Society. Unhappily its importance was not realized, and so the triumph of antisepticism and asepticism in midwifery was postponed; for the child-bearing mother the full realization of freedom from risk of sepsis, which is a fair claim for every expectant mother to make, is still far from being realized. Our national figures show improvement in the death-rate from puerperal sepsis; a less improvement in regard to the accidents and diseases of pregnancy and childbirth. Even in regard to puerperal sepsis alone, the record is not entirely satisfactory.

From puerperal fever and the accidents of childbirth

In the ten years	1877–86	one mother	died	to 226	births
,, ,,	1887–96	,,	,,	197	,,
,, ,,	1897–1906	,,	,,	228	,,
In the year	1907	,,	,,	261	,,
,, ,,	1908	,,	,,	280	,,
,, ,,	1909	,,	,,	270	,,
,, ,,	1910	,,	,,	281	,,
,, ,,	1911	,,	,,	262	,,

This means that an annual saving of upwards of 700 lives of mothers in connexion with childbirth is being secured as compared with the experience of thirty years ago.

So far as London is concerned, I have recently authorized the admission of patients suffering from puerperal fever into the hospitals of the Metropolitan Asylums Board; and there is no reason why, with an improved service of midwives, better nursing, and the strictest asepticism in practice, puerperal fever should not become extremely rare.

Koch. It is difficult to speak of tuberculosis without merely repeating the already familiar. In devoting a few minutes to this important disease, I wish to associate my remarks with the name of Robert Koch, who lifted our knowledge of this disease from the empirical to the scientific. Koch was one of many instances in medicine of genius triumphing over poverty. He was an obscure practitioner in a small country place, where he was able to demonstrate improvements of technique in bacteriology, which opened up a new epoch in this science. In 1882 he proved that tuberculosis is caused by the tubercle bacillus, and demonstrated the bacillus. He caused much searching of heart at the Congress in London in 1901 by his frank expression of opinion that bovine infection had very small importance in the production of human tuberculosis. More recent investigation has shown that bovine infection cannot be disregarded as a cause of tuberculosis in children; but his statement has doubtless led to greater concentration than there would otherwise have been, on measures to prevent infection from man to man. He steadfastly held to the view that tuberculosis in the main is a disease of the dwelling, more particularly of the bedroom.

I cannot express this better than in Koch's own words, uttered in his Nobel lecture in 1906 :

The danger of infection becomes especially great when healthy people have to sleep in the same rooms with sick people, and even, as unfortunately still frequently happens among the poor, in the same bed. This kind of infection has struck attentive observers as so important that tuberculosis has been frankly and justly called a dwelling disease.

In Berlin during the last decade more than 40 per cent. of the cases of pulmonary phthisis died in hospitals. In Stockholm, too, the state of affairs must be very favourable in this respect. The number of patients that are thus placed in circumstances under which they can no longer infect is very considerable and cannot fail to influence the course of the pestilence.

The improvement of the situation of the lower classes and the better knowledge of the danger of infection, the consequence of which is that people no longer unwittingly expose themselves to it, have certainly helped to reduce tuberculosis. But I am firmly convinced that the better provision for patients in the last stage of pulmonary phthisis—namely, the lodging of them in hospitals, which is done in England and in Prussia to a comparatively large extent—has contributed most to the improvement. From this, however, we are to derive the lesson that the greatest stress is to be laid on this measure—namely, *the placing of cases of pulmonary phthisis in suitable establishments*, and far more care should be taken than hitherto that such patients do not die in their dwellings, where, moreover, they are for the most part in a helpless situation and inadequately nursed. If cases of pulmonary phthisis are no longer, as hitherto, rejected by the hospitals as incurable ; if, on the contrary, we offer them the best imaginable nursing gratis and can even in some cases hold forth the hope of cure ; if, moreover, their families are cared for during their illness, not the slightest compulsion will be necessary to induce a still much greater number of these unfortunate patients to go to hospitals than at present.

Factors in reduction of tuberculosis. The causes of the remarkable reduction of tuberculosis in this and some other countries during the last thirty or forty years cannot, however, be stated in a single proposition ; and looked at from various standpoints, diverse statements are possible, each containing a considerable share of the entire truth.

In the three years 1909–11, tuberculosis of all parts of the body caused 151,137 fewer deaths in England and Wales than would have occurred had the experience of 1871–80 been continued.

Comparing the percentage reduction for phthisis and for all non-tuberculous diseases in the aggregate for five yearly periods, from 1876–80 onwards, we obtain the following results :

Percentage reduction (—) or increase (+) between	*Phthisis in males.*	*All causes, omitting tuberculosis, in males.*
1876–80 and 1881–5	—9·8	—6·2
1881–5 and 1886–90	—8·3	—0·5
1886–90 and 1891–5	—9·5	unaltered
1891–5 and 1896–1900	—8·3	—4·1
1896–1900 and 1901–5	—7·0	—9·0
1901–5 and 1906–10	—9·7	—8·8

It will be seen that there has been a fairly steady decline in the death-rate from phthisis, while the decline in the death-rate from all causes, excluding tuberculosis, has been irregular in amount, and only in recent years has approached that from phthisis.

Improvements in housing, progress in average social conditions, higher nutrition, all have doubtless borne their share in bringing about this great reduction in tuberculosis. More cleanly habits of the people must be given a large share in securing the result. The habit of indiscriminate spitting, although still prevalent, is much less so than in the past. The standard of domestic cleanliness has improved, and this must have cleaned out many of the former centres of infection. Even more importance must be attached to the diminished overcrowding of bedrooms.

Overcrowding in bedrooms under all circumstances is liable to become a source of disease, still more so when sick persons are participants in the overcrowding. This being the case, there can be no hesitation in accepting the statement that consumption is spread largely in the bedroom, and that, as Koch has repeatedly pointed out, one of the most important means for stopping its spread is to prevent the sleeping of consumptives in the same bedroom, and, still more important, in the same bed as healthy persons.

A partial antidote to urbanization. In this connexion further stress may be laid on the fact emphasized by Koch that the reduction of phthisis in this country has been associated with a steady increase in the use of hospitals for the tuberculous sick. The use of these hospitals has not been directed to the segregation of the sick. Their use has formed part of the greater humanity of the nineteenth and twentieth centuries, and of the increasing demand for treatment of the sick under the most advantageous conditions. Incidentally, such increased expenditure on the hospital treatment of the sick has been made possible by our increased national prosperity; and a not very inaccurate index of national welfare in any country would be constituted by a comparative statement of its hospital provision. But let me state the facts for England and Wales. A comparison of 1881 with 1910 is given below.

In	*Percentage of the total deaths from all causes occurring in Public Institutions.*			
	London.		*England and Wales.*	
	1881.	1910.	1881.	1910.
Workhouses and workhouse infirmaries	13·1	22·9	7·0	10·9
Hospitals	7·3	17·6	2·8	7·5
Lunatic asylums	0·4	0·8	1·0	2·1
Total	20·8	41·3	10·8	20·5

In the country, as a whole, in 1881, one out of every nine deaths, and in 1910, one out of every five deaths, occurred in public institutions;

in London in 1881, one out of every five, and in 1910, two out of every five deaths occurred in public institutions. So far as phthisis is concerned, it is significant that in 1911, in the whole of England and Wales, 34 per cent. of the male and 22 per cent. of the female deaths, and in London 59 per cent. of the male and 48 per cent. of the female deaths from this disease occurred in public institutions. Inquiries at these institutions show that the patients had usually spent several months in them prior to death. By this means, therefore, not only had the families of the patients been relieved of a serious burden, but also had been released from the risk of infection, at a time when this was especially difficult to control.

The rapidly increasing urbanization of our population must be remembered in connexion with this reduction of phthisis and increase in its institutional treatment. No fact is better established than that tuberculosis claims more victims out of a given number living in towns than in rural districts. In London the phthisis death-rate in males in 1911 was 1·68 per 1,000 of population, being 1·50 in the aggregate county boroughs, 1·04 in other urban districts, and 1·32 in the aggregate rural districts. Now the proportion of the total population living under the relatively unfavourable conditions of town life has during this period of declining tuberculosis steadily increased. The proportion of the total population in England and Wales living in rural districts has decreased from one half to less than a quarter between 1851 and 1911. It is evident, therefore, that some important influences have been at work counteracting the effect of urban conditions as a whole on tuberculosis. Among these, important place must be given to the hygienic effect of the stay for months in an infirmary or hospital of a high proportion of the total consumptive population, at the most infectious and helpless periods of their illness.

At the present time, the prospect of complete control over tuberculosis is more promising than ever before. Not only is public administration, with its magnificent past effect on tuberculosis, becoming increasingly efficient; but the National Insurance Act happily has given further important means of effective attack against this disease. The sanatorium benefit was necessarily confined to insured persons, with possible extension to their dependants. The capital grant of 1½ millions for the building of institutions for the treatment of tuberculosis was made for the entire community; and the further promise by the Chancellor of the Exchequer to contribute half the cost of schemes of treatment of tuberculosis—beyond the cost of treating the insured—for the entire community, has opened the way for complete schemes of prevention and treatment, which are now in process of gradual realization throughout the length and breadth of the land. These schemes ensure early diagnosis, prompt treatment, and the removal of sources of infection, by adequately linked up measures of domiciliary treatment, and of treatment at dispensaries, hospitals, and sanatoria. When these schemes are matured,

there will, it is believed, be no need for any patient to lack the means of treatment under the conditions best adapted to his welfare.

In giving these illustrations of what medicine and the sanitary service of the country have accomplished, my survey has necessarily been incomplete. Not only is there a marvellous record for typhus and enteric fever, and for tuberculosis, but also for the diseases and accidents for which medical aid is required, and to a less extent for puerperal fever. A still more striking illustration could be found in small-pox, and even measles and whooping-cough in recent years appear to be losing some of their power under the influence of the child-welfare work which is gradually becoming systematized in many sanitary areas. The marvellous advances in tropical medicine are well known to all of you. Future generations will hold in gratitude the memory of Laveran, Manson and Ross, of Reed and Carroll, and of many others who are rapidly transforming the tropical countries, formerly known as white men's graves, into places in which the European can live with safety, if not also with comfort.

The saving of life in this country has not been confined to the diseases ordinarily regarded as preventable and curable. Even cancer, if only recognized and treated at an early stage, and when accessible to the surgeon, loses a portion of its terrors; and it cannot be that the concentration on investigation of this disease in nearly every civilized country will fail during the next few years to add it, like tuberculosis, to the diseases doomed to insignificance if not actual annihilation. When the next International Congress of Medicine meets in London, who among you will be the hero to be honoured in regard to this disease, as Pasteur was honoured as the leader of a new era of preventive medicine when you met in this place in the year 1881?

With the discovery by Schaudinn of the spirochæta of syphilis, and with that wonderful application of chemistry to therapeutics which the genius of Ehrlich has placed at your service for the rapid treatment of this disease, we may hope for more systematic and universal treatment of syphilis, and the removal of one of the great cankers of humanity.

I have not time to speak of the improvement in infant and child mortality which has been realized in recent years, thanks in large measure to the active work undertaken by the officers of sanitary authorities, acting in co-operation with voluntary associations. Full details of this are given in a recent report issued by the Local Government Board, and the subject was fully discussed at last week's important conference, over which I presided.

Alcoholism and disease. Nor can I linger for more than a moment over the improvement in regard to alcoholism which our national statistics reveal. Although in our national life vast sums of money are still wasted on alcoholic drinks, the amount is declining, and I cannot refrain from associating this fact in some measure with the declining death-rate from all causes in the aggregate, and more particularly with the declining

death-rate among infants and from tuberculosis. The association in regard to infant mortality obviously is through the diminution of neglect and the improved standard of domestic life.

In regard to alcohol, the responsibility of the medical man is very heavy. The medical opinions of one decade are the popular opinions of the next; and for the inordinate belief in the dietetic and therapeutic value of alcohol, is not the medical profession in some degree responsible? It is gratifying to find that this is now altered. The following figures from the excellent work on alcohol by Sir Victor Horsley and Dr. Sturge may be quoted. In 1862 the patients occupying 2,254 beds in seven metropolitan hospitals consumed alcoholic liquids to the value of £7,712; in 1902 the amount spent in connexion with 2,309 beds in the same hospitals was £2,925. In 1875, 1,405 patients were treated in the Wandsworth Union Infirmary, and the alcoholic expenditure was £371; in 1905, 5,451 patients were treated, and the alcoholic expenditure was only £2 7*s*. 5*d*. Again, during the year ended March 31, 1912, the total expenditure on alcohol in the workhouses and infirmaries of the metropolis was £1,743. Taking the average daily number of inmates as 50,900, this amounts only to about 1*d*. per inmate per week.

My review of our indebtedness to medicine for public health progress would be incomplete did I not refer to the aids which biology and chemistry are now giving us in the diagnosis and treatment of disease, and to the important subjects of hospitals and of nursing.

The application of science in medical practice. The teaching of recent years is bringing home even to the man in the street the importance of all branches of science in relation to medicine. All knowledge sooner or later is pressed into the service of humanity, and it is therefore the bounden duty of the people as represented by the Central Government and by local authorities to give all practicable assistance, not only in utilizing to the full extent our present knowledge, but also in extending its borders.

The value of the X-rays in the diagnosis and treatment of disease, the possibilities opened out by radium, the almost magical contributions of chemistry as illustrated in the work of Ehrlich and others can only be mentioned in passing.

Happily there are signs that medical research will be assisted by the State in future on a scale not previously contemplated; and in this connexion special mention must be made of the promise contained in the prospective expenditure in this country on sound and broad lines of the sum of some £58,000 per annum in connexion with the National Insurance Act.

Another branch of work in which research and scientific diagnosis are admirably commingled is exemplified by the splendid work which is now being done in the clinical laboratories by many of our large general hospitals. These laboratories provide facilities for the accurate and complete diagnosis of disease occurring among the poorest of the population

which are as yet inaccessible to the vast majority of the total population, who are treated in their own homes. This limitation of valuable life-saving and disease-preventing work ought not to continue. I do not forget the fact that a large number of local authorities have provided facilities for the diagnosis of the three diseases—diphtheria, enteric fever, and pulmonary tuberculosis. It may be hoped that in the near future it will be practicable to extend the work done in laboratories, and to make it available for the wider needs of general practitioners, thus enabling them to bring up their work more nearly to the level of that carried on in well-equipped hospitals. If this is to be secured, it is essential that every medical practitioner should have the opportunity of utilizing for every patient the best means for accurate diagnosis which modern medical science provides.

The ever increasing institutional treatment of disease. I have already spoken of the increasing extent to which the sick are treated in institutions. Although at first sight the connexion may not be obvious, I regard this as an essential branch of the subject of housing. If overcrowding is dangerous among those who are well, its danger is multiplied if the sick are commingled with the healthy ; and it is almost certain that the universal excess of disease in town over country would have been much greater had it not been for the assistance given by the hospital to supplement domestic housing and nursing. It is not too much to claim for our hospitals that they have gone far towards removing the harmful effects of aggregation of populations in towns. Think, for instance, not only of the relief to the patients themselves, but of the easing of the burden borne by the overworked family, which is involved in the simple statement that in the year 1911 in the whole of England and Wales 24 per cent., and in London 47 per cent., of the total deaths from cancer occurred in public institutions. But I am speaking to those who, with the philanthropists, have rendered this supremely beneficent work possible, and who have in very large measure carried it out ; and I need, therefore, only add a few figures which will make still clearer the enormous extent to which the home has been relieved by the hospital. I am indebted for these figures to *Burdett's Hospital Annual*. In the United Kingdom there were in 1911, 49,854 beds in voluntary hospitals, or about 11 beds to 10,000 inhabitants, 80 per cent. of these being in daily occupation. The average stay for each patient was twenty-five days. These hospitals, including dispensaries, had in 1896 an income of £2,528,000, which in 1911 had increased to £4,185,000. The beds here enumerated do not include the much larger number in our workhouses and infirmaries, lunatic asylums, isolation hospitals, &c.

The rôle of the nurse. The noble work of the medical profession in the treatment and prevention of disease would have been in large measure ineffective but for the devoted work of the nurse. In every age of the world there have been ' born ' nurses, but the ' trained ' nurse is a product of recent times. It is scarcely for me to sum up the qualities of the ideal

nurse ; but I may safely say that no amount of training will compensate if there is not also the tenderness of touch, the kindness of heart, and the desire to serve, without which the highest and best nursing cannot be secured. Personal experiences of many, if not of all of us, make us confident that paid service is not incompatible with the devotion and self-denial which we associate specially with unpaid service, and without which skilled nursing fails in an important respect.

The history of nursing is bound up with that of Florence Nightingale. Until beyond the middle of the nineteenth century Sairey Gamp and Betsy Prig were scarcely exaggerated types of the hired nurse. But in 1851 Florence Nightingale entered the Deaconess's Institute at Kaiserwörth on the Rhine ; and thus apparently the impetus for improved nursing in this country appears to have originated in a German charitable nursing institution. The great work of ' the lady with the lamp ' in the Crimea is well known. With courage and knowledge, and with inflexible discipline at the military hospital at Scutari she changed a seething mass of loathsome contagion and confusion into a cleanly organization of sick patients.

With the gift of £50,000 raised for her by a grateful public she endowed at St. Thomas's Hospital the first great nursing school in this country, and she thus founded our modern system for the training of nurses.

The growth of the profession of nursing is among the most striking social facts of the last thirty years. As medicine progresses and becomes more exact the need of nurses to carry out the details of medical instructions becomes more clamant. In Poor-law institutions alone some idea of progress may be gained from the statement that in 1897 the total nurses employed numbered 4,100 ; in 1907 they had become 6,500 ; in 1911 nearly 7,500 ; and in 1912 over 7,600. For the voluntary hospitals, complete figures are difficult to give, but the following figures, taken from that valuable compilation *Burdett's Hospital Annual*, will give some conception of the magnitude of their work in institutions.

	1911.	
	No. of occupied beds.	*Total nursing staff.*
A. *Hospitals with Medical Schools* :		
Eleven London hospitals . .	4,031	2,055
Nine provincial hospitals . .	2,295	833
Five Scottish hospitals . .	2,511	848
B. *Other General Hospitals* :		
In London	970	368
In provinces	3,385	1,151
In Scotland	281	92
C. *Special Hospitals* :		
In the United Kingdom . .	2,027	736
	15,500	6,083

For every five patients in voluntary hospitals two nurses are provided, including day and night service.

These numbers do not take into account the large number of nurses engaged in private nursing. Nor do they include the nurses whose work is even more closely associated than that of the institutional or private nurse with the prevention of disease. In London and the chief centres of population we have the Queen's Jubilee nurses, in many counties county nursing associations, and in nearly every parish a nurse who is engaged in helping the poor in sickness. This help does not consist solely in the valuable aid rendered at time of distress. Good district nursing also implies the teaching by example of methods of nursing and personal hygiene, which in the after-life of the assisted family are probably more valuable than the immediate aid.

Did time permit, one might advantageously discuss the valuable work of female sanitary inspectors, health visitors, tuberculosis nurses, and school nurses, which is having important influence in reducing infant mortality, in preventing the spread of infection, and in securing a higher standard of domestic and personal cleanliness. But I must conclude my brief review of the progress of medicine and the work ancillary to it in relation to public health.

National interdependence in relation to health. Your presence here to-day emphasizes the international aspect of health. Rapid travel by land and water implies greatly increased possibilities of spread of disease. Happily, owing to intercommunication of information and improved administrative measures, precautions can more than counterbalance the increased possibilities of disease. News of danger can be transmitted by telephone, telegraph, cable, or wireless ; a whisper is heard from one hemisphere to another, and so the benefits of intelligence and supervision can be universally shared. In other directions the possibilities of healthy life have been indefinitely increased. Our food is collected from all countries, and we send in exchange our manufactures, each country according to its special facilities or aptitude. Commerce and finance have formed a meshwork which covers the earth.

Medical knowledge in particular is cosmopolitan. Each country has brought its contribution. I have already mentioned the names of Pasteur and Lister, of Semmelweis and Koch, of Laveran, Manson, and Ross, and others. They stand out ; but every great discovery has its background of smaller contributions to the common stock of knowledge without which no great achievement is secured ; and every nationality has added its quota. So is it also with reforms. A reform in one country initiates or gives added impetus to efforts towards reform in other countries ; and the tendency to follow becomes almost irresistible. In some countries it has already become a duty of civilized life to prevent anxiety as to the means of subsistence in old age ; and so old age pensions have been inaugurated. In every country increased attention is being paid to the need to aid, when necessary, the widow to bring up her children ; and insurance against accidents and disease is already in operation in Germany and in the United Kingdom. Every effort forwards is valuable not only

to the country immediately concerned, but also to every country; even mistakes by one nation add to our common stock of wisdom.

In no department of advance is cosmopolitanism so important as in regard to social reform, an important share of which consists in the development of the medical possibilities of improved health and well-being. The chief impediment to such reform is wastefulness, and one source of waste, expenditure on armaments, stands head and shoulders above all others. If this waste could be prevented, gigantic sums would be released for the reforms waiting to be financed. The chief among these is concerned with the problem of poverty, and this problem will need to be attacked on an enlarged scale by removing the known causes of poverty, not merely by applying palliatives to its effects.

It is not too much to hope that this Congress will help to hasten the period of international peace and goodwill, earnestly desired by us all, in which our only rivalries will be in commerce, in progress, in science, and in the art of healthy and pure living.

SERMON

PREACHED before the members of the Seventeenth International Congress of Medicine by the RIGHT REV. THE DEAN OF ST. PAUL'S, on Sunday, August 10, 1913, in St. Paul's Cathedral.

THE MEDICAL PROFESSION AND SOCIAL REFORM

'That there may be no schism in the body,' 1 Cor. xii. 25.

A PREACHER who stands up to deliver his message in the presence of so many members of the International Medical Congress must wish heartily that he could change places with some one of the eminent physicians who sit below him. An annual 'Doctors' Sunday', on which the sermons would be preached by members of the medical profession, would be an institution of no small value. The doctors have much to teach us; I am going to urge very respectfully in the course of this sermon that they do not make their voices heard quite enough. In their private practice they have to a large extent succeeded to some of the functions of the mediæval priests. It is they who now hear the confessions of anxious and conscience-stricken penitents; it is they who prescribe dietary disciplines and various quaint penances; it is they who send people on pilgrimages to distant lands. Moreover, owing to the state of neglect into which the art of spiritual therapeutics has fallen in Protestant countries, the physician usually knows more than the clergyman about the real springs of action, the secret causes of sin and sorrow, the subtle and delicate influences by which soul and body affect each other, the mysterious and melancholy trammels of morbid heredity, and the unrecognized heroism of struggles against it. To him, and not always to the clergyman, are known the storm and stress which often accompany the beginning and sometimes the end of the sexual life, the nervous instability which may show itself in mental depression, in foolish and violent partisanship, in loss of natural affection, and in many other obscure ways. In short, when a physician of the soul preaches to physicians of the body, he is presuming to instruct those who probably know more of his own trade than he does himself.

It would be tempting to draw a parallel between modern developments of medical science and the advice which the most experienced spiritual guides give to those who consult them. For instance, we are no longer encouraged to muffle up our throats to keep out bronchitis, and to pad our chests against consumption. We are not bled to reduce our temperatures, or taught to be afraid of fresh air. In many cases no frontal attack is made on a local disease, but attention is given to the general health. Recently it has been recognized that the microbes are not all our enemies, and whole armies of Friendlies are sometimes injected to do battle with the intruders. So in the spiritual life we have learned the value of fresh air. Solitude is dangerous; active life among our fellow men is salutary. Many faults resist obstinately any direct assault, but yield to a treatment which aims at strengthening the whole character. We are warned that we shall never keep the commandments by striving to obey them one by one; nor, on the other hand, can we, properly speaking,

keep any of them if we deliberately allow ourselves to break one. As St. James says, 'Whosoever shall keep the whole law, and yet offend in one point, he is guilty of all.' Again, the wise spiritual guide desires to build up a strong character with positive characteristics: he is not in favour of 'letting blood' to reduce exuberant vitality. He remembers that where the old Law dealt in prohibitions, the Gospel lifts us into a region where prohibitions are needless. He who has learned that 'Blessed are the pure in heart, for they shall see God', needs no more the seventh commandment; he who has learned that 'Blessed are the poor in spirit' need not trouble himself about the tenth. Once more, we have learned how in the life of the soul, as in the life of the body, there are laws of growth and habit. The mischief of years cannot be cured in hours. Habit can only be cured by habit. The *law* of the spirit of life alone can deliver us from the law of sin and death.

There is nothing surprising in these parallels; it would be strange if spiritual and bodily health did not follow the same laws. And though the mutual understanding between physicians of souls and bodies is by no means so complete as it should be, common sense and observation do frequently suggest to the physician an appeal to moral and religious motives, and to the clergyman that the case before him is one for the physician rather than for himself. I am not competent to say whether a physician would find it worth while to study the best Catholic manuals of spiritual direction; but I have no doubt that the clergy would find themselves amply repaid for time spent in acquiring a knowledge of medical psychology. Even a little knowledge of this kind will sometimes enable us to see what is the matter with a person whose conduct is causing his friends anxiety. Possessed of this knowledge, a clergyman might often be a valued helper to the doctor in the sick-room, instead of only coming in at the last as the herald of the undertaker. Heaven forbid that we clergy should take advantage of the present recrudescence of superstition by posing as medicine-men and miracle-workers—there is too much of this in London already—but the true priest does wield a spiritual dynamic which, if he knows the limitations imposed on him by nature's laws, may be a potent adjunct to the art of the physician. The religion of Christ does heal as well as console. The forgiveness of past errors through Christ is a fact, and not only in the transcendental sphere. 'From the hygienic side,' writes an American professor of medicine, 'there is a tremendous advantage to be gained from the religious appeal whenever it can be used.'

After this preamble I come to the point which I wish with all possible respect to press upon the leaders of the medical profession. They are, as I have said, our modern father-confessors. Men and women now go to them who formerly went to the clergy. They have earned and they have received the confidence of all who are in trouble about their bodies, and of many who are in trouble about their souls. No one can speak too gratefully of the way in which this work is done, of the skill, the kindness, the understanding sympathy, the charitable but not too easy-going toleration of human infirmity, which the patient can depend on receiving from his medical adviser. The public has no cause to grumble if the physician says that his duties to his patients leave him no time for anything else. Nevertheless, I do wish to put this question. Does the medical profession, as a whole, take its proper part in guiding and influencing public opinion in those matters wherein its members alone can speak with authority? I remember a short biography of that great man, Sir James Paget, written by a former canon of this cathedral, a friend

and admirer of that eminent surgeon. After doing justice to his many great qualities, the biographer (if I remember right) complains that Paget seems to have suffered from ' State-blindness ', by which he means to indicate that his hero was too indifferent to social reform. Would it be altogether untrue to say that the majority of physicians and surgeons are content to do their best for their patients, and keep for the most part out of public affairs even when matters which they understand better than any one else are under discussion ? There are none who in private conversation talk more sanely and wisely about such questions. Their freedom from prejudice and sentimentality is most refreshing, and justifies the saying of Nietzsche that the best thing about science is the pure and wholesome air that blows around it. We have felt this invigorating atmosphere in reading the reports of this great Congress. But when a measure is brought in, such as the Mental Deficiency Bill this year, where is the medical profession ? Some leading physicians did speak out. But did the profession throw its whole weight into the scale in support of the Government, when two or three unscrupulous obstructives were trying to destroy the Bill ? A few did so, but most of the work, I think, was left to a society of laymen. There are other matters in which very much might be done by legislation for the health of the nation, matters which, no doubt, are as yet not practical politics, but which might become so if science would speak out and go on speaking. For scientific men are listened to, even in an age which despises intellect. A man may forget that he has a mind, and deny that he has a soul ; but he cannot get rid of his body. All those problems of heredity and schemes for the improvement of the human stock, which are grouped under the new word Eugenics, are attracting increasing attention, even among the intelligent working-classes. Would it not be possible for the medical profession to recognize that they are called to an even higher duty than that of keeping the breath in their patients' bodies as long as possible, a duty which, as none know better than they, is often of doubtful public advantage ? To educate public opinion is a painful duty, but in a democratic State it must be faced, and each of the great professions has its allotted task in keeping the public well-informed in the subject on which it can speak with authority. If they do not undertake this work, it will not be done at all. Professional politicians cannot do it. If the State is an organism, as we are constantly told, the great professions are its organs, with definite functions to perform in the economy of the whole. If they fail in this, there will be what St. Paul calls ' schism in the body ' ; there will be disease in the body politic.

So far as one can predict, natural science, and the science of health in particular, must hold an ever-increasing prominence in the life of nations. In all dreams of a future Utopia, large drafts are made on the future resources of medical science. And the history of the last hundred years justifies the hope that far more may be achieved in this way for human welfare and happiness than by any political or economic changes. We may look forward to a time when many of the diseases which now ravage humanity will have gone the way of leprosy and typhus ; when some morally unobjectionable way of regulating population will have been discovered ; when the average health, strength, and beauty of the people will be greatly enhanced ; and when those crimes and vices which crop up, as if by fatality, in tainted stocks will be comparatively rare. The knowledge which makes this real progress possible is a divine revelation intended to be used. As it advances, it will lighten the task of the religious teacher, the moralist, and the social reformer, and will make the

work of the doctor less tragic than it often is now. The human race at present is a mere caricature of what it may be in the distant future, if science is allowed to pursue her beneficent course unchecked by that false kind of humanitarianism which is kind only to be cruel. Intellectual and moral improvement will be as great as physical; the race will make a long stride forward towards 'the perfect man'. All this may be; we cannot say that it must be. There is no *law* of progress; all will have to be done by purposive efforts. The most inspiring faith of our day is the confident belief that purposive effort, guided by expert knowledge, may achieve great things for the human race. This faith already directs our political life; it should also be a beacon light to science. I have hinted, perhaps presumptuously, that the medical profession might use this weapon of concerted and collective action in the public interest more than it does at present. The criticism is at any rate prompted by the sincerest respect, and by the wish to see the new knowledge which God is every year putting within our reach used to the fullest extent for the good of our contemporaries and the still greater good of those who shall come after.

SERMON

PREACHED before the members of the Seventeenth International Congress of Medicine by the RIGHT REV. BISHOP RYLE, Dean of Westminster, on Sunday Morning, August 10, 1913, at Westminster Abbey.

'THE BODY OF OUR HUMILIATION'

(Phil. iii. 21.)

THERE are few phrases of the Authorized Version that have earned such a widespread familiarity as the rendering 'our vile body'. We may not all of us have yet grown reconciled to the alternative which is given by the Revised Version. But of this I am quite certain, that 'the body of our humiliation' literally and accurately represents the Greek words used by St. Paul, and that it does get rid of an epithet, which has lost its earlier meaning of 'common', and which, in its present significance at any rate, conveys the idea of contempt and depreciation. The faith of the Apostle was such that he could say, 'To me to live is Christ, and to die is gain, . . . having the desire to depart and be with Christ; for it is very far better' (Phil. i. 21–3). But he is also the man who could say that 'every creature of God is good, and nothing is to be rejected, if it be received with thanksgiving' (1 Tim. iv. 4).

There can be no doubt that the words, 'our vile body,' were constantly employed by our forefathers as the expression of St. Paul's teaching and as corresponding with a phase of fashionable theology.

It was just one of those phrases which stick in the memory and form an easy nucleus of ignorant assumptions about the character of religion.

For this there were two or three reasons.

1. There was always a tendency to exaggerate and pervert the words of our Lord's teaching: 'What is a man profited if he shall gain the whole world and lose his own soul? or what shall a man give in exchange for his soul?'

The life of the body seemed to some enthusiastic minds to be distressing and burdensome. And though our Lord's ministry of healing and of ministering to the bodily diseases of His countrymen should have corrected the mistake, the mistake was one which became prevalent in the Church. The concerns of the body came to be regarded as lying outside the range of true spiritual sympathy and the highest Christian interest.

2. There is always reappearing in the Church some revival of the widespread Eastern doctrine that the spirit alone is good, that all matter is evil; that while the God of light and truth created the spiritual world, it was a god of darkness and falsehood who created the body and the world of matter. To us Westerners it may appear strange that a speculative opinion of Persia and India should have taken so strong a hold of European phases of opinion during the Middle Ages.

INFLUENCE OF MONASTIC ASCETICISM

3. There is the influence of monastic asceticism, which treated the body as the enemy of the soul, and forgot that though it was a channel of temptation to evil, it was also the appointed instrument of serving God

upon earth. Books of devotion often lapsed into an attitude which seems to assume that because self-discipline, restraint, and abstinence are indispensable for the freedom of spiritual life from the evils of self-indulgence, therefore the body is inherently bad, pleasure of any sort pernicious, and physical health and activity a subtle form of evil.

It is, I suppose, part of our inheritance from the Middle Ages which is responsible for a good deal of this kind of language. To our ears it sounds unnatural, overstrained, and insincere.

When you hear a crowded chapel of schoolboys or schoolgirls singing with fervour :

> Seeking Jerusalem, dear native land,
> Through our long exile on Babylon's strand,

you feel that the language which was suitable for the aged occupant of the monastery or nunnery has an unreality, a false ring in it, when it is uttered by happy, healthy boys or girls on the threshold of a life of active service for their country's welfare.

Even in the lofty atmosphere of the simple devotion of Thomas à Kempis you realize that the practical directions for a life of piety are addressed not to a citizen of the world, but to one who has taken the vows of a religious community.

Swing of the Pendulum

On the other hand, in the present day the pendulum has swung almost to the opposite extreme. It is no longer sin which seems to be regarded as the foe, but physical disease, suffering, and death. The warfare which excites public sympathy is the warfare against ' bacterial rulers of darkness ', against epidemics, against fevers, against cancer.

For this there are a variety of reasons. In the religious world the doctrine of material rewards and punishments has no longer the exclusive popularity which it enjoyed a hundred years ago. It is no longer the pictures of the delights of heaven or the pains of hell which fill the periods of the popular preacher.

In the second place, the admiration for athletics has tended to surround physical prowess and distinction with a halo of glory. The culture of the body is part of modern education ; and though it may easily be overdone, it is part of the more reasonable recognition of God's gifts to man.

There was real discernment as well as cynicism in Beaconsfield's phrase, ' Sanitas sanitatum omnia sanitas.'

Thirdly, the whole movement in the direction of social reform has had the salubrious effect of compelling the Church of Christ to admit that ' religion is concerned with man's actual state in this world as well as with his possible state in the next '. To quote the striking words of a famous book : ' Both in the public press and in the pulpit, from Nonconformity and Orthodoxy alike, we have the note sounded in varying keys, that, after all, Christianity was intended to save not only men, but man, and that its mission should be to teach us not only how to die as individuals, but how to live as members of society.'

Progress of Medical Science

Fourthly, there has been the well-ordered progress of medical and surgical science. The names of Pasteur, Virchow, and Lister, as we have been reminded in the last few days, are the names of men who have revolutionized the conception of disease, who have laid out new methods

of combating sickness and pain, who have mitigated the sufferings of humanity. The art of healing has entered into a renown which it never previously enjoyed. It has attracted some of the most famous intellects of the world. The discussions of modern medical science have fascinated the imagination of all civilized races. We may have passed the highest peaks of discovery, but, as has been said by the President of the Congress, we have entered upon a lofty and fruitful tableland of continuous progress and invention. The achievements of modern medical and surgical skill work effects that one hundred years ago would have been pronounced 'supernatural'. God performs His miracles through the agency of human means. And we rejoice to see in this ever-ascending progress of discovery which makes for the amelioration of the human lot and for the lessening of human suffering, a sign of the Divine Presence and a pledge of the Divine mercy and love. It is not reasoning faith, but light-hearted and superficial scepticism which makes some persons turn their backs upon the patiently acquired results of thought and study and science, and have recourse to the well-advertised methods of untrained healers.

But as much as ever is the body 'the body of humiliation'. The skill and science of the most skilful and most scientific have made no alteration there.

While you and I daily have reason to thank God for the wonderful ministrations of healing, yet everywhere we are brought face to face with the facts of suffering, illness, weakness, increasing infirmity, the approach of death. It is the humbling of our self-confidence; it is the reminder of our short span on earth; 'it is the body of our humiliation.'

It will be strange if you and I have not, most of us, experienced something of the humiliation either of great pain, or of grave illness; or have not at least been partakers in the weakness and illnesses of those most dear to us. All the circumstances of sickness, its petulance, its physical distress, have been burnt into our recollections. Maybe we have emerged from it in safety, singed, yet not consumed, and look back upon it all as a kind of dream. But we know it was not a dream; and we know, probably, that the other world, which is not that of the earth, has become closer and more real to us; that the room of the invalid has been blessed to our enduring benefit.

Servants of the Human Race

And we know that the whole brotherhood and sisterhood of doctors, surgeons, and nurses, are now acknowledged by us with growing and unceasing gratitude to take rank among the truest servants, friends, and benefactors of the human race. This is no mere compliment; for it carries with it a responsibility which is scarcely, perhaps, realized to the full. The influence of the medical profession extends far beyond the limits of the physical. Under certain conditions the doctor will be a more effectual teacher of temperance than the clergyman. The doctor is the person to whom is confided the secret of individual or family trouble. The doctor is able to speak about purity, self-control, and chastity in a manner that will certainly command the attention, and possibly the obedience, of his hearer.

Does he always use his opportunities? He is shy, driven for time, preoccupied, awkward, and does not wish to interfere in private concerns. But he is indisputably a genuine messenger of God, and he must be a faithful servant of the people. And he is bound, where he has the opportunity, to deliver to the people the Word of God—that is, to testify for purity and chastity, as well as for cleanliness and temperance.

The horrors of a disease of which it is a shame to speak would never have reached their present pitch, if it had not been for the accursed doctrine preached in the name of medical counsel, that a young man could not keep himself healthy if he kept himself pure

Doctor and Priest

The doctor will not encroach upon the province of the priest ; but he will support and reinforce the moral and spiritual outcome of the teaching of the Gospel of Jesus Christ. And the priest on his side has a message to bear and work to do in conjunction with and in support of the doctor. We are not merely subjects for an operation ; we are flesh and blood, creatures of love, and feeling, and friendship, and emotion.

The doctor or the surgeon should, so far as possible, make it possible for the patient to receive the assistance of prayer and Holy Sacrament, of thought, quiet and steadying, on things of eternal life and of Divine love.

It is true that a hurried petition, a repeated text, at the last moment, can do but little for an unprepared and careless soul. Nevertheless, it is at once the duty and the privilege of the doctor to secure for his patients the support and calming influence of the Divine grace which spiritual consolation pre-eminently administers. And it is the clergyman's most solemn and most imperative duty to obey the doctor's summons. There is nothing in parochial organization and business that can take precedence of the ministration to the sick and the dying.

Finally, let us as faithful servants of the Lord realize that of which the Apostle testifies in the passage from which these words, ' the body of our humiliation,' are taken. It is but the earthly vesture of the soul. It is destined ere long to be resolved into its original elements. But there is that in us which will survive the death of ' the body of humiliation '. It is the seed of the spiritual body. It is that which is to be quickened into life by the Spirit of Christ. And though we have no certain word of direct revelation to go upon, yet may we believe that the body of our glorious resurrection, like the risen Body of Christ, will be such as to maintain the identity of personality and such as to continue the relationships of affection and friendship, even as in the appearances of the Risen Lord we are furnished with the materials of a good and joyful hope.

' For our citizenship is in heaven ; from whence also we wait for a Saviour, the Lord Jesus Christ, who shall fashion anew the body of our humiliation that it may be conformed to the body of His glory.'

SERMON

PREACHED before the members of the Seventeenth International Congress of Medicine by His Eminence CARDINAL BOURNE, Archbishop of Westminster, in Westminster Cathedral, on Sunday, August 10, 1913.

'I WAS SICK, AND YOU VISITED ME'

(Matt. xxv. 36.)

THE coming of our Divine Master, Jesus Christ, has changed the outlook of men on their fellow creatures. Until He came few were conscious of the great fact that, as all human beings have the same origin and the same ultimate destiny, they ought not, and, indeed, cannot be indifferent to the lot of other members of the race, as they pass on their way from their birth into this world until their departure for the world that exists beyond. His teaching repeated in so many forms that we are all alike the creatures of the one same Creator, and may if we wish become in baptism all alike children of the Father who made us; and that it is His purpose that we should, again all alike, be united for ever in His, our Father's kingdom; these are facts which, viewed in the light of the great atoning Sacrifice offered for us, each and all, have built up in the consciousness of the whole of Christendom a sense of mutual dependence and responsibility.

The old human relations of father and child, of master and servant, of ruler and subject, of citizens of the same state, of dwellers in the same land, of speakers of the same tongue, whereby men have from the beginning been knit together, have not been destroyed. They exist as always, and they will ever continue. But they have been raised to a higher plane, and around them has been cast a new and all-embracing chain, uniting to them in various degrees those who in former days stood without any recognized bond of fellowship. In the full acceptance of the Fatherhood of God, and in our knowledge of our Brotherhood in Jesus Christ, we are all linked together, and shall have to answer one day to our Father, who is in Heaven, for the care that we have given to our brethren. There is no possibility of mistaking the far-reaching import of that series of our Master's sayings from which my text is taken.

It is clear that the responsibility involved in our Lord's declaration does not affect all men equally, for the degrees of our opportunity of thus rendering service to our brethren and, through them, to Jesus Christ, vary incalculably. It is your privilege, and, therefore, your responsibility, to form, by reason of your profession, a distinct class of those to whom our Lord's thanks, as well as His reproach, will one day be addressed, 'I was sick, and you visited me.' 'I was sick, and you did not visit me.'

To none should it be more easy, to none is it more necessary, to see Jesus Christ in even the least of His brethren, than to those who are called to the ministry of healing. They are brought closer to the real facts of life than any outside the priesthood. They see life at its very dawn, they watch over it through the helplessness of infancy, they are constant witnesses of vital processes which their science can follow up to a certain point, but is powerless adequately to explain. The farther

their research may carry them, the more wonderful are the mysteries that they discover. If the heavens are continually telling the glories of God, still more are the marvels of the human body continually proclaiming the boundless resourcefulness of the Creator.

You are daily witnesses, too, my brethren, of the dependence of men upon their fellow men. You see how they look for your coming, and hang upon your words, and give you their obedience, trusting alike to your wisdom and to your uprightness. In virtue of your calling you exercise an authority and you claim an attention which men gladly and willingly give to you on account of the trust that they have placed in you. And you know, in the depths of your hearts, that you are worthy of that trust only if you, in your turn, acknowledge the sovereignty of God, from whom all your knowledge comes, and who alone can guide you safely and guard you from error and mistake.

In the unveiling of the secrets of bodily misery and disease, you see also, often very closely, the weakness and frailty of human souls. You watch the interplay of mind and body, of spirit and matter, which so often explains, and often, too, renders more difficult of explanation, the phenomena that you are tracing to their source. You are brought very close to immortal souls, and your characters, your words, the principles which animate your lives are, consciously and unconsciously, influencing your patients not only for the healing of their bodies, but for the development or stunting of their spiritual lives as well. You are God's instruments for this life and for the next, and you can discharge your duty only if you remember your responsibility to and your dependence upon Him.

I know well that in your choice and practice of the medical profession, the claim upon your service that I am sketching is not the only one, not even the only legitimate one. You have chosen that career, doubtless, from many motives, all having a due place in the thoughts that ultimately led you to that choice. Family circumstances, scientific bent of mind, hopefulness of not improbable worldly success, desire to follow a special line of study, keen philanthropy, other undefined and undefinable, seemingly fortuitous, influences, have set before you the particular profession that you have embraced ; and have probably determined similarly the nature of the work to which you are devoting yourselves. And all these things have acted and are still acting as so many incentives to enable you to overcome difficulties and to give your talents, your time, your energies to the service of your brethren. God shows His Will, and leads us on to do work for Him in all these ways, and we may be safely guided by such indications so long as they do not blot out from before our eyes the hand that has traced them. On an occasion like this, when the leaders of your great calling are gathered together from the ends of the earth, united, in spite of differences of nationality, of speech, of education, of political conception, of scientific views, as one brotherhood to co-operate in the service of mankind, it is right to rise by a more conscious and sustained effort to a remembrance of the only fully adequate motive for the work of devotion to which your lives are dedicated. Other motives there are, more or less perfect, but for those who accept at the hands of the Catholic Church the teaching of the Gospel, one motive is supreme and embraces all the rest, namely, the seeing of Jesus Christ in the person of His brethren our fellow men.

It is for this reason that, not content with a private rendering to God of that homage of Sacrifice which week by week the Church claims from you in His name, you are gathered together, as Catholic members

of a great International Congress, to testify by a corporate act of worship to your full acceptance of God's claims on the service which, by very reason of your profession, He has made you fit to render to your fellows. As, my brethren, you stand around this altar, whereon the Divine Victim is being offered up for all our needs, look forward to that day when, so far as the life of the body is concerned, all healing arts shall have failed, and your souls will be alone before Him who died to save you. May He then say to each and every one of you, 'I was sick, and you visited me. Enter thou into the joy of thy Lord.'

CLOSING MEETING

The Closing Meeting took place in the Albert Hall on Tuesday, August 12.

The President took the Chair at 4 p.m.

The Secretary of the Permanent Commission presented his report as follows :

RAPPORT DE LA COMMISSION PERMANENTE DES CONGRÈS INTERNATIONAUX DE MÉDECINE

La Commission permanente des Congrès internationaux de Médecine, dans sa réunion du 8 août, a accepté les résolutions suivantes :

I. Ville où sera tenu le Dix-huitième Congrès international de Médecine

La Commission a résolu à l'unanimité des voix d'accepter l'invitation faite par le Gouvernement bavarois et par la ville et l'Université de *Munich*, à tenir le prochain Congrès dans cette ville, en 1917.

II. Prix du Congrès

Les commissions chargées de décerner les prix du Congrès ont résolu d'attribuer :

A. *Le Prix de Moscou* à M. le Professeur Charles Richet, de Paris, pour ses travaux sur l'anaphylaxie.

B. *Le Prix de Paris* à M. le Professeur A. von Wassermann, de Berlin, pour ses travaux de thérapie expérimentale et d'immunité.

C. *Le Prix de Hongrie* à M. le Professeur A. E. Wright, de Londres, pour ses travaux sur l'immunité et la vaccinothérapie.

La Commission permanente des Congrès internationaux de médecine, dans sa réunion du 11 août, a accepté les résolutions suivantes :

I. Bureau de la Commission permanente

Sont élus pour la nouvelle période de quatre ans :

Président : M. F. von Müller, de Munich, président élu pour le dix-huitième Congrès.

Vice-présidents : Baron Calman Müller, de Budapest, président du seizième Congrès ; Sir Thomas Barlow, de Londres, président du dix-septième Congrès.

Secrétaire général : M. H. Burger, d'Amsterdam.

Secrétaire-adjoint : Dr. Ph. M. von der Haer, de La Haye.

Membre : M. L. Dejace, de Liège, président de l'Association internationale de la Presse médicale.

II. Vœux des Sections

Les vœux suivants ont été émis par les Sections du Congrès :

A. Section IX (Ophtalmologie)

La Section émet le vœu :

1. Qu'un Comité international, qui aura le pouvoir de coopter encore d'autres personnes, soit élu par le Bureau de cette Section du Congrès.

2. Que ce Comité soit chargé de rapporter sur le minimum d'acuité d'appréciation de forme, de couleur et d'illumination, que doivent posséder ceux qui fonctionnent comme navigateurs dans la marine commerciale.

3. Que ce Comité soit aussi chargé de considérer et de recommander les méthodes les plus convenables pour constater ce minimum.

B. Section XIII (Dermatologie et Syphiligraphie), Section XIX (Médecine légale)

Les Sections émettent le vœu :

Que, profondément touchés par les ravages causés par la syphilis dans la santé de la communauté et déplorant l'inefficacité des moyens actuellement existants pour combattre sa propagation, le Congrès international de Médecine appelle l'attention des Gouvernements de tous les pays y représentés :

1° Pour instituer un système de notification confidentielle de la maladie à une Commission sanitaire, partout où ce système de notification n'existe pas encore.

2° Pour qu'un système de provision soit légalement assuré tant pour le diagnostic que pour le traitement de tous les cas de syphilis non autrement rétribués.

C. Section XV (Rhino-Laryngologie), Section XVI (Otologie)

Les Sections émettent le vœu :

Qu'il serait très avantageux au public si des spécialistes en Otologie, Rhinologie et Laryngologie étaient attachés aux hôpitaux réservés aux maladies épidémiques.

D. Section XVIII (Hygiène)

La Section émet le vœu :

Que l'enseignement en plein air est extrêmement avantageux pour la santé des enfants, et que les Gouvernements doivent être recommandés de le porter à la connaissance publique.

E. Section XXI (Médecine et Hygiène tropicales)

1. (*a*) La Section est d'avis que le béribéri chez les indigènes, qui se nourissent principalement de riz, est amené par l'usage prolongé et trop exclusif de grains soumis à une moulure et un blutage trop complets, d'où résulte la disparition des couches corticales et sous-corticales du grain.

(*b*) La Section recommande à toutes les autorités, ayant la charge de la santé d'agglomérations d'indigènes, d'encourager par tous les moyens en leur pouvoir l'interdiction de l'emploi de cette farine dans l'alimentation des coolies.

(*c*) Étant donné le caractère non-infectieux bien démontré du béribéri, la Section propose que tous les ports et toutes les autorités sanitaires abolissent les quarantaines et autres mesures restrictives dirigés contre cette maladie.

2. La Section propose que la maladie connue jusqu'ici sous le nom ' fièvre de Malte ' soit dénommée ' fièvre ondulante '.

Tous ces vœux seront considérés par la Commission permanente.

F. Vœu émis par plusieurs Sections

Les Sections constatent leur conviction que les expériences sur les animaux vivants ont rendu les plus grands services à la médecine dans le passé et qu'elles sont indispensables à l'avenir. Elles désapprouvent fortement toute infliction de douleur évitable, mais sont néanmoins persuadées, aussi bien dans l'intérêt des hommes que dans celui des animaux, que — sous la supervision jugée nécessaire — toute facilité doit être donnée aux personnes compétentes de faire de telles expériences.

La Commission permanente propose au Congrès d'approuver ce vœu.

This resolution was proposed to the Congress and carried.

III. Motion de Reconnaissance

La Commission permanente, considérant que l'hospitalité des collègues anglais a surpassé toute attente possible, considérant que ce Congrès gigantesque a été préparé et dirigé d'une manière des plus admirables, exprime sa sincère et profonde gratitude envers les membres du Bureau du Congrès, et, en leurs personnes, envers la profession médicale entière de l'Angleterre.

The President then proposed a vote of thanks from the Congress to the Government of Her Majesty the Queen of Holland for their generosity in defraying the expenses of the Permanent Commission of the International Congress of Medicine. This was carried by acclamation.

The Government delegates then rose in turn to bid farewell to the Congress.

UNITED STATES OF AMERICA—Dr. A. R. Allen spoke as follows:

Mr. President, Ladies, and Gentlemen: Permit me on behalf of the Government of the United States of America to present affectionate gratitude and thanks to His Imperial Majesty, King George V, for the regal welcome and hospitality accorded us by His Majesty's Imperial Government, as well as for the deep personal interest evinced by His Majesty in the work of this great Congress.

Permit me to express to you, the loyal subjects of this great empire, our sense of deep obligation for the courtesies and entertainment of the past week, in number beyond record.

Permit me to felicitate every member of this Congress for the increment accruing to science through his effort.

And now naught remains but to bid you God-speed.

Au revoir, mes amis. Auf Widersehen, meine Brüder.

Fare thee well, O most excellent English hosts. We thrill with pride at the achievements of the Mother-country.

May God prosper us all and grant that we may meet again after four years in beautiful Munich, with even greater *vis a tergo* in our search for truth, the result of the seed sown at this Seventeenth International Congress of Medicine holden in your glorious city of London. Farewell.

AUSTRIA—Professor Dr. Franz Ritter von Haberler spoke.

ARGENTINA—Professor Eliseo Segura spoke.

BRAZIL—Dr. Marcos Cavalcanti spoke.

CHINA—Dr. Wu Lien Teh spoke.

CUBA—Dr. Aristides Agramonte spoke.

DENMARK—Dr. Madsen spoke.

FRANCE—Dr. Lucas-Championnière spoke as follows:

Je suis tout particulièrement heureux d'être chargé d'adresser aux organisateurs du XVII[e] Congrès médical et à la profession britannique les remerciements des Français qui viennent d'être leurs hôtes. J'ai connu l'hospitalité britannique dès ma toute jeunesse et je dois à la science chirurgicale anglaise une grande partie de mon éducation. J'étais à l'avance convaincu qu'elle serait à la hauteur des grandes difficultés de l'heure présente. Mais le succès et le nombre dans ce Congrès a dépassé tout ce qu'on pouvait attendre. Nous avons le devoir de témoigner

hautement notre admiration et notre reconnaissance pour ceux qui ont réalisé la perfection dans cette œuvre difficile.

Des résultats scientifiques de ce Congrès ce ne serait ni l'heure ni le lieu de parler. Personne du reste ne pourrait avoir la prétention de juger des travaux aussi multiples et aussi complexes, et l'activité si grande des nombreuses sections. Certains ont su très heureusement réaliser par leur réunion l'alliance de la médecine, de la chirurgie et des spécialités. Il ne peut être douteux que, dans le monde entier, l'écho de ces assises brillantes dans lesquelles les médecins et les chirurgiens de tous pays ont apporté leur part personnelle de pratiques et de découvertes n'ait des conséquences heureuses sur la diffusion de la science et sur le progrès de toutes les pratiques favorables à l'humanité. L'avenir seul pourra nous faire connaître l'étendue et l'importance de ces suites heureuses.

Mais il y a une constatation que nous pouvons et que nous devons faire aujourd'hui et pour laquelle il y a une parfaite unanimité. L'accueil parfait de nos hôtes, la préparation excellente du travail de tous, les fêtes où s'est allié l'éclat des réceptions les plus brillantes avec la cordialité la plus exquise. Nous ne saurions en exprimer trop haut notre reconnaissance à nos hôtes.

Nous pensons que cette expression serait tout à fait incomplète et insuffisante s'il nous était permis seulement de l'adresser à nos confrères britanniques. Nous tenons à adresser notre hommage reconnaissant aux dames qui, par leur accueil si brillant et si bienveillant, ont bien voulu prendre à la réalisation des réceptions du XVII[e] Congrès une part si capitale dont chacun de nous emporte en son pays le souvenir ému.

GERMANY—Professor Waldeyer spoke.

GUATEMALA—Dr. José Azurdia spoke.

HOLLAND—Dr. Rijnberk spoke.

HUNGARY—Professor Emil von Grósz spoke as follows :

Mr. President, Ladies, and Gentlemen : Only a poet laureate could express my thanks towards you—being no poet at all let this laurel speak for me.

[Dr. de Grósz presented to the President, the General Secretary, and the Secretary of the Reception Committee a spray of laurel tied with the Hungarian colours.]

ITALY—Professor Leonardo Bianchi spoke.

JAPAN—Dr. Inokichi Kubo spoke as follows :

Mr. President and Gentlemen : In the name of Japan I have great pleasure in joining the previous speakers in their expressions of gratitude for the extremely hospitable reception with which we have all met, and in congratulating you upon the important scientific success of this great Congress.

MONACO—Dr. Caillaud spoke.

NICARAGUA—Dr. Alejandro César spoke.

RUSSIA—General Raptchewski spoke.

SPAIN—His Excellency Dr. Camilo Calleja spoke as follows :

Mr. President, Ladies, and Gentlemen : One of the highest pleasures is to teach others what we know. That pleasure has been yours these days, for we Spaniards came here to learn from you. Would we could adequately pay you in kind ! I have tried to do my best, submitting to

your consideration a very important problem, which doubtless is the most imperative and, at the same time, the most adequate for this International Congress of Medicine, as at present medical practice urgently demands the general rules that are required to prevent current abuses in therapeutics, above all in symptomatic medications.

It frequently happens, in the treatment of diseases, that the effects which are intuitively seen as indications are contra-indications, and these are rationally derived from a sound study of the adaptive power of the organism. Thus I have composed a 'Code of Contra-indications', in which the humanitarian precept 'primum non nocere' is involved, as it contains the rules to avoid wrong prescriptions in order to do no harm.

I beg leave to thank the Committee for the honour they have conferred upon me in naming me one of the Presidents of Honour of this Congress.

Now, as your guests, we are highly thankful for that cordial welcome which England knows so well how to give the stranger within her gates; and, as an official Delegate of the Spanish Government, I beg leave to convey to this great nation and to this fruitful Congress its good wishes and the regards of the Spanish medical fraternity.

SWITZERLAND—Dr. Sahli spoke.

The Secretary of the International Association of the Press, Dr. Raoul Blondel, spoke as follows:

L'Association internationale de la Presse médicale manquerait à un agréable devoir si, invitée à se faire représenter dans cette réunion, elle n'exprimait au Comité exécutif de ce Congrès toute sa reconnaissance. Vous avez bien voulu nous compter depuis deux ans parmi vos collaborateurs immédiats. Nous avons donc été les témoins de l'énorme travail que vous avez fourni pour réaliser cette œuvre colossale, qui a provoqué la juste admiration de tous.

Nous sommes des professionels en cette matière, et c'est en connaisseurs, oserai-je dire, que nous avons pu apprécier cette organisation excellente, qui a su tout prévoir, distribuer intelligemment le travail des Sections et réaliser ces fêtes merveilleuses d'où nous revenons en remportant un vif sentiment de gratitude pour cette hospitalité anglaise si large et si délicate à la fois.

Parlant ici le dernier, je sens plus que tout autre la responsabilité de retarder la minute suprême de ce Congrès. Je me borde donc à dire, mais je le dis avec toute la sincérité de mon cœur: Le Président, le Sécrétaire-Général et tous les organisateurs de ce Congrès ont bien mérité de la Science médicale.

Nous leur gardons à tous une profonde reconnaissance et la Presse médicale en particulier n'oubliera jamais la bonne grâce avec laquelle ils ont facilité sa tâche.

A nous revenait la lourde responsabilité de répandre à travers le monde les résultats scientifiques de ces vastes assises internationales. C'est grâce aux heureuses dispositions prises par vous que nous aurons pu parvenir.

Au nom de tous mes collègues de l'Association internationale de la Presse médicale je vous dis merci.

The President then delivered the following address of farewell:

It is now my duty to bid this Congress good-bye. It must have been abundantly evident to every thoughtful person attending these meetings that the influence of medicine and of medical practitioners

is growing enormously. At no previous International Medical Congress has the Government of the country taken so warm and persona an interest in the work of the Congress; so far as I know, at no previous Congress has one of the addresses been delivered by a member of the Government. In every possible way the Government has shown its interest in the work of the Congress, and this is the more noticeable because it is not usual for our Government to concern itself specially in the work of congresses of this kind. No thoughtful person can doubt that in the next generation we shall see an enormous advance in the importance of medicine; it is not a question of rank but of influence. You are well aware that the great commercial class has had a great influence in Europe in endeavouring—successfully at times—to prevent the extension of war. Surely, with the growing influence we possess, we ought to be able to influence our respective countries in this particular regard. I ask you to say is it conceivable, when we have assembled and discussed, without any question of race or country, the various problems of the scientific advance of medicine and its application to the relief of the sickness of humanity, that we should go back to our countries and make no effort to prevent the nations to which we belong from engaging in bloody strife? I do in these parting words beseech my brethren to make their undoubted influence felt in the direction of blessed peace. Now it is my duty to wish you 'farewell'. That English phrase, 'Good-bye,' has been translated 'All good be with you'. I will venture to retranslate it in the words of our Saxon forefathers, and say, 'God be with you.'

The President then closed the Congress.

XVIIth International Congress of Medicine

LONDON: 1913

SECTION XV

Rhinology and Laryngology

SECTION XVI

Otology

PART I

LONDON

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This volume is the first part of the Transactions of the Section. It will not be reprinted, and Members of Congress will not receive another copy of it, but surplus copies will be on sale. The second part will contain (*a*) such reports as arrived too late for insertion in the first part, (*b*) the report of the Meetings, including the Independent Papers.

Ce fascicule est la première partie des comptes rendus de la Section. Il ne sera pas réimprimé, et les Congressistes ne recevront pas un autre exemplaire, mais les exemplaires superflus seront en vente. Le deuxième fascicule contiendra (*a*) les rapports qui ne sont pas arrivés à temps, (*b*) les comptes rendus des séances avec les communications libres.

Dieser Band bildet den ersten Teil der Sektionsverhandlungen. Derselbe wird nicht wieder zum Abdruck gelangen. Etwaige überflüssige Exemplare sind nur käuflich zu erhalten. Der zweite Teil wird (*a*) diejenigen Referate, die nicht rechtzeitig zum Druck gelangt sind, (*b*) die Berichte der Sektionssitzungen samt den Original-Vorträgen enthalten.

CONTENTS OF SECTION XV

PAGE

Discussion No. 1

THE RECENT PROGRESS OF ENDOSCOPIC METHODS AS APPLIED TO THE LARYNX, TRACHEA, BRONCHI, ŒSOPHAGUS, AND STOMACH

Report by Chevalier Jackson, M.D., Pittsburg, Pa., U.S.A. . 1

Discussion No. 2

(Jointly with Section XVI, Otology, Discussion No. 3)

TREATMENT OF DISEASES OF THE THROAT, NOSE, AND EAR BY SALVARSAN AND OTHER ARSENICAL COMPOUNDS

Rapport par M. le Dr. André Castex, Chargé de Cours à la Faculté de Médecine de Paris 35

Referat von Professor Dr. P. Gerber, Königsberg i. Pr. . . . 59

Discussion No. 3

INDICATIONS FOR AND RELATIVE VALUES OF TONSILLOTOMY AND TONSILLECTOMY

Referat von Professor Dr. H. Burger, Amsterdam 111

Report by Dr. J. L. Goodale, Boston, U.S.A. 119

Discussion No. 4

(Jointly with Section XVI, Otology, Discussion No. 5)

SPECIAL TREATMENT OF THE THROAT, NOSE, AND EAR, DURING THE ACTIVE STAGES OF CERTAIN INFECTIOUS FEVERS

Report by E. W. Goodall, M.D. 135

Discussion No. 5

THE PATHOLOGY AND TREATMENT OF MALIGNANT GROWTHS OF THE NOSE AND NASO-PHARYNX (FIBROMA TO BE EXCLUDED)

Relazione del Prof. Gherardo Ferreri, Regia Università di Roma 149

CONTENTS OF SECTION XVI

PAGE

Discussion No. 1

THE SURGICAL ANATOMY OF THE MASTOID

Rapport par M. le Dr. Jules Mouret, Professeur adjoint à la Faculté de Médecine de Montpellier : Que doit-on entendre par Région mastoïdienne chirurgicale ? 205

Discussion No. 2

THE PATHOLOGY OF ACQUIRED DEAFMUTISM

Report by Professor Dr. Holger Mygind, Copenhagen . . 291

Discussion No. 6

CLIMATIC AND OCCUPATIONAL INFLUENCES IN DISEASES OF THE EAR

Report by Clarence John Blake, M.D., Professor of Otology, Harvard University 309

Rapport par M. le Prof. G. Gradenigo, de Turin . . . 319

SECTION XV

RHINOLOGY AND LARYNGOLOGY

DISCUSSION No. 1

THE RECENT PROGRESS OF ENDOSCOPIC METHODS AS APPLIED TO THE LARYNX, TRACHEA, BRONCHI, ŒSOPHAGUS, AND STOMACH

REPORT BY CHEVALIER JACKSON, M.D., PITTSBURG, PA., U.S.A.

KILLIAN, the Father of Bronchoscopy, three years ago wrote, 'A vast amount of material has accumulated which must be tested, observed, and arranged.' If I shall fail, as doubtless I shall, to report the extent to which this material has been tested, I can but point to the next sentence by the same great master, 'Day by day this scientific data increases so that soon it will be difficult to keep pace with it.' Knowing the magnitude of the task and my inability to fulfil it, as well as to prevent overlapping, it was thought best to leave to Professor Killian the difficult task of reporting the recent European progress of endoscopy. To report all, even of American recent endoscopic progress, would be impossible in the present limits of time and space.

Who shall do endoscopy of the larynx, tracheo-bronchial tree, œsophagus, and stomach? For the purpose of collecting statistics, I sent out circular letters of inquiry, requesting reports of cases, to 380 laryngologists in America. I did not include European workers because my distinguished co-referee, Professor Killian, will report to you the European progress in endoscopy. The majority of the 380 were examining the larynx by direct laryngoscopy, but only 79 of them had attempted bronchoscopic or œsophagoscopic foreign body work. Only 35 of the laryngologists cared to do this work, stating that they believed it should be referred to a few men. The views of many are well expressed in the following letter from Dr. Derick T. Vail: 'I have referred such cases to those who are equipped for the work. I think this is a specialty within the specialty which is better followed by one or two men in such a community as this [Cincinnati] rather than by all.' One man had spent three months in a European clinic, studying, among other things, endoscopic technic. After coming home he made the serious error of not doing any tube work at all for over a year, had no trained assistant or nurse, and a numerically insufficient armamentarium, which he brought home with him. He concludes his letter thus, 'I have had only two cases, both œsophagoscopy for coins near the cricoid. Both patients died on the

table. I shall never do another œsophagoscopy, because I do not see a sufficient number of cases to do good work.' The emergency character of many cases demands that each large city shall have at least one man properly trained and equipped for any sort of peroral endoscopy; but every laryngologist should be able to examine the larynx of every child by the direct method without any anæsthesia, general or local, because the direct method is the only way in which the very young child's larynx can be examined, and it is but a platitude to say that a laryngologist must be able to examine the larynx. When it comes to endoscopy of the tracheo-bronchial tree, to do the work promptly, skilfully and safely, and always through the mouth, requires not only facile hands, but, like modern surgery, a trained organization which shall have such constant training as to do its work well. More than this, in tracheo-bronchoscopy, unique in the whole realm of surgery, promptness and precision often make the difference between the life or the death of the patient. There must be no failure at a critical moment upon the part of any factor (person, instrument, or apparatus) in the organization. It is such failure that makes the wide difference between the percentage of mortality shown in the statistics of the work of the occasional operator and of the large clinics. In œsophagoscopy the same holds true in regard to foreign body work, where lack of skill may cause dangerous dyspnœa, which, with lack of promptness and precision in bronchoscopic oxygen insufflation, may prove fatal. In the diagnosis and treatment of disease the same degree of skill is necessary for safe introduction, and promptness and precision are necessary in order that œsophagoscopy for diagnosis will be advised as a simple and minor procedure of slight discomfort to be resorted to on the slightest indication. In no other way can œsophagoscopy accomplish its full measure of usefulness in the diagnosis and treatment of œsophageal disease, and especially of precancerous diseases such as leucoplakia, abdominal and hiatal œsophagismus, stasis, erosion, ulceration, and also in the early detection of malignancy.

Mortality and results of bronchoscopy for foreign bodies. In considering the mortality of bronchoscopy two facts stand out prominently. The first is that we should distinguish between the mortality of the method on the one hand, and the mortality from the lack of promptness and precision in performing it. A number of the reports of the fatal cases show that the patients died upon the table of asphyxia for want of a prompt bronchoscopy. The reports received show very clearly that the mortality was in the main in inverse ratio to the skill, and especially to the experience, of the operator. The best work with the lowest mortality has been done by those who have sufficient endoscopic work to keep not only themselves but their assistants, nurses, in fact their entire organization, in constant training. In 171 cases of foreign body in the trachea and bronchi, bronchoscoped during the last two years by various operators, there were 9 deaths (5·3 per cent.). This does not include 4 deaths due to asphyxia for want of promptness in performing bronchoscopy. Of

these, 156 were removed, 140 by peroral bronchoscopy, 22 by tracheotomic bronchoscopy. Of the 15 unsuccessful cases, 12 were failures to find the foreign body known to be present, and only 3 were failures to remove it when found. In the 12 cases mentioned as failures to find the foreign body are included 4 in which the foreign body had been seen when higher up, but after escaping into the deeper minute bronchi it could not be re-located bronchoscopically, though still showing in the radiograph. The statistics of the author's own clinic and of his cases elsewhere, which are not included in the foregoing, are as follows : Of the last 182 consecutive cases of bronchoscopy for foreign body there was a total of 3 deaths (1·7 per cent.) from any cause whatever within one month, though not all of the cases could be followed this long. Of the 182 cases all were peroral bronchoscopies. The only tracheotomic bronchoscopy for a foreign body ever done by the author was previously reported. It was unsuccessful as to removal. It failed to convince me of any advantage in the lower route, and the disadvantages are many. Of the 182 cases the foreign body was removed in 177. Of the 5 failures to remove a foreign body known to be present, all were failures to find a small foreign body that was in a small branch bronchus close to the periphery of the lung. Two of these cases were recent. The percentage of my failures will doubtless increase in the future, since I now get the cases upon which others have been unsuccessful, and doubtless I shall be equally so ; though I have hopes that the elsewhere-mentioned recently perfected means of locating small bodies in small bronchi near the periphery will diminish for everyone the number of cases in which the intruder cannot be found,

Mortality and results of œsophagoscopy for foreign bodies. Of 193 cases of œsophagoscopy for foreign body, by various operators, the intruder was removed in 155. Of the 38 not removed 26 went down. There were 12 deaths (7·8 per cent.). It is interesting to note that of the 12 deaths from œsophagoscopy for foreign bodies 8 were for bodies in the upper third, 4 of the patients dying during operation, and in all 4 the foreign body was not removed until after death. All had been given chloroform, though this was probably only indirectly the cause of death. In 7 of the 8 the œsophagoscopy was done by operators whose total number of cases was less than 3. In the large clinics (from previously-published statistics), out of 210 cases of foreign bodies in this location, all were removed but 12, and these went down. The mortality in the large clinics was 3 per cent. It is also interesting to note that in the present series of cases there were two deaths from laceration of the œsophagus from violent removal of large foreign bodies ; an artificial denture in one case, a large and rough bone in the other. In both instances the operators stated, in effect, that they believed they could have succeeded in devising methods of safe removal had they realized the danger of œsophageal trauma. Of the 206 cases of œsophagoscopy for foreign bodies in the hospitals of Pittsburg and in the author's work in other cities, the foreign body was removed in 198 and escaped downward in 8.

There were 4 deaths, one in a woman of 56 with advanced nephritis. The other 3 deaths were in patients admitted with severe lacerations of the œsophagus from previous attempts at œsophagoscopy. Four other cases seen *in extremis* are not included, because owing to profound shock no œsophagoscopy was done. There is not, and there never will be, an absolutely safe œsophagoscope that can be used otherwise than with care and caution, for even the soft stomach-tube has caused perforation and death. But all endoscopists are now agreed that skilfully passed under the guidance of the eye, œsophagoscopy is practically without mortality if considered apart from the trauma incident to foreign bodies and their extraction.

Mortality of gastroscopy. That there is practically no mortality from gastroscopy in very careful hands has been shown by the replies to the author's circular letter of inquiry. Out of 110 cases done by eight different endoscopists there was no mortality, within two weeks after operation, from any cause. The author has now examined the interior of 238 living stomachs with the peroral gastroscope, and so far only one patient has died from any cause whatever within one month after the gastroscopy. As previously reported, this patient was moribund from a bleeding ulcer of the stomach when admitted to the hospital. But taking the figures just as they stand the mortality is only a fraction of 1 per cent.

Radiography in its relation to peroral endoscopy. Excellent progress has been made in the radiographic localization of foreign bodies. This is especially true in regard to the technical improvements which have rendered possible practically instantaneous radiography, as it has quite recently been recognized (Tilley and others) that an instantaneous radiograph will often show foreign bodies not visible with longer exposures. A deep inspiration held during the exposure creates an artificial emphysema which causes the foreign body to show, because it lessens the density of the thorax ; though it must be borne in mind that the more horizontal position of the ribs and the displacement of the viscera, including the foreign body, must be allowed for in the localization. The steady progress made by the radiographer in lateral radiography of the thorax has not only been of great aid in the general localization from bony and visceral landmarks, but also in conjunction with the caliper-guide suggested by Dr. Boyce and developed by the author. In conjunction with the lateral radiograph the caliper-guide will bring the point of the bronchoscope in close relation with the foreign body by greatly diminishing the number of small bronchial tubes to be searched, this method being used, of course, only in the case of small foreign bodies which have fallen into a very small bronchus far down or far out near the periphery of the lungs. The lateral placement of the point of the bronchoscope depends upon a mark placed on the skin by the radiographer who determines the point by an antero-posterior radiograph. Another aid in this same class of difficult cases has been used by the author. A positive transparent film of the tracheo-bronchial tree is laid over the negative

of the patient showing the foreign body, when the foreign body will show through the transparent tracheo-bronchial tree of the overlying positive film. In placing the film, bony landmarks are not reliable and visceral landmarks are necessary. The two important visceral landmarks are the dome of the pleura and the dome of the diaphragm. It is needless to say the tracheo-bronchial tree necessarily lies in the body of the lung between these two landmarks, and lines corresponding to these are placed on the film. All this work is done, of course, in a darkened room with a strongly illuminated shadow-box ; and in the event of the foreign body showing very faintly on the radiograph of the patient, it is strengthened by an ink-mark on the uncoated side of the negative, which can be readily erased afterward if desired. There are twelve sizes of film positives, from which one to correspond to the size of the patient's negative is selected. One source of error, of course, is that the positives of the tracheo-bronchial tree are made from the tracheo-bronchial tree of a cadaver, whereas bronchoscopic study of the tree shows that it is not quite in the same position as the living. I have found the injection preparations of Brünnings to come nearer those of the living tree than any other that I have been able to find, and therefore I have used them in making the positive films. Lateral positive films are useful in case of children. It is customary in the interpretation of a radiograph, when one lung shows dark and the other light, to consider that the dark side contains the foreign body which has occluded the main bronchus with perhaps compensatory emphysema on the opposite side. Iglauer reports a very interesting case where this reading was erroneous because the foreign body had, by a valve-like action, imprisoned more air in the obstructed side, so that there was a very marked emphysema shown by the radiograph on the obstructed side. The negative report from the radiographer remains to-day as it always has been, unreliable, because many bodies are not opaque to the ray, and, moreover, the foreign body may not be the same as that of which we get a history. In addition to this, even metallic bodies at times do not show. For instance, in one of the author's cases, that of an enormous woman of 53 years, expert radiographers, for a period of two years, made quite a number of exposures, but were unable to demonstrate a tack which they finally demonstrated to be present, and which the author removed from the right bronchus. Such occurrences will doubtless be less and less frequent because of the steady advance in the technical perfection of radiography. A number of recent cases have made it quite clear that it is necessary to do a bronchoscopy if there is any reason to suspect from the history that there is a foreign body located somewhere in the air-passages or œsophagus, notwithstanding a negative ray finding and a total absence of symptoms, for it is remarkable how tolerant the trachea, bronchi, and œsophagus become to the presence of foreign bodies after the initial symptoms immediately following the accident have subsided. Radiography has been of great aid in the study of diseases of the œsophagus, but it has been clearly demonstrated that they are apt to

be quite misleading if taken alone without endoscopy. For instance, one case sent to the author for the removal of a diverticulum was found not to be a diverticulum at all, but an enormous dilatation of the entire œsophagus due to an abdominal œsophagismus. This dilatation was filled with food, so that when the bismuth porridge was swallowed it simply lodged on top of the mass of food. The food did not throw a shadow, but the porridge did in a mass closely resembling a diverticulum. On introduction of the œsophagoscope, nearly two quarts of stale food were removed, and the diagnosis of abdominal œsophagismus was readily made. Furthermore, to attempt to make a diagnosis and treat diseases of the œsophagus on the ray findings alone, or supplemented with laboratory and blind methods is to ignore the greatest of recent progress in the treatment of diseases of the œsophagus. It is not enough to know that there is a diverticulum, a dilatation, or a spastic stenosis. To do effective

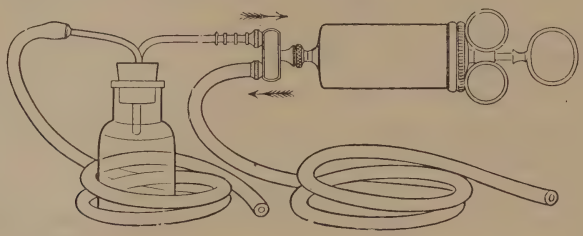


FIG. 1. Aspirator for œsophagoscopy with additional tube connected with the plus-pressure side for use in case of occlusion of the œsophageal drainage-tube.

work it is necessary to know the condition of the mucosa, and especially to know of the presence or absence of lesions in the hiatal and abdominal œsophagus, and in the cardial portion of the stomach. There is absolutely no way of knowing these things without the œsophagoscope.

Instruments. Quite a number of new forms of illumination and new tubes for peroral endoscopy have been devised, and they practically all have done good service in the hands of skilful men. In the statistics which the author gathered there was practically no difference either in the mortality or the percentage of successful removals of foreign bodies between the different kinds of tubes and illumination. Far more depended on the individual skill of the operator. Doubtless the best instrument for each operator is the one with which he has practised most. Efforts to produce jointed and angular œsophagoscopes and gastroscopes continue, and all such should be encouraged (provided great care is exercised in their use), because all effort results in increased attention to the œsophagus and its diseases. There is absolutely no hope that any œsophageal instrument will ever be devised that will be safe unless carefully used, because even the soft rubber stomach-tube has been known to cause fatal perforation. One of the most successful of the angular œsophagoscopes is the indirect one of Lewisohn. I saw it passed upon a patient with practically no discomfort, and the view was good. In its present

form it is, of course, adapted to simple inspection, not for the removal of foreign bodies or specimens, nor for probing or palpation, wiping or medication. For dealing with foreign bodies and disease high up in the œsophagus, the author has found exceedingly useful an elongation of his laryngeal speculum. This instrument is 25 cm. long for use in adults, and with it there is very much less risk of overriding foreign bodies in the high situation than with the œsophagoscope. This œsophageal speculum has also been found particularly useful for the breaking up of those rare congenital webs first described by Mosher and Clark, and of the high strictures of the œsophagus which follow decubitus ulcers resulting from the mixed infections complicating enteric fever, scarlatina, diphtheria, and like conditions, and the breaking down of gummata. All of these yield quite readily to the breaking up and stretching with this speculum.

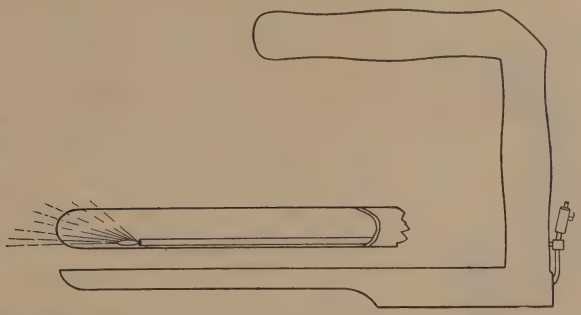


FIG. 2. Œsophageal speculum for foreign body work and for operations upon the upper end of the œsophagus.

Some few forms of cicatricial stenosis, especially those following the breaking down of the gummata, have a tendency to recur, and it is necessary to repeat the treatment frequently, but in most of the conditions a very few treatments are sufficient, a divulsor being only occasionally required. Yankauer has perfected an aspirator for the removal of secretions operated by a small exhaust fan in connexion with an electric motor. He has also used a jet of compressed air blowing sidewise across the proximal tube-mouth to blow away the secretions coughed out by the patient, thus preventing them from soiling the mirror of the Brünnings lamp or Kirstein head-light. It certainly is a comfort to see the stream of coughed-up secretion being projected off sidewise instead of getting it in one's face. Ingals uses an electric aspirating pump originally devised for massage of the ear. A number of endoscopists are using various forms of aspirators attached to a water faucet. In using these it is necessary to exercise precaution, if commercial circuits are used for illumination, lest the current be ' grounded ' through the water-pipes, especially when withdrawing long aspirating tubes. The author prefers, for œsophagoscopy, an aspirator in the wall of the œsophagoscope or gastroscope, the exhaust being an aspirating syringe. The positive pressure

side of the syringe also has a soft rubber tube, and in case the aspirating canal in the wall of the esophagoscope becomes obstructed, a change of the soft rubber tube from the negative pressure to the positive pressure will force out any clots or other obstructions which may have entered the canal. The author does not use any form of aspirator, either in the wall of the tube or otherwise in bronchoscopy. He has found that the best of all ways to remove abundant secretions and blood during bronchoscopy is to insert a large swab on the usual long Coolidge sponge-carrier, pushing it down until the large gauze sponge goes beyond the distal end of the bronchoscope. Then the patient will cough the bronchoscope full of the fluid, and the withdrawal of the carrier and swab will pull up often as much as an entire tubeful of secretions at a time, just as the water above the plunger of an ordinary pump is lifted by the plunger. This is one of the advantages in working under slight anæsthesia or none at all. Contrary to many of the statements that have appeared, the form of aspiration used by the author has absolutely nothing to do with distal illumination. There is no form of illumination that will enable the operator to see through a pool of blood and secretion. How to remove the fluid in the least possible time is the study of all endoscopists, and the aspirator in the wall of the œsophagoscope is used because there is no interruption of the work. It is a common thing at a gastroscopy to remove a pint of fluid without any interruption. Safety-pin closers continue to be devised. The last one by Hubbard is quite ingenious, and doubtless will be useful for purposes other than closing pins. As is well known, wide gagging of the mouth interferes very seriously with endoscopy. All that is necessary is to keep the patient from biting the tubes. For this purpose the bite-block devised by Boyce and modified by my assistant McKee, has been of great service. The anæsthetising tube is used for insufflation of anæsthesia during œsophagoscopy, though it is rarely that I use general anæsthesia. If general anæsthesia in bronchoscopy is used, the anæsthetic is insufflated through the bronchoscope. For amputation of the cancerous and the tuberculous epiglottis, I have found most useful a very heavy snare canula armed with No. 5 steel piano wire, and fitted to the massive handles of the Peters tonsil snare. By firm downward pressure on the canula the loop can be made completely to amputate the involved epiglottis *en masse*, as demonstrated by dissection of the removed tissues. The advantage of removal *en masse* over morcellation is generally accepted. The canula is passed beside the laryngeal speculum, not through its lumen.



Fig. 3. Thimble-bite block originally suggested by Boyce and improved by McKee and McCready. Ether is insufflated through the tube, if needed, for œsophagoscopy.

Preparation of the patient. In all instances of obstructive diseases of the œsophagus, it is very essential to have the patient regurgitate after taking a copious drink of water in order to make sure that dilatations or diverticula, if present, may be emptied. Diverticula can be emptied sometimes by pressure on the neck, but in any event the presence of large quantities of food in the œsophagus interferes very seriously with œsophagoscopy and introduces a great element of danger if a general anæsthetic be used. In all cases in which time permits, it is very essential before bronchoscopy, œsophagoscopy, or gastroscopy that the patient be prepared as for any operative procedure by a cathartic, rest in bed, and the mouth should always be thoroughly cleansed by numerous brushings of the teeth and rinsings with 30 per cent. alcohol, which is the best non-toxic antiseptic. The author's early insistence upon strictly aseptic operating-room technic in all forms of peroral endoscopy, while much ridiculed at the time, has come recently to be recognized everywhere as quite essential in a procedure which necessarily frequently comes in contact with tuberculosis, pneumonia, diphtheria, erysipelas, and other infectious diseases. It is a matter of great gratification to the author that in fifty examinations of swabs used for wiping secretions from the bronchi, in no one instance was there found any trace of epithelial cells or of such forms of bacteria as would prove that the instruments have been in any way contaminated by contact with the mouth. This is worthy of note in connexion with the obtaining of inoculation material for the production of autogenous vaccines in cases of chronic bronchitis.

Position of the patient. For adults the sitting position is very satisfactory for diagnosis. For infants and children the dorsal position is better because of better control of the patient. In foreign body cases, whether adults or children, the patient should always be recumbent, never erect, because in the erect position gravity works against the operator, and the foreign body may reach a deeper point in the air-passages than it would in the recumbent position. This is particularly true of foreign bodies in the larynx and pharynx, which should never be touched unless the patient is in the Trendelenberg position. Quite a large proportion of the foreign bodies in the bronchi that have been sent in to the author were originally in the larynx or pharynx, and fell down when displaced by the attempts of the operator, who first saw the case, to remove the intruder. There seems to be quite prevalent a faulty conception of the position of the patient. The correct position for direct laryngoscopy was demonstrated by Kirstein many years ago. It consists chiefly in an extreme anterior movement of the cervical spine, with extension at the occipito-atlantal joint. In the recumbent position the anterior placing of the cervical spine means raising the head above the level of the table, as demonstrated by Boyce. To allow the head to ' hang over the end of the table in the Rose position ', as recommended in so many text-books, is to render endoscopy extremely difficult or impossible. Dr. Johnston has demonstrated the usefulness of flexing the head for

direct laryngoscopy on the recumbent patient, putting a pillow under the patient's head, the operator standing to the left side of the patient. The Johnston position is particularly advantageous where the operator is without a regularly-trained assistant with whom he is in the habit of working, because any one can hold the head on the pillow. The position is not adapted to bronchoscopy, though Johnston uses it to start



FIG. 4. Showing the best position of the operator, patient, and assistant for direct laryngoscopy on adult patients under local anæsthesia. The sitting position of the operator renders laryngeal exposure easy for patient and operator; whereas the usual standing position of the operator throws the patient into a posture that renders laryngeal exposure difficult, as well as throwing the trachea out of line. The assistant's right index finger is used for counter-pressure either backward or laterally as needed.

the tube, and then the head of the patient is brought into the Boyce position. This requires skill and care to prevent any trauma of the trachea in making the change. The Boyce position requires a trained assistant, and operator and assistant must work together, under which circumstances the Boyce position is ideal for bronchoscopy, œsophagoscopy, and gastroscopy. I have found the Johnston position exceedingly useful in disease of the cervical spine, where the children were fixed in an apparatus which I did not need to disturb to get an excellent view

of the larynx. The most important point of all position in tube work is the extreme anterior position of the cervical spine, and for this the operator should be sitting also, if the patient is in the sitting position, as shown in the photograph. The lateral position for bronchoscopy and œsophagoscopy has found but little favour in America. Its only real advantage is the facility with which secretions drain from the lowermost corner of the mouth. This can be accomplished almost as well in the dorsal position with a wick of gauze hanging out over from the pharynx, the outer end the longer. If secretions are too thick to drain by capillarity, the gauze is frequently replaced by a fresh piece. An aspirating drainage-tube of metal connected with the author's œsophagoscopic aspirator, is hooked into the lowermost portion of the patient's mouth in bronchoscopy, thus ridding the mouth of secretions while the patient is in the dorsal position.

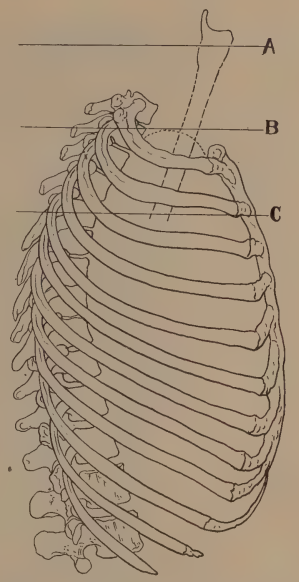


FIG. 5. Schematic illustration of normal position of the intra-thoracic trachea, and also of the entire trachea when the patient is in the correct position for peroral bronchoscopy, such as the original Kirstein position, or that shown in Fig. 4. When the head is thrown backward (as in the usual or in the Rose position) the anterior convexity of the cervical spine is transmitted to the trachea of which the axis is no longer straight.

Anæsthesia. The greatest of recent progress in anæsthesia is toward the use of no anæsthetic at all. In general anæsthesia the total abolition of the cough-reflex should only be for short periods. The facile operator will do good work in many cases in spite of moderate degree of cough, and the cough can be advantageously used to rid the air-passages of secretion as elsewhere herein explained. After a short period of tubal contact in bronchoscopy coughing lessens and often practically ceases, especially in infants, without any anæsthetic having been used. In general surgery anæsthesia is required for three purposes: (1) the obtunding pain (really analgesia); (2) the abolition of reflexes (relaxation); (3) for psychic effect (mainly abolition of apprehension). For peroral endoscopy analgesia is not required, for the pain, in careful work, is exceedingly slight. Anæsthesia for the lessening of the reflexes is only occasionally necessary. Apprehension can be abolished or greatly diminished by the control of the patient's mental state through the personality of the operator. The degree of this control varies widely with the personal equation of the operator as well as of the patient. The operator who will make a special study of it can keep his patient free from apprehension. He

will keep his patient's mind fixed on the task of breathing slowly, deeply, and regularly, and will get along without any anæsthetic and do better work than another operator with a general anæsthetic. I quite agree with Brünnings that the use of general anæsthesia to overcome faults in technic is unjustifiable. The author's personal views are here submitted for discussion.

1. *In children* under 6 years of age no anæsthetic, general or local, should be used for direct laryngoscopy, peroral bronchoscopy, or œsophagoscopy except that general anæsthesia may be advisable in case of very sharp foreign bodies as safety-pins or fish-hooks threatening perforation from coughing or vomiting. Even in such cases general anæsthesia is absolutely contra-indicated if there is the slightest dyspnœa. Cocaine is needless and dangerous in children.

2. *In adults* no anæsthesia, general or local, is necessary for œsophagoscopy for diagnosis or for foreign bodies, save in case of very large and sharp foreign bodies to relax the œsophageal and accessory musculatures, thereby lessening the liability to local trauma from the extraction of very large foreign bodies that are sharp or rough through a spasmodically contracted lumen. The use of a general anæsthetic in cases of foreign bodies in the œsophagus enormously increases the danger of respiratory arrest as soon as manipulation of the foreign body is commenced, and especially if the position of the patient be faulty. Therefore chloroform should never be used, though it may be added, if necessary, for relaxation after the stimulant effects of ether ensure safety. Respiratory arrest is of no consequence if the operator be facile at bronchoscopic-oxygen insufflation. Local anæsthesia of the œsophagus is needless. The œsophagus is quite insensitive, as any one can demonstrate upon himself by swallowing very hot tea. After the cricoid level is passed there is no sensation. Therefore local anæsthesia, if used at all, should be limited to the laryngopharynx, but in most cases even this is quite unnecessary, and most patients can distinguish little difference in discomfort. For direct laryngoscopy for foreign bodies in adults cocaine should not be applied with any form of applicator. A spray is much less likely to dislodge the intruder, and often none at all is needed. For diagnosis and the removal of specimens or neoplasms local anæsthesia is often required. For the removal of small angiomata and œdemata of the larynx general anæsthesia may be necessary, because these tumours shrink and may disappear under local anæsthesia so as to render accurate work impossible. For bronchoscopy for diagnosis anæsthesia of the larynx including the epiglottis is needed. It is better not to apply cocaine below this point until a primary inspection has been made because of the altered appearances due to ischæmia.

3. If tracheotomy is to be done, whether in adults or children, it should be under local infiltration anæsthesia, never, under any circumstances whatever, under general anæsthesia. Davis reports the use of the intra-muscular injection of ether into the buttock of a child primarily

rendered unconscious by ethyl chloride. Intra-tracheal insufflation anæsthesia calls for direct laryngoscopy for the insertion of the anæsthetizing tube into the trachea, especially to eliminate the possibility of laryngeal disease, as insisted upon by Elsberg. Insufflation anæsthesia is used by Henry Janeway for gastroscopy, and he has done remarkable work in the insufflation of nitrous oxide and oxygen for this purpose,



Fig. 6. Lateral radiograph (by Dr. George C. Johnston) of a child of 4 years, showing the normal direction of the trachea. A pale streak is seen extending backward as well as downward, ending at the foreign body in the right bronchus. There is a narrowing of this streak at the bifurcation, representing a flattening from before backwards. Compare schema.

thereby doing away with one objection that has been raised to gastroscopy, namely, ether anæsthesia. In the last 107 bronchoscopies and œsophagoscopies for foreign bodies in children under 6 years of age, done in my clinic, no anæsthetic, general or local, has been used. Ten of these cases have been in the children of physicians, every one of whom expressed his delight that no anæsthetic was used, and in each instance the physician was present at the removal of the foreign body from his

own child. There is no question in the author's mind but what all forms of anæsthesia, general or local, introduce a great element of danger into the handling of foreign body cases in children, more especially when chloroform is used. In adults, and with ether, the risk is probably very slight.

Technic. Many interesting points in regard to technic were brought out by replies to my circular letter of inquiries. Space forbids taking up these, but one point is worthy of mention. I was very much astonished to find some men were still using a mandrin to pass an œsophagoscope, and one endoscopist uses a mandrin even for passing a bronchoscope. In œsophagoscopy the tubes should always be passed by sight, lest a foreign body be overridden or a diseased wall encountered. In bronchoscopy a mandrin is a needless encumbrance, and I believe has altogether been abandoned by bronchoscopists of large experience everywhere.

Direct laryngoscopy. Considering the fact that it is now eighteen years since Kirstein demonstrated the feasibility of examining the larynx by direct methods, and considering the developments by Killian, who made it practical, and by Brünnings, von Eicken, and others, and taking into consideration also the fact that the larynx of any child can be examined in a few seconds without any anæsthesia, either general or local, it is a very remarkable thing that there remains in the world any laryngologist who is to-day not availing himself of this method, but is depending upon an inferential diagnosis from the appearance of the epiglottis, for it is but seldom that we can see more than this by the indirect method in younger children. There has been a tendency in the last few years to question the value of direct laryngeal methods for diagnosis in adults. While it is undoubtedly true that the indirect method is of the greatest value and always will be, it is also true that in many instances diagnosis of laryngeal infiltrations is frequently cleared up by the different view-point obtained in direct laryngoscopy. Brünnings states that the early hopes of direct laryngeal operating have not been entirely fulfilled. This is true only so far as it relates to the hopes of those who did not fully understand the work, and who supposed that because the operative procedures were direct that it would be easy without special training for the operator already facile in ordinary faucial and pharyngeal work. Of course it is true that until a man has worked a long time, the difficulty of which Brünnings speaks, and which I presume he has observed in pupils, namely, that 'delicacy and fineness of movement of the right hand is considerably interfered with by the left hand being exerted simultaneously'. Yet on precisely the same basis it might be argued that the pianoforte is a failure, because the accuracy of execution in the treble is interfered with because 'one hand only and that the left one' has to pound out those rich, full, deep, wide interval chords that are the great charm of the pianoforte. The difference is that the pianist expects to practise for years to acquire skill, whereas many laryngologists think that long

years of laryngology should enable them to do skilful endoscopic work with little practice. If the pupil is taught to practise constantly the introduction of the laryngeal speculum with the left hand only, and using his right hand only to move the patient's upper lip out of the way of injury (with the index finger), he will soon acquire perfect mastery of prolonged left-handed exposure and co-ordinate movement of the two hands in direct operating. I believe this has been too little insisted upon, though it is one of the most important for the advancement of the usefulness of



FIG. 7. Illustration of a positive film used for overlaying to assist in localization of foreign bodies or lesions in the thorax. The lower white line (D, D) corresponds to the diaphragm, the middle line (P, D) to the dome of the pleura. These lines assist in placing the overlay. The upper line (V, C), corresponding to the vocal cords, is occasionally useful. Twelve photographic enlargements are on hand, so that a film of the size (rather than the age) is available for any sized patient.

direct endoscopic operating. Greater skill can be acquired and all work expedited if all handling of tubes and specula be done with the left hand only, leaving the right hand free at all times for the manipulations of forceps and other instruments. Trained in this way, I believe the pupil will not be disappointed, especially if he has a trained assistant to place the patient's head and neck in the proper position, and to make counter-pressure with the index finger upon the thyroid cartilage externally. This counter-pressure lessens, to a very great degree, the amount of pressure exerted with the left hand upon the tip of the speculum, with consequent less exertion with the operator's left hand, and of course less discomfort to the patient. Papilloma in adults is always curable by

repeated removals by direct laryngoscopy with interim applications of alcohol. Eventually there results a thickening and hardening of the laryngeal mucosa, which makes a bad soil for the growth of papillomata. The author has never yet failed to cure a case in adults in this manner; but the treatment has extended over a number of years in some instances. Papillomata in children in many instances will yield to the same treatment. In a few, however, it has been necessary to do laryngostomy, with Röntgen-ray applications to the open larynx. The author is utterly at a loss to understand why writers on this subject persist in advising general anæsthesia for the removal of papillomata in children. A general anæsthetic is absolutely contra-indicated in every disease of the larynx in children. It is, moreover, absolutely unnecessary. Cocaine is very toxic for children, and should never be used, and it also is quite unnecessary. Skilful direct removal is almost painless in children, and after a number of sittings children usually do not even cry, though infants usually do. The work of Harmon Smith on fulguration for the treatment of papillomata in children by endoscopic methods has yielded excellent results, and marks a distinct advance in the treatment of these stubborn cases. Abundance of evidence has been forthcoming proving the great usefulness of the galvano-cautery in the treatment of tuberculous infiltrations in the larynx, and all of the laryngologists who have used the direct methods for these applications are enthusiastic as to the precision with which the caustic point can be applied. The direct method exposes to view the anterior surface of the posterior wall of the arytenoid masses, and thus the point can be applied practically perpendicularly to the surface, which is in great contrast to the indirect method by which a more or less lateral application of the point renders accurate puncture more difficult and sometimes impossible. Furthermore, it matters little how intolerant the patient may be to the laryngoscopic mirror. He cannot in any case whatsoever prevent the skilful operator from making an accurate application. Direct laryngoscopy has opened up a new field in the local treatment of tuberculous lesions. The usefulness of endoscopy in malignant disease of the larynx ends with the inspection of both sides of the party wall and the taking of a specimen. Following the initiative of Sir Felix Semon and Mr. Butlin, my series of cures of malignant disease of the larynx from the operation of thyrotomy in intrinsic disease of small extent has been such a satisfaction to me that I feel that it would be a step backward to attempt endo-laryngeal removal, and I have seen nothing in the recent progress of endoscopy to lead me to modify my views. The precision with which a specimen can be taken by the endoscopic method, so as to include a little of the bordering normal tissue, gives a character of certainty and reliability to the preliminary examination of a specimen that seldom pertains to the little fragments so often obtained by the indirect method.

Bronchoscopy. The closure and removal of open safety-pins in the œsophagus has now become a relatively common procedure with the

endoscopist, but the closure and removal of an open safety-pin from the trachea by oral bronchoscopy has been recently very skilfully done by Hudson Makuen. Space forbids mention of many mechanical problems of removal successfully coped with. A number of them go to prove that,



FIG. 8. Illustrating a positive radiograph of the tracheo-bronchial used as a film for overlaying to assist in localization of a foreign body. The left-hand illustration shows the film laid over a negative of a patient in whose left main bronchus was a pin. Localization verified by bronchoscopy. The shadow of the pin is strengthened with ink.

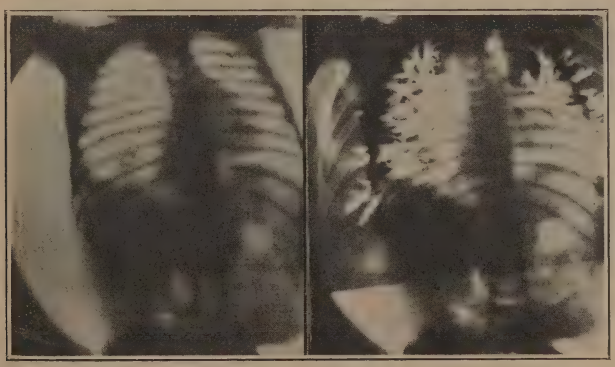


FIG. 9. Illustrating on the right a lung abscess (retouched). On the left the abscess is localized in the right inferior lobe bronchus by the method of overlaying. The localization coincided with the endoscopic findings when the abscess was evacuated bronchoscopically.

however difficult the problem, the temptation to remove a foreign body by main strength must be resisted. Instead, careful thought will evolve a plan, by a new device or by a modification of the old, or by version of the foreign body—in short, some means by which the high percentage of mortality sure to attend forcible extraction may be obviated. Benign tumours within the lumen of the trachea seem to be quite rare, though

possibly when bronchoscopy for diagnosis comes to be more frequently resorted to they will be more common. Emil Mayer reports the very interesting case of a child with a peculiar crowing respiration and physical signs of absolute obstruction of the lower portion of the right lung ; ray examination negative. At bronchoscopy, a papilloma was found, filling the lower portion of a main bronchus, and its endoscopic removal was followed by a cure. The endo-bronchial treatment of asthma and of other diseases, advocated by Ephrian and others, has been used by Emil Mayer, Yankauer, Ingals, Horn, and others in America, and is becoming everywhere a recognized method of treatment in obstinate cases. As Ingals has stated, it is not to be advocated for general use by every one without previous endoscopic training. As von Eicken has pointed out, it is unnecessary to introduce the bronchoscope in order to medicate the bronchi locally. Dr. Emma E. Musson introduces a soft tube through the glottis by means of the laryngeal speculum, and has found that after the examination of local conditions is made bronchoscopically, an occasional bronchoscopy during the course of medication will suffice.

Indications for bronchoscopy. The following indications for bronchoscopy are offered for discussion that they may be added to, or subtracted from, as the consensus of opinion may indicate. In most instances an œsophagoscopy as well as a bronchoscopy is indicated, especially in cases of suspected thoracic disease.

1. The appearance, in the radiograph, of a foreign body or of any suspicious shadow.

2. In any case in which there is a clear history of the patient having choked on a foreign body, and in which the foreign body has not been afterwards found.

In this connexion it must be borne in mind that foreign bodies may be multiple, as in one case of my own in which a bronchoscopy was not done because, after the accident, a gourd seed was found in the stools. Three months later I removed a gourd seed from the bronchus. The child had been playing with a number of gourd seeds.

3. In any case in which there are signs of stenosis of the trachea or of a bronchus.

4. In the absence of any foreign body history, the patient showing symptoms of pulmonary tuberculosis in which the bacilli cannot be found in the sputum, and especially if the physical signs are at the base, particularly the right base, and above all, if there are physical signs of pleural effusion.

A number of instances have been recorded (Ingals and others) in which prolonged sojourn of a foreign body in the lung had produced symptoms for which the patient was repeatedly tapped for a pleural effusion without getting fluid. I have myself observed two such cases, one in which the foreign body was present seven years, and the other in which it was present ten years. These patients had both been treated for tuberculosis, and both had been tapped.

5. All cases of bronchiectasis should be bronchoscoped for foreign bodies and also for local treatment.

Emil Mayer found a foreign body in one case of bronchiectasis where its presence had never been suspected. I, myself, have found bronchiectasis present in two cases of prolonged sojourn of a foreign body in the right inferior lobe bronchus, though in both of my cases the foreign body had been discovered radiographically.

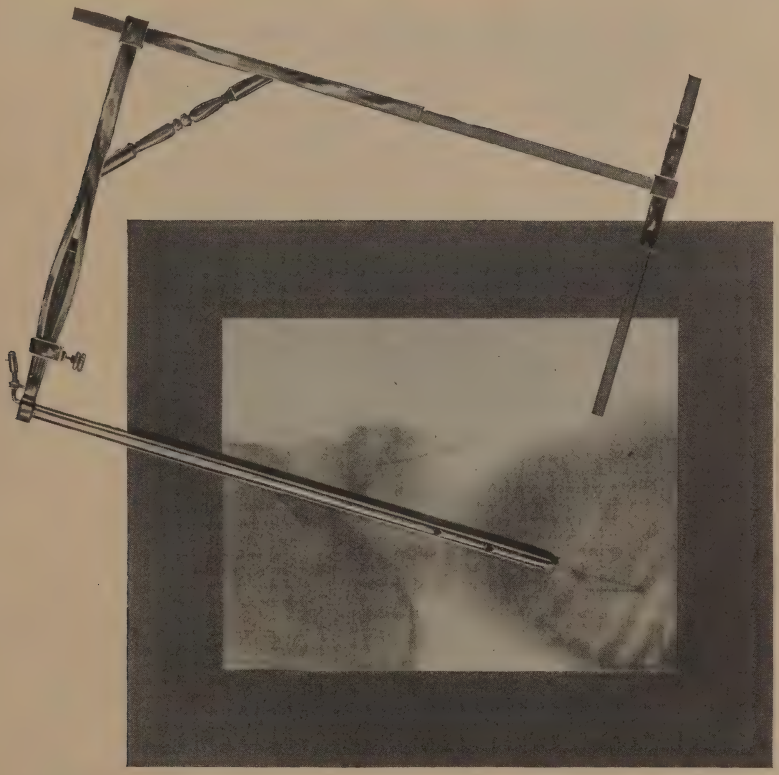


FIG. 10. Illustrating the position of the calliper-guide in getting the adjustments by which the point of the bronchoscope can be brought, later at bronchoscopy, in close proximity to a foreign body. For use in case of small foreign bodies in minute bronchi. Suggested by Dr. John W. Boyce.

6. Every case of dyspnœa, except, of course, pneumonia and similar well-understood conditions, calls for bronchoscopy.

7. Every case in which tracheotomy does not relieve the dyspnœa should be bronchoscoped to determine why the tracheal canula does not give relief.

8. All cases of hæmoptysis, which are not definitely proven to be tuberculous, should be bronchoscoped for diagnosis, and in case of severe bleeding endoscopic packing, as advised by Killian, should be tried.

I have examined a great many cases of hæmoptysis to determine the

cause of bleeding from the lungs. All of these cases have been, at some time or other, diagnosticated tuberculosis, and many of them proved to be tuberculosis. But among the non-tubercular ones, there were three cases of aneurysm, two of cancer, three of lues. In quite a number of cases it has been possible to make a diagnosis of tuberculosis from lesions found and specimens removed in cases where the physical signs had been negative, or at least negative opinions had been given by competent physical diagnosticians.

9. Every case of paralysis of the recurrent nerve, the cause of which is not positively known.

10. Any case of thoracic disease in which any element of doubt exists, valuable information may be gained by bronchoscopy.

11. In case of doubt as to whether bronchoscopy should be done or not, bronchoscopy should always be done.

Subglottic œdema. The cause of subglottic œdema is worthy of our most careful consideration. In the author's opinion, it makes a very great difference what instrument is used and how it is handled. We all know that the O'Dwyer intubation tube may stay in six or seven days without being followed by subglottic œdema, except in rare instances. The O'Dwyer tube is short, so that no leverage is exerted, and it is never larger than will comfortably fit the lumen of the subglottic larynx. To my mind, that is the keynote to the situation. Large-sized tubes, roughly manipulated, and what is more important, manipulated without a trained assistant whose business it is to keep the head in such position that the trachea will at all times be in line with the axis of the tube, so that the laryngeal tissues do not become the fulcrum upon which the bronchoscope may bear and press. Judging by the printed reports, it is customary with most operators to use a far larger diameter of tubes than is good for the infant larynx. Since 1911, by closely observing these points, not one case of subglottic œdema has occurred in the practice either of Dr. Ellen J. Patterson or myself in thirty successful removals of foreign bodies in the trachea and bronchi of infants under one year, the youngest $2\frac{1}{2}$ months. Every case was done by oral bronchoscopy. In a number of instances, the child has become dyspnœic within twenty-four or thirty-six hours after the bronchoscopy, but on passing the bronchoscope, a large quantity of secretion was removed with complete re-establishment of quiet respiration and the disappearance of the dyspnœa. In other words, the child was drowning in its own secretions, because children expectorate with difficulty. This was especially noticeable in cases where the foreign body had been some time in the air-passages, or where it was of an irritating nature like a peanut kernel. We have had altogether five cases of peanut kernels in the bronchi and in all there was present a severe bronchitis with abundant secretion. From these observations, and from the written report of cases where tracheotomy has been done for supposed post-bronchoscopic subglottic œdema, the author feels sure that in a portion of these cases complete relief would have followed

the peroral bronchoscopic removal of secretions, and he would especially urge that all cases of post-bronchoscopic dyspnœa require not only a direct laryngoscopy, but a bronchoscopy for the removal of secretions which infants cannot expectorate. Tracheotomy, except in extreme cases, should not be done until after this, save when needed in following the cardinal rule of tracheo-laryngeal surgery that 'in a dyspnœic patient tracheotomy should always be done early, never late.' Unfortunately,

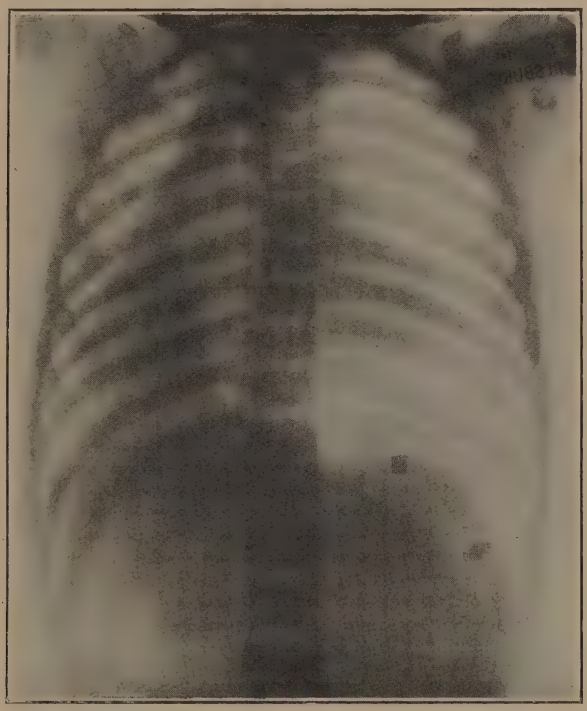


FIG. 11. Radiograph by Dr. George C. Johnston, showing foreign body (primer) at the bottom of the pleural cavity of a child of 4 years. Foreign body removed by pleuroscopy.

the statement crept into the early literature that the child's larynx is more easily stretched than that of the adult, and this had led to the use of tubes entirely too large. In the author's clinic, both Dr. Patterson and myself use tubes of 4 mm. and 5 mm. internal diameter, for children under six years of age, the 4 mm. tube being for infants under one year. Our youngest patient from whom a foreign body was removed was an infant of $2\frac{1}{2}$ months. This was a common pin removed from the right bronchus with a tube of 4 mm. internal diameter. Faulty direction of the tube on introducing may easily cause trauma by gouging into the subglottic wall, if the axis of the bronchoscope and that of the trachea do not coincide at the moment the tube passes the glottis. In ten different

publications within the last two years the operators stated that they placed the patient in the Rose position. If the patient actually was in the Rose position, it was just exactly rightly placed for the bronchoscope to gouge into the subglottic wall, and to risk a production of subglottic œdema, especially if the head of the patient was a little more to one side than the other. Heavy hand and heavy handles, it must be conceded, are more likely to cause injury to the subglottic tissues than light tubes delicately manipulated. Therefore I think that heavy introducing instruments should be removed, leaving only the light delicate bronchoscopic tube for endo-bronchoscopic work.

Peroral or tracheotomic bronchoscopy. Which? Unfortunately, the statement has crept into the literature that in infants or small children it is preferable to do a tracheotomic bronchoscopy. In the opinion of the author this is due to two things, namely, the ignoring of the precautions mentioned under subglottic œdema, and also the fact that when this statement was originally made, illumination was not in the relatively perfect condition that is seen on the instruments of to-day. In making this statement the author hopes he will not be misunderstood as referring to any difference between distal and proximal illumination. He means simply that the light on all forms of instruments to-day is far superior to what it was in the early days. At that time it made a great difference whether the tube was a long one or a short one. To-day I doubt very much whether any one can tell by looking through the lumen whether the tube is 30 cm. or 50 cm. long. The author has often tested this and found the observer unable to tell with a pair of concealed tubes which was the longer and which was the shorter, even though one was an 80 cm. gastroscope. Therefore a short tube is no advantage so far as illumination is concerned. In regard to the manipulation of forceps, &c., an additional length of 10 or 15 cm. is of no disadvantage whatever. It is true that a somewhat larger tube can be used through a tracheotomic wound than through the glottis, with safety to the subglottic structure, but Dr. Patterson and I have found that a tube of 4 mm. internal diameter is amply large for delicate manipulations under the guidance of the eye, such as the placing of a hook through the eye of a shoe-button in the bronchus of a child six months of age. If one is not accustomed to work through small tubes, doubtless it is better to do a tracheotomic bronchoscopy than to force a large tube through the larynx. In upper lobe bronchoscopy almost as favourable an angle can be obtained by shifting the tube to the opposite corner of the mouth, as could be obtained by a tracheotomic bronchoscopy, provided the assistant holding the head and the operator have worked years together, so that they co-operate, and the head of the patient is carried along with the tube to the extreme opposite position from the lobe to be explored. All of these things are readily demonstrated on the patient, but, unfortunately, the statements in the early literature have led men into hasty tracheotomy rather than to develop the necessary technic to work with very small tubes and thus to avoid damage to the

subglottic area. Out of 706 bronchoscopies for all purposes, no one in the author's clinic has ever done a tracheotomy for the purpose of bronchoscopy. The only lower bronchoscopy ever done by me for a foreign body was in the early days of bronchoscopy. The general surgeon had already done a tracheotomy for a compressive stenosis due to a goitre. In that case I failed to find the foreign body, a small pin. With that exception, it has always been our custom to insert the bronchoscope through the

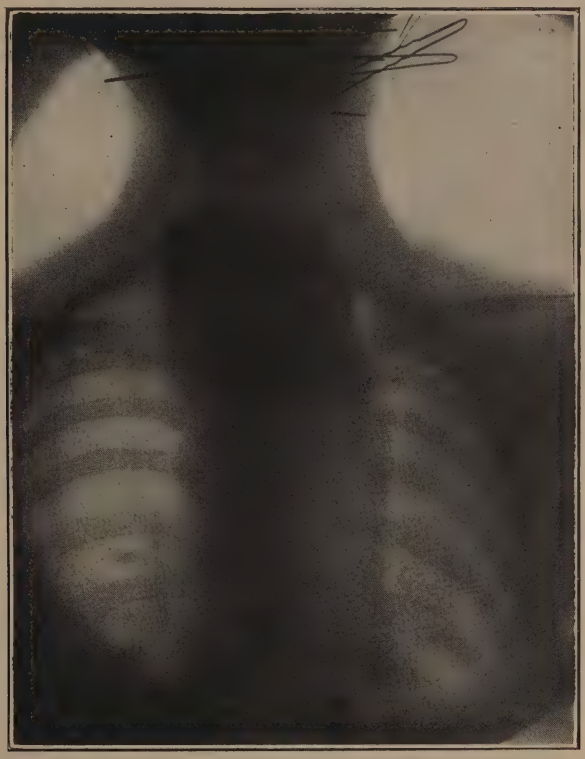


FIG. 12. Radiograph of woman of 22 years simulating diverticulum. The bismuth porridge rests upon about 1½ litres of stale food in an enormous dilatation as demonstrated œsophagoscopically after emptying. A case of abdominal œsophagismus.

mouth even in the cases which required tracheotomy for dyspnœa. Very often patients come in with such severe dyspnœa that it is unwise to leave them over-night without a tracheotomy. In such cases the absolute rule in tracheal surgery, to do a tracheotomy always early, never late, is followed ; but in the first management of the case we have always found that a bronchoscope introduced through the mouth is much better for the temporary relief of dyspnœa, with artificial respiration by insufflation of oxygen, &c., and in foreign body extraction the bronchoscope introduced through the mouth is much more freely manipulated and much more

satisfactory with which to work, because the patient's head is very much less in the way, and all of the movements and manipulations are the habitual ones in peroral endoscopy, while tracheotomic bronchoscopy requires new and different manipulations.

Pleuroscopy. The author has, in one instance, removed a foreign body, a primer from a shotgun cartridge, from the pleural cavity through a small opening made in the chest by Dr. J. Hartley Anderson. This was done immediately after the accident, and there was no odour or pus at any time. General anæsthesia was given, and the child, after the chest opening was made, was placed in the sitting position in order that the foreign body would fall to the diaphragm. Healing was prompt and the air began to enter the lung on the fifth day. The child made a prompt recovery, and now, about a year after the operation, is in perfect health. In this class of cases, pleuroscopy promises excellent results if done immediately, before infection or inflammation has set in. Only a small opening is necessary, and this does not involve anything like the shock consequent upon the large osteoplastic flap. The only shock is the pleural shock, which is slight, and which in my case was present anyway, because there was already a pneumothorax before the chest was opened. In a future case I shall aspirate the air from the pleural cavity after closure of the wound.

Contra-indications to œsophagoscopy. Water-hunger is the most serious of all contra-indications to œsophagoscopy. Patients have frequently been sent to the author who have been able to get but little liquid down for a number of days, and it is the author's custom always to have a surgeon in readiness on arrival of the patient to have a gastrostomy done immediately, should the patient prove to be in a serious state of water-hunger. In less serious states fluid is introduced into the circulation by hypodermoclysis and enteroclysis simultaneously. The author agrees with Brown Kelly that it is unwise to wait for a diagnosis before advising gastrostomy. In foreign body cases there is no absolute contra-indication. If there is anything to be gained by it a careful œsophagoscopy may be undertaken by the trained hand and eye, which will stop the procedure when an abnormal tissue which must not be passed or even touched is encountered.

Œsophagoscopy for foreign bodies. With the relatively high mortality from external œsophagotomy, it certainly seems as though the operation is rarely if ever justifiable in foreign body cases. The author's personal experience is that with the full relaxation of general anæsthesia, preferably with ether (perhaps adding a little chloroform, if necessary, after the patient is under the stimulant effect of the ether), any foreign body that has gone down the œsophagus can be brought up the same way, if the œsophageal musculature were prevented from clamping reflexly the foreign body. But in many instances, in fact, perhaps in nearly all, mechanical ingenuity will devise a way to get the foreign body out either by turning or morcellation, as in the historic case of Killian where a



FIG. 13. Lateral radiograph (by Dr. Lange) of same patient as in Fig. 9, the shadow of the bismuth mixture simulating diverticulum. This mass protruded in the neck and could be evacuated by external pressure with the patient's hand.



FIG. 14. Radiograph (by Dr. Russell H. Boggs) of a woman of 38 years. The shadow, which so much resembles a diverticulum, was œsophagoscopically proven to be a dilatation above a luetic stricture. Endoscopic dilatation resulted in a cure.

vulcanite tooth-plate was divided. Ingenuity will succeed where he who relies upon main strength and powerful forceps will meet disaster.

'Cardiospasm', phrenospasm, and abdominal œsophagismus. Undoubtedly the old word 'cardiospasm', like many of the old words of medicine, covers a number of different conditions of independent ætiology and pathology, and it behoves us as endoscopists carefully to study the different endoscopic pictures throughout the entire œsophagus, and as much of the stomach as possible, that we may bring order out of chaos. Much progress has recently been made. The diagnosis of cardiospasm is easy in the typical case with an enormous dilatation, a white, pasty, macerated mucosa, and a contracted abdominal œsophagus which, however, permits a large gastroscope to pass into the stomach after a delay; but in the early, or in the less typical cases without dilatation, it is often exceedingly difficult to distinguish between purely spasmodic conditions and those of local lesions in the neighbourhood of the œsophagus, but not themselves showing in compressions or very marked deviations of the abdominal œsophagus. In such cases, while many œsophagoscopists feel sure of their diagnosis, many do not agree as to what the endoscopic pictures are, and many endoscopists describe a picture which is seen by other endoscopists at the hiatus or in the perfectly normal œsophagus abdominalis. I believe it would be very profitable to all of us if every endoscopist present would describe the different endoscopic pictures he sees in phrenospasm or hiatal and abdominal œsophagismus. No one to-day believes in a sphincter at the cardia, and the narrowing at this point that has been shown in so many text-books on anatomy is a misfortune. Hill quotes McAllister to the effect that there is no histologically demonstrable sphincter, and he states that the circular musculature at this point is weak. Brown Kelly and Williamina Able, by careful special dissections, have demonstrated that 'it is quite apparent to the naked eye that both muscular coats are of uniform thickness, and that no special aggregation of fibres exists at or near the cardia', and that 'nothing was found in any of the dissections or in the anatomical works consulted to justify Dr. Hill's statement that the circular fibre musculature was specially weak in this region'. As endoscopists, however, I think that it would be better for us to abandon the word 'cardiospasm' and to substitute for it the three clinical types, which, according to my observation, can be made out as existing either separately or together. One of these is spasm at the hiatus œsophagus, which from its location, and from the fact that it is the diaphragmatic, not the œsophageal musculature that is active, should be called phrenospasm or hiatal œsophagismus, while that occurring below should be called abdominal œsophagismus, because it is very clearly proven by the endoscopic picture, and by the radiographs, two of which are here shown, that the entire abdominal œsophagus is involved in the spasm, and it is not simply a spasm of the cardia. The word cardia is properly used as the name of the œsophageal orifice of the stomach. Spasm limited solely to this orifice is certainly exceedingly

rare, while spasm of the abdominal œsophagus and of the œsophagus at the hiatus either separately or together are relatively common, and should be called by their proper names, and the word cardiospasm should either be dropped as a misnomer or limited to those rare cases of true

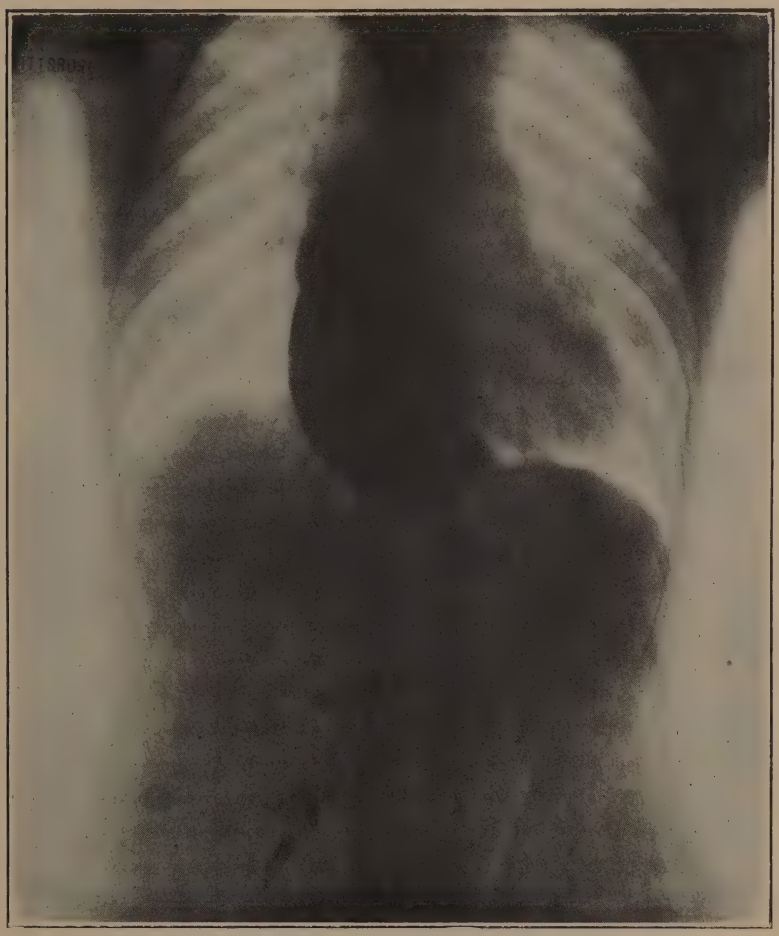


Fig. 15. Radiograph (by Dr. George C. Johnston) of a woman of 45 years showing an abdominal œsophagismus which was afterwards cured by endoscopic mechanical divulsion. The 'flat floor' of the dilatation shows why previously used blind methods had failed to introduce any instrument through the hiatus.

cardial œsophagismus. Brown Kelly has demonstrated the experimental fact that section of the vagi without stimulation is followed by dilatation of the lower part of the œsophagus and contraction of the cardia, which he rightly says corresponds to the supposed condition in cardiospasm. But as a clinical fact I have rarely found such a condition in the disease commonly known as cardiospasm. It is quite certain that in a large

majority of the cases there is a certain basic general nerve disorder. In one instance a patient who was quite hysterical would get an attack of abdominal œsophagismus whenever everything did not please her. For instance, she took the notion that the bi-weekly endoscopic divulsions that I was applying to the abdominal œsophagus were doing her so much good that they ought to be done every week. Regularly the day before the one week was up her abdominal œsophagus would shut up and the dilatation above it would fill. By this I do not mean that she had voluntary control of it, but that the emotions sought their outlet through habitual nerve-channels, producing a recurrence of the abdominal œsophagismus, which she had had since childhood. This ' nerve-cell habit ' is one of the most frequent causes of recurrences. In certain cases there are undoubtedly lesions of the mucosa in the œsophagus and also in the stomach which could easily excite spasms, and it is equally certain that stagnation due to the spasm, and consequent fermentation of food, detention of secretions and maceration, could very easily excite or perpetuate the lesions. Thus we have a ' vicious circle ' in hiatal and abdominal œsophagismus. Observations by Guisez, MacKinnie, and also some observations of my own, point clearly to the fact that these lesions can produce organic stricture. The frequency with which spasm of the abdominal œsophagus occurs in connexion with gastric carcinoma indicates very plainly that the reflex can have its source in the stomach, which seems to confirm the theory that hyperacidity may be a cause in some instances. The theory that diffuse dilatation is the cause rather than the effect of spasmodic closure of the abdominal œsophagus has been negatived by Jesse Meyer, who has proved that after the cure of a ' cardiospasm ' and the complete relief of all the symptoms, the dilatation of the œsophagus remains. A number of observers have confirmed the observations of Kovacs and Stoerk in regard to the kinking of the œsophagus by enlargement of the left auricle. I have myself since observed in two cases very marked deviations from this cause. In both cases there had been no symptoms referable to the œsophagus, but a large mass of meat had lodged and was removed œsophagoscopically. In a number of other cases an enlarged aorta has deviated the œsophagus as in the radiograph here shown, and Boyce has demonstrated that there is a type of ' neurasthenic ' patient with an enlarged but not definitely pathologic aorta that is particularly prone to phrenospasm and abdominal œsophagismus. In these cases Boyce has demonstrated an unusually palpable and enlarged abdominal aorta. Cases of phrenospasm and abdominal œsophagismus, even more than diverticulum, remind one of the ingluvies of birds, inasmuch as the dilated œsophagus fills quickly, and yet there is a constant leakage, which allows a certain proportion of the food to pass on through at a relatively slow rate. In one of my patients the cure of the abdominal œsophagismus resulted in food going through so promptly into the stomach that taking food excited nausea for quite a long time, until the stomach became accustomed to the unusual sensation of having food

go through directly when swallowed. Treatment of abdominal œsophagismus and hiatal œsophagismus has lead to the devising of a number of different dilating water-bags and air-bags, which have yielded good results. In some cases, however, it is impossible to introduce them.

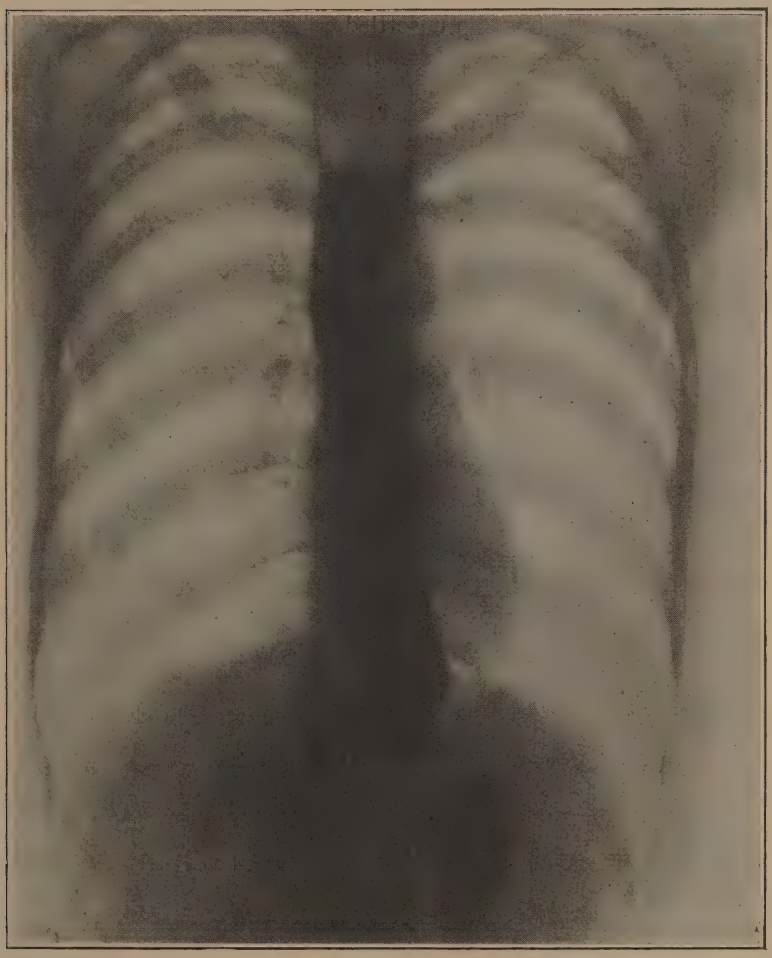


Fig. 16. Radiograph (by Dr. J. C. Bowen) of a woman of 28 years, showing an abdominal œsophagismus with only very slight dilatation above it. The deviation of the œsophagus by the aorta was verified œsophagoscopically.

The author's personal preference, like that of Brünnings, is for a mechanical divulsor inserted through the œsophagoscope where the sense of touch and the precision of a steel instrument give one an accurate control without ' hour-glass ' faults. I use the divulsor of Mosher. Undoubtedly there are a few cases that do not get well after dilatation of the abdominal œsophagus, and the most stubborn are those existing since

childhood, with consequent infantile stomach and long-established 'nerve-cell habit'. The neurologist has cured some cases after both I and the internist had failed. In addition to the three forms that I have mentioned, cardial œsophagismus, phrenospasm, and abdominal œsophagismus, there are, of course, the spastic neuroses of the œsophagus higher up, but it is still, I believe, an unsettled question as to what extent these so-called neuroses have an anatomic ætiologic basis. Progress has been made, but to analyse it would unduly lengthen this paper.

Cancer of the œsophagus. The diagnosis of malignancy here, as elsewhere in the body, is usually readily made by gross appearances in advanced cases, but, as elsewhere, the very much to be desired early diagnosis depends upon the histologic examination of a specimen. The specimen should be ample, and should, if possible, include a little of the adjacent normal, though greater care is needed here than elsewhere as to the amount of normal that may be taken. In no case should a portion of the normal muscular layer be excised. A little of the mucosa is sufficient. The only contra-indication to the taking of a specimen is such a profoundly anæmic condition that the oozing which may follow may turn the balance against the patient. This anæmic condition is usually only found in those cases that have been permitted to become moribund from hunger and thirst from too long delayed gastrostomy. In such cases the gastrostomy should be done at once and the patient fed until the specimen may be taken with safety. Unfortunately such patients, especially if there has been a few days of water-hunger, make exceedingly bad surgical subjects, so that the minor operation of gastrostomy assumes a high mortality. It is usual among endoscopists in the diagnosis of cancer to attach a great deal of weight to asymmetrical respiratory movement of the walls of the œsophagus. In doing this it is very necessary that the patient's head be exactly in the median line and the body straight. The twisting of the head to one side is very apt to alter the symmetry of the visible normal œsophageal lumen. This is demonstrated graphically in the radiograph here shown. The twisting of the child's head sidewise causes the image of the coin to be elongated, owing to the fact that the coin is moved diagonally instead of antero-posteriorly by the normal respiratory movements, owing to the child's head being strongly rotated to one side. Cancer of the œsophagus has at the present day 100 per cent. mortality, but there is good reason to believe that the surgeon will show a certain percentage of cures so soon as physicians will promptly refer to the œsophagoscopist all patients showing the slightest abnormality referable to the œsophagus. Thus only can we hope to discover and to treat the precancerous conditions of leucoplakia, erosion, maceration, chronic œsophagitis, &c. That the physician may do this without hesitation it behoves the œsophagoscopist so to perfect his technic and develop his skill that a patient may be œsophagoscoped without distress and without anæsthesia, or at most with local anæsthesia limited to the pharynx. The work of Henry Janeway has convinced me that resection of the thoracic

œsophagus is a practicable procedure and will be frequently resorted to as soon as early œsophagoscopies shall make the necessary early diagnosis. In the palliative treatment of inoperable œsophageal cancerous stenosis, gastrostomy may be postponed by œsophageal intubation in many instances until very nearly the termination of the case, though by this I do not mean that gastrostomy should be postponed one day after it is clear that nutrition is going to suffer. It is, of course, much more satis-

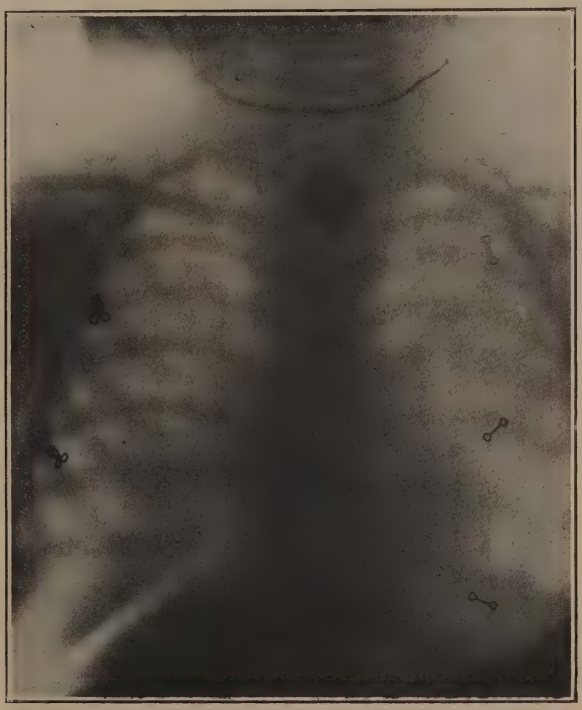


FIG. 17. Radiograph (by Dr. George J. Boyd) of a coin in the œsophagus, showing the diagonal respiratory œsophageal movement, illustrating the necessity of the exact median position of the head if any diagnostic importance is to be attached to asymmetrical respiratory œsophageal movement in œsophagoscopy for suspected peri-œsophageal lesions. This illustrates one of the disadvantages of the lateral position for œsophagoscopy.

factory to the patient to swallow his food, even though it be liquid, than to have it poured in through the abdominal wall. Œsophageal intubation has been very satisfactory in the author's hands. He is not prepared to say that endoscopic methods are in any way preferable to those of Charters Symonds for the introduction of a tube, but in all cases endoscopic study should precede intubation. All forms of clear liquids will go through œsophageal intubation tubes of 3 mm. internal diameter, and raw or very slightly cooked eggs can, with care, be swallowed with much satisfaction by the patient whose œsophagus is intubated. In fact,

finely masticated food of any kind will go through, though occasionally imperfectly masticated particles may lodge in the smallest tubes. The author has had these tubes worn for quite a number of months without exciting ulceration, though of course cancerous ulceration was already present in some instances. Of course the tubes were removed at frequent intervals for cleaning and were replaced. It is essential to have a duplicate tube for immediate replacement, else the œsophageal channel will quickly close so that a smaller tube will be needed. Eventually a smaller and still smaller tube is needed anyway, until none can be introduced.

Gastroscopy. Recent developments have been noteworthy. In different parts of the world earnest workers have been perfecting technic and instruments. It has been said that the time wasted in an effort to develop a substitute for the straight tube would have been better spent in perfecting the operator's personal skill in introducing the straight tube. While this may be true, it is unwise to discourage effort. It is now universally accepted that safety demands that introduction shall be with a tube devoid of a lens system, in order that lesions may be detected and avoided, and that the axis of the instrument may be readily kept in line with the œsophageal axis. After the distal end of the tube has reached the stomach, a plug with a window has been used in the proximal end of the tube, so that positive pressure from an oxygen tank (Janeway) or a hand bulb (Mikulicz, Hill, Mosher, and others) may be used to push away mucosal folds. Then a lens system is inserted in the open tube in the form of a long tube with a window in the side of the distal end, an excellent view of the distended stomach being obtained. The author, personally, can testify to the beautiful view of the pylorus obtained in the Janeway gastroscope, and while the use of inflation and of a lens system for gastroscopy is at least thirty years old, yet the optical formula and the particular combination of illumination, lenses, inflation apparatus, and tubes in the Janeway gastroscope make it one of the greatest of all recent advances in gastroscopy of which the author has personal knowledge. Excellent results have also been obtained by Hill, Elsner, and others, and all of these instruments are now doubtless developed to a point where the personal skill of the operator counts for more than the particular instrument. The usefulness, safety, and practicability of the gastroscope are an accomplished fact. The need now is for careful skilful men who will use it. The value of gastroscopy in establishing a diagnosis in severe and obscure stomach diseases has been abundantly proven. The tendency, however, to resort to it only in very serious conditions prevents its greatest usefulness, which would be in the early diagnosis of cancer and of precancerous conditions. When endoscopists, who have developed the gastroscopic technic are sufficiently numerous and sufficiently skilful that the physician or the surgeon may feel justified in sending them cases before the patient's condition becomes desperate, gastroscopy will be of great use to the physician and surgeon, but gastroscopy probably will never be done by the physician and surgeon themselves. They will take

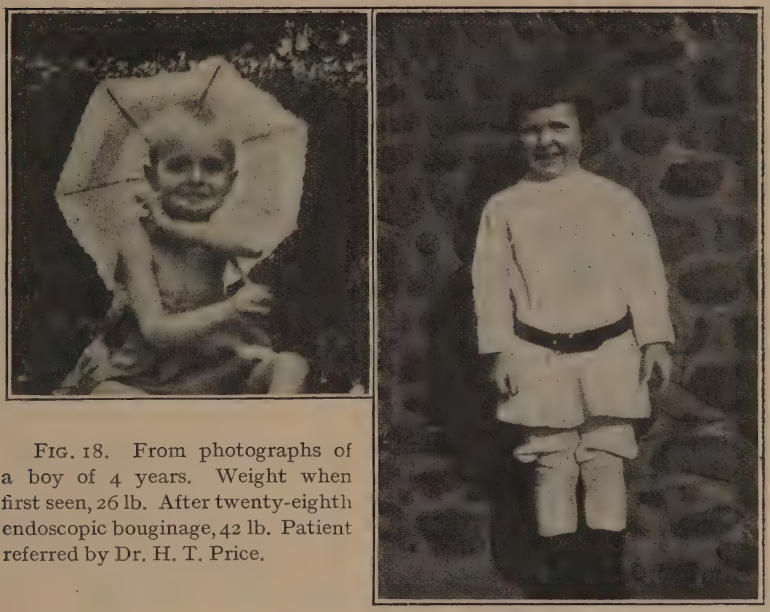


FIG. 18. From photographs of a boy of 4 years. Weight when first seen, 26 lb. After twenty-eighth endoscopic bouginage, 42 lb. Patient referred by Dr. H. T. Price.

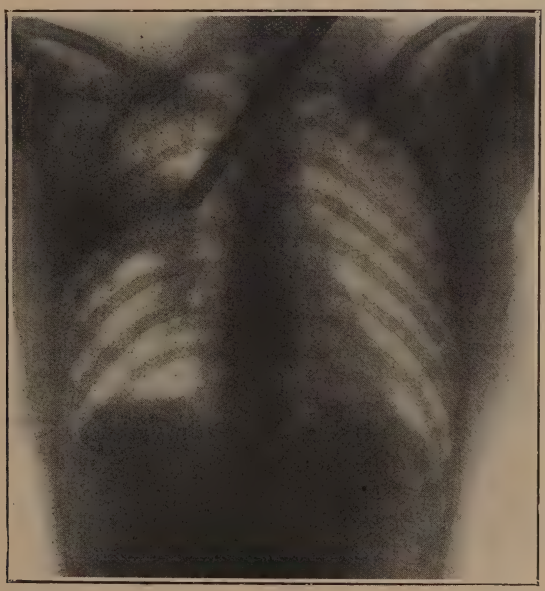


FIG. 19. Radiograph (by Dr. George C. Johnston) of bronchoscope in the right upper lobe bronchus of a woman of 25 years (patient of Dr. Otto C. Gaub). The bronchoscope was inserted through the mouth, and the angle is shown to be as advantageous as would be possible through a tracheotomic wound. The position of the patient is easy and natural in this instance, the radiograph being made for verification of the overlay localization in a suspected case of interlobar abscess. Had demonstration been the object, the upper part of the tube could easily have been brought to the clavicle.

the endoscopist's report along with that of the radiographer and analyst and decide as to the best handling of the case, just as the otologist in a brain case takes the report from the internist, the laboratory, and the ophthalmologist. Unlike tracheo-bronchoscopy and œsophagoscopy, it may be said of gastroscopy that while its positive findings are extremely valuable, its negative findings are less so, just as with ophthalmoscopy in brain disease, the Wassermann reaction, and many others of our most valuable aids in medicine and surgery. A very important point in increasing the range of mobility of the distal end of the gastroscope in the stomach has been demonstrated by Henry Janeway. It consists in an elevation of the knees of the recumbent patient to the vertical or flexed position, with consequent relaxation of the abdominal wall.

Conclusion. In conclusion, I wish to say that recent progress in endoscopy of the larynx, trachea, bronchi, œsophagus, and stomach has shown very clearly that all future progress will depend on those who have the time and opportunity to develop an organization with assistants and nurses well trained, and to develop, by continual practice, the skill which the musician must have with his instrument ; and while it is given to but a few like Killian, von Eicken, and Brünnings, to be the Paderewskis of endoscopy, yet any one who will practise continually and keep himself in continual training, may do good work and contribute to the future progress of peroral endoscopy.

SECTION XV

RHINOLOGY AND LARYNGOLOGY

DISCUSSION No. 2

(Jointly with Section XVI, Otology, Discussion No. 3)

MÉTHODES ET RÉSULTATS DU TRAITEMENT DES MALADIES DE LA GORGE, DU NEZ ET DE L'OREILLE PAR LE SALVARSAN ET LES AUTRES COMPOSÉS ARSÉNICAUX

Rapport par M. le Dr. ANDRÉ CASTEX, Chargé de Cours à la Faculté de Médecine de Paris

Première Partie : Méthodes et Techniques

A l'apparition des nouveaux moyens de traitement pour la syphilis, les spécialistes en oto-rhino-laryngologie, qui rencontrent souvent cette infection dans le champ de leur pratique, ne pouvaient manquer d'y recourir aussitôt.

S'ils s'en étaient désintéressés, des accidents graves survenus aux oreilles par l'emploi des médicaments nouveaux les auraient obligés d'y donner toute leur attention.

C'est à ce double point de vue, ce me semble, que ce rapport a été proposé.

Les principaux agents dont nous aurons à examiner les effets dans les affections du pharynx et du larynx, du nez et de l'oreille, sont :

Le cacodylate iodohydrargyrique, l'énésol, l'atoxyl, l'hectine, l'arsénophénylglycine, le Salvarsan, le néo-salvarsan.

Le cacodylate iodohydrargyrique est un mélange de cacodylate de soude (dont la formule chimique est : $As . O(CH_3)_2ONa$) et de biodure de mercure (dont la formule chimique est : HgI_2).

L'énésol est du salycilarsinate de mercure. C'est une combinaison de l'acide méthylarsinique (dont la formule chimique est : $CH_3-AsO_3H_2$) et de salicylate basique de mercure (dont la formule chimique est : $C_6H_4\left\{\begin{matrix}CO_2\\O\end{matrix}\right\}Hg$).

L'atoxyl : Anilarsinate de soude, ou aminophénylarsinate de soude : Formule :

$$C_6H_4\left\{\begin{matrix}AzH_2\\AsO\left\{\begin{matrix}OH+4H_2O\\ONa.\end{matrix}\right.\end{matrix}\right.$$

L'hectine : Benzosulfure paraaminophénylarsinate de soude :
Formule :

$$C_6H_4\begin{cases}AzH-SO_2-C_6H_5\\AsO\begin{cases}OH\\ONa.\end{cases}\end{cases}$$

L'arsénophénylglycine (préparation No. 418 d'Ehrlich-Hata).
Formule :

$$CO_2Na-CH_2-AzH_1-C_6H_4-As=As-C_6H_4-AzH-CH_2-CO_2-Na.$$

Le Salvarsan (préparation 606 d'Ehrlich-Hata).
Bichlorhydrate de dioxydiamido-arsénobenzol.
Formule :

$$\begin{matrix}OH\\AzH_2\end{matrix}\Big\}C_6H_3-As=As-C_6H_3\Big\{\begin{matrix}OH\\AzH_2.\end{matrix}$$

Le néo-salvarsan (préparation 914 d'Ehrlich-Hata).
Ce corps a la constitution du Salvarsan, plus du formaldéhyde-sulfoxylate de soude :
Formule :

Celle du Salvarsan $+ CH_2O$. SO Na.

Nous avons classé le plus méthodiquement possible les divers documents que nous avons recueillis dans notre pratique personnelle ou dans nos lectures.

Quand les observations étaient incomplètes, j'y ai pris du moins le document utile. Là, comme souvent ailleurs, la difficulté est de suivre les malades.

J'ai examiné parallèlement les avantages ou les inconvénients de ces divers agents.

Je dois beaucoup, pour la rédaction de ce rapport, à l'obligeance de mon ami, le Professeur Gaucher, qui a mis à ma disposition les malades de son important service des maladies syphilitiques de l'Hôpital Saint-Louis. Je l'en remercie vivement.

J'adresse aussi mes meilleurs remerciements à mon ami et collègue, le Docteur Maurice Bloch, qui m'a aidé dans mes recherches, à M. Rameau, externe des hôpitaux, qui a bien voulu rédiger les observations, et à mon collègue, le Docteur Druelle, à qui je dois les détails de chimie organique.

1° *Hectine*

Nous avons spécialement étudié, mon élève, le Docteur Berruyer, et moi, les résultats de l'hectine en oto-rhino-laryngologie.[1]

L'hectine ou benzosulfure paraaminophénylarsinate de soude, découverte par le Docteur Mouneyrat en 1908, est entrée dans la thérapeutique antisyphilitique, sous les auspices du Docteur Balzer, depuis 1909.

[1] Voir *Bulletin d'Otolo-rhino-laryngologie*, 1911, p. 288.

L'expérimentation et la clinique ont démontré que ce médicament est parfaitement toléré et sa toxicité pour ainsi dire nulle. Si des phénomènes d'intolérance ont été relatés, ils sont dus, soit à l'absorption de doses trop fortes, soit à une sensibilité spéciale du sujet.

L'hectine peut s'administrer par la voie buccale. La solution à employer dans ce cas doit être dosée à 5 centigrammes d'hectine par cuillerée à café. On peut prescrire par jour : 10, 15, 20 centigrammes du médicament. L'hectine se donne aussi sous forme de gouttes (20 gouttes renferment 5 centigrammes d'hectine). On donne 40 gouttes par jour les deux premiers jours — les jours suivants, on augmente de 40 à 100 gouttes par jour pendant 10 jours. Enfin, l'hectine peut être prise sous forme de pilules, une ou deux pilules renfermant 10 centigrammes d'hectine, pendant 10 à 12 jours.

La voie hypodermique présente de sérieux avantages sur la voie buccale. Le Docteur Balzer a établi la posologie de l'hectine en injections hypodermiques de la façon suivante :

' Injecter dans les muscles fessiers, d'abord tous les deux jours, pendant la première semaine, à titre d'essai, une dose de 10 centigrammes d'hectine dissoute dans un centimètre cube d'eau distillée.

' Injecter ensuite la même dose tous les jours. Comme la dose totale de la cure est de deux grammes, quelquefois trois, on peut accorder au malade un repos de deux ou trois jours au milieu du traitement.'

On peut employer des doses plus élevées — administrer même des doses massives hebdomadaires de 0 gr. 60 d'hectine. Nous avons traité par la méthode des injections de 0 gr. 10, répétées trois fois par semaine, un certain nombre de malades atteints d'affections syphilitiques (chancre de l'amygdale — gommes ulcérées du nez, du pharynx, etc.).

Nos observations portent sur une douzaine de cas — qui peuvent se décomposer de la façon suivante :

Chancre primitif de l'amygdale	1 cas
Manifestations secondaires de la bouche et du pharynx	1 cas
Manifestations tertiaires (gommes du nez — gommes ulcérées du pharynx)	7 cas
Manifestations héréditaires	2 cas

Chez tous ces malades, la tolérance au médicament a été parfaite. Nous n'avons observé ni troubles auditifs tels que ceux signalés par Duhot, ni troubles oculaires. L'état général des malades est resté excellent pendant toute la durée de la cure — même chez le malade atteint de chancre de l'amygdale, qui pourtant était porteur d'une syphilis maligne.

Dans la plupart des cas, les injections faites dans la masse musculaire ont été complètement indolores. Nous n'avons noté ni réaction inflammatoire, ni empâtement profond, ni rougeur. Deux malades seulement accusaient, dans les heures qui suivaient l'injection, une cuisson douloureuse, irradiant à la cuisse.

Quant au résultat thérapeutique, il ne nous a pas donné toutes les

satisfactions que nous attendions. L'impression d'ensemble qui se dégage des cas observés est la suivante : sous l'influence des premières injections, une amélioration brusque, rapide, a toujours été constatée. C'est ainsi que le malade atteint de chancre a vu sa plaie presque cicatrisée avec deux grammes d'hectine. La roséole a été tellement discrète qu'il a fallu la rechercher. Elle n'a pas duré plus de 24 heures. Il n'est apparu aucune plaque muqueuse. Deux autres malades qui avaient au pharynx une vaste ulcération occupant presque toute la largeur de la paroi, déterminant des troubles douloureux intenses de déglutition ont semblé cicatriser leur vaste plaie anfractueuse avec 8 à 10 injections d'hectine.

Mais cette amélioration, si rapide au début, s'est considérablement ralentie par la suite, au point de nous obliger, pour terminer la cure, à recourir à la médication hydrargyrique. Bien plus, le malade atteint de chancre amygdalien, malgré l'absorption de trois grammes d'hectine, a vu apparaître des plaques muqueuses buccales nombreuses, étendues et rebelles. Ses mains ont été couvertes de syphilides papuleuses. La cicatrice du chancre s'est rouverte. Tous ces accidents n'ont cessé qu'après l'absorption de doses considérables de mercure. De même chez un malade atteint de gomme ulcérée du pharynx, chez lequel l'amélioration semblait assez rapide, les accidents ont subi une recrudescence brusque et intense. L'ulcération, malgré deux grammes d'hectine, a envahi le voile du palais, les piliers, etc. Enfin, dans un cas de syphilis héréditaire du nez (fillette de 16 ans) avec tuméfaction et rougeur du côté droit du nez, empâtement douloureux, le traitement par l'hectine a été absolument inefficace : les accidents ont brusquement disparu par l'absorption du sirop mercuriel.

2° *Salvarsan*

Sur la valeur du Salvarsan, les opinions diffèrent. Pour les uns, tels Leredde, il est héroïque, même contre la parasyphilis. D'autres, Milian, Neisser, Hoffmann, Rosenthal, conseillent d'employer d'abord le 606 et ultérieurement le mercure et l'iodure. D'autres enfin ne croient qu'à ces deux derniers médicaments et n'ont recours au 606 qu'exceptionnellement.

Nous avons pour nous édifier deux intéressantes statistiques de Benario et de Valentin.

Benario sur 14,000 sujets traités par le 606, trouve 126 cas de neurorécidive sur lesquels 62 fois le nerf acoustique était atteint. Sur ces 62 cas :

Lésions du nerf cochléaire seul	29 cas
Lésions du nerf vestibulaire seul	5 cas
Les deux branches de l'acoustique intéressées	17 cas
Lésions combinées de l'acoustique et des autres nerfs crâniens	11 cas

(*Münchener med. Woch.*, 1911, No. 14.)

Valentin (de Berne) mentionne 45 cas de lésions de l'auditif après injections de Salvarsan :

Lésions de la branché cochléaire	9 cas
Lésions de la branche vestibulaire	7 cas
Lésions des deux branches	20 cas
Lésions associées de l'auditif et des nerfs crâniens	9 cas

(*Internat. Zentralbl. für Ohrenh.*, Band ix, Hefte 11a, 12.)

3° *Néo-salvarsan*

Tout récemment, le Professeur Ehrlich, cherchant un dérivé du Salvarsan qui fut absolument neutre au point de vue chimique et physiologique, a traité le Salvarsan par le formaldéhydesulfoxylate de soude et obtenu le néo-salvarsan (ou 914), qui répond à la formule :

$$\begin{matrix} AzH_2 \\ OH \end{matrix}\left\{C_6H_3AS = AsC_6H_3\right\}\begin{matrix} AZH\,.\,CH_2\,.\,O\,.\,SONa \\ OH. \end{matrix}$$

La teneur en arsenic est d'un tiers inférieure à celle du 606.

Il a cet avantage d'être directement soluble dans l'eau distillée et de donner une solution neutre.

Il n'exerce pas d'action coagulante sur l'albumine et reste dans le sang comme un corps physiologiquement indifférent, et il a sur le Salvarsan cet avantage de n'être pas précipité par les sels acides du sang, de s'y maintenir à l'état de sel soluble, sans addition de soude et par conséquent sans les risques imputés à l'hypoalcalinisation et d'arriver ainsi dans les organes les moins vascularisés et même par infiltration dans la lymphe et les espaces lymphatiques, ce que ne pouvait faire le Salvarsan qui reste dans le sang à l'état de précipité.

MM. Lévy-Bing et Durœux (de Paris) ont fait une centaine d'injections intra-veineuses et utilisé des doses 45, 60, 75 et 95 centigrammes de néo-salvarsan, laissant s'écouler entre deux injections consécutives un intervalle d'au moins six jours.

Chaque dose dissoute dans 100 à 150 centimètres cubes d'eau distillée et stérilisée froide est injectée lentement, en cinq minutes environ.

Ils n'ont noté aucune réaction ni pendant ni immédiatement après l'injection. Pas de congestion faciale, pas de sensation de malaise, pas de douleur épigastrique comme on l'observe souvent avec le Salvarsan. La tension artérielle est peu modifiée et le néo-salvarsan est très bien supporté par les syphilitiques nerveux (Sicard). Cependant les malades ressentent presque toujours un engourdissement dans le bras et des fourmillements dans l'avant-bras et la main. Aucune induration de la veine. Pour éviter l'hémolyse, Queyrat emploie comme véhicule du sérum à 4 pour 1,000.

Balzer qui emploie les injections intra-musculaires a constaté que, vu sa grande solubilité, ce sel se prête beaucoup mieux que le 606 à ce mode d'injections.

Les réactions tardives ne diffèrent guère de celles du Salvarsan : céphalées, courbatures, nausées, vomissements, quelquefois diarrhées, mais ces accidents sont moins intenses et durent moins qu'avec le Salvarsan. Chez deux malades, il y a eu de l'albuminurie et de l'érythème généralisé. D'autres auteurs (Hudelo, Montlaur et Bodineau) ont signalé des exanthèmes ortiés, bulbeux ou pemphigoïdes. Chez un autre malade, Lévy-Bing a observé de l'anurie passagère.

Malgré toutes les précautions la température monte le plus souvent à 38°, voire même exceptionnellement à 40°.

L'intensité de ces réactions n'est d'ailleurs pas en raison directe des doses.

Sa faible toxicité estimée par Kersten à 0 gr. 20 par kilogr. de lapin, explique les doses qui ont pu être employées.

Les injections peuvent se faire à 4 ou 5 jours d'intervalle, au lieu de 8 jours, parce que l'élimination du 914 est plus rapide que celle du 606 (Leredde).

L'effet thérapeutique de ce nouveau sel arsénical est analogue à celui du Salvarsan, mais peut-être se produit-il plus lentement. Il agit particulièrement bien sur les lésions des muqueuses qui disparaissent très vite. L'examen ultra-microscopique a montré la disparition complète des tréponèmes 24 heures après la première injection (Queyrat).

Il est à noter que le processus de cicatrisation est surtout rapide au pharynx où les infiltrations et les ulcérations même douloureuses disparaissent en 3 ou 4 jours.

Au total, le néo-salvarsan est d'un emploi plus facile que le Salvarsan et il est mieux toléré (Lévy-Bing et Durœux).

Entre autres contre-indications il y a les maladies du foie et des reins. On doit toujours interroger d'abord les organes par l'examen clinique et les procédés de laboratoire avant de recourir à la Salvarsanothérapie. Le médicament est un vaso-dilatateur puissant, ce qui peut expliquer certains troubles qu'il entraîne.

Dans certaines circonstances, la nouvelle méthode est indiquée chez le nouveau-né. Le Professeur Chambrelent (de Bordeaux) dans un rapport à la Société obstétricale de France (Paris, 10–12 octobre 1912) a indiqué la meilleure technique à employer chez eux.

Elle sera indiquée quand le nourrisson présentera des manifestations muqueuses graves qu'il sera urgent de faire disparaître ou quand le mercure n'aura pas donné de résultats satisfaisants.

La voie intra-veineuse est très employée en Allemagne, mais le Professeur Chambrelent estime moins dangereuse la voie sous-cutanée ou intra-musculaire. Quant aux doses, il faut s'en tenir aux doses maximum de 15 milligrammes par kilogramme du poids de l'enfant, sauf à renouveler l'injection après avoir noté la tolérance pour le médicament.

MM. Fabre et Bourret (de Lyon) ont traité 20 nourrissons par des injections intra-musculaires de Salvarsan. L'absorption se fait lentement et les piqûres peuvent produire des sortes de cavités kystiques qu'ils

ont vu persister pendant quatre mois. Avec le néo-salvarsan, l'induration locale est moindre.

Sur les lésions locales des hérédo-syphilitiques, le Salvarsan a une action très nette et très rapide, mais comme les récidives ne sont pas rares, il faut parfois en venir au mercure et à l'iodure.

A la clinique Tarnier, de la Faculté de Paris, le Salvarsan a été employé chez treize nourrissons. Seul le traitement direct a influencé rapidement la réaction de Wassermann vers la négative. Il a paru très dangereux chez les nouveau-nés ayant des accidents confluents. Sur l'emploi du Salvarsan, le Professeur Bar concluait (Société obstétricale de France, 10–12 octobre 1912) : 'c'est le moyen le plus puissant pour guérir rapidement les plaques muqueuses, mais c'est un médicament dangereux parfois jusqu'à la mort.' D'autre part, il paraît produire souvent une fixation du poison syphilitique sur le système nerveux. En tout cas, on ne doit pas répéter les injections sans avoir vérifié l'élimination de l'arsenic.

L'atoxyl produit plus facilement des névrites que le 606.

Je viens pourtant d'observer le cas d'un soldat d'infanterie coloniale qui, pour la maladie du sommeil, avait reçu un grand nombre de piqûres d'atoxyl sans accidents aux oreilles.

Les injections qu'on fait intra-musculaires, dans l'épaisseur des fessiers, loin des branches du sciatique sont pratiquées, soit à la solution neutre insoluble qui présente l'inconvénient de s'enkyster en retenant une partie du produit, soit à la solution alcaline soluble qui est plus active mais plus douloureuse.

Le Professeur Ehrlich a recommandé par ordre d'efficacité, d'abord les injections intra-veineuses avec les solutions alcalines dans 200 centigrammes de sérum physiologique, puis les injections alcalines solubilisées intra-musculaires, enfin les injections acides intra-musculaires.

On reproche surtout au 606 de ne pas donner de résultats durables après une seule injection, mais rien n'empêche de les répéter, après qu'on a constaté son élimination pour éviter l'accumulation.

Un des grands avantages du néo-salvarsan est de simplifier la technique. Pour l'injection intra-veineuse, on n'a plus besoin de soude caustique. Le produit se solubilise par une simple agitation, soit dans le sérum faible chloruré, soit dans l'eau distillée.

On peut au besoin employer le remède en lavement, Carle (*Lyon médical*, 29 septembre 1912) indique les doses suivantes : $0^{gr},45$ de néo-salvarsan pour 200 c. d'eau distillée. Donner un lavement une fois par semaine, pendant quatre semaines consécutives.

A propos de la réaction de Wassermann, je dois signaler un antigène préparé par A. Desmoulière. Les nombreuses expériences faites chez le Professeur Gaucher ont montré que cette méthode décèle la syphilis dès ses premiers jours, à une période où l'ancien Wassermann ne le permettait pas, et il révèle son existence dans des cas où l'ancien Wassermann ne donnait que des résultats douteux ou négatifs.

Le mercure lui-même n'est pas inoffensif. Dans un travail que j'ai

présenté au XIII[me] Congrès International, Paris, 1900, sur ' Les poisons de l'oreille ', je concluais que, *par exception*, il pouvait être rendu responsable de la surdité chez les syphilitiques.

Je viens d'observer le cas chez une jeune femme traité par des injections de benzoate ; elle a été prise de surdité bilatérale et prolongée.

D'après le Professeur Gaucher, le néo-salvarsan est plus dangereux que le 606 et les neuro-récidives doivent être considérées comme des accidents du 606 ou des syphilis méconnues.

Deuxième Partie : Applications et Résultats

Après cet exposé d'ensemble sur les diverses méthodes et techniques spéciales, nous devons examiner les résultats successivement pour les oreilles, le nez et la gorge.

Le procédé le plus rationnel nous paraît être d'exposer d'abord les observations qui sont venues à notre connaissance et d'en déduire les conclusions naturelles.

1° *Oreilles*

Observation 1. Le premier cas que j'ai observé, alors qu'il n'était pas encore question d'accidents auriculaires est celui d'un homme de 35 ans, qui avait contracté la syphilis à 20 ans. Comme il avait une gomme du tibia gauche, il lui fut fait trois injections de 606 à Francfort. La première en septembre 1910, une deuxième en novembre et la troisième le 3 janvier 1911. Dix jours exactement après cette troisième injection, il fut pris de douleurs généralisées en même temps que de douleurs fortes dans la tête et les deux oreilles. La crise était plus accentuée à l'oreille droite. Un écoulement de sang s'y produisit. Quand je vis le malade, je constatai une surdité complète à droite avec Rinne normal et une demi-surdité à gauche avec Rinne normal.

Toute la membrane tympanique droite et surtout la partie voisine du conduit auditif était d'un rouge ecchymotique sans perforation. A gauche, congestion légère des mêmes parties.

Il me paraît bien que le 606 devait être tenu pour responsable de ces congestions graves.

Observation 2. Jeune homme de 20 ans, que j'examine pour la première fois, le 2 décembre 1911. Il a eu en avril dernier deux chancres à la verge qui ont été reconnus pour syphilitiques à l'hôpital Cochin.

On lui fait six piqûres mercurielles. Deux mois après éruption pustuleuse sur tout le corps.

A ce moment, 3 piqûres de 606 sur un espace de trois semaines. La première fut de $0^{gr},60$ et les deux autres de $0^{gr},30$. Dès la première, les pustules disparurent et le malade se croyait complètement guéri.

Or, le 15 novembre 1911, se montrent des vertiges et une paralysie faciale gauche.

Le 25 novembre, une surdité s'installe à gauche, en deux ou trois jours de temps, avec bourdonnements et douleurs spontanées vives à la mastoïde gauche.

Ni le Barany, ni la giration ne font apparaître le nystagmus.

La surdité est complète à gauche.

Observation 3. Femme de 28 ans, syphilis contractée en septembre 1911. Trois mois après roséole, qu'une injection unique de Salvarsan fait disparaître en trois jours. Vers le milieu de février 1912, deux mois après l'emploi du Salvarsan, surdité et bourdonnements à l'oreille gauche, avec vertiges. Tous ces accidents, accompagnés de maux de tête, s'étaient développés en trois jours.

Les injections de mercure et l'iodure améliorèrent l'ensemble de la maladie, mais la surdité tympano-labyrinthique persiste à gauche.

Étant donné que deux mois s'étaient écoulés entre une seule injection de 606 et la surdité, je me demande si la complication auriculaire ne doit pas être attribuée, plutôt à la syphilis qu'à l'arsénobenzol ?

Observation 4. Femme de 38 ans, nerveuse qui a eu des otorrhées presque continuelles aux deux oreilles depuis son enfance.

En septembre 1910 un chancre de la langue. La malade, qui était enceinte, sent peu à peu que l'enfant ne remue plus et le 4 décembre 1910 elle avorte au huitième mois de sa grossesse, et, d'après la sage-femme, l'enfant était mort depuis un mois.

A ce moment la malade semble avoir eu un peu d'eczéma.

En janvier 1911, éruption de syphilis à la face, à la tête et au cou.

En avril 1911, on commence le traitement mercuriel — cent pilules et vin arsénical. L'éruption disparaît brusquement en masse.

En juillet 1911, apparaît brusquement une surdité double et complète : bruits intenses, vertiges qui provoquent plusieurs chutes sérieuses. La malade est prise d'idées noires.

En septembre 1911, la malade se présente dans une clinique privée où on institue le traitement par le 606. En quinze jours, on lui fait trois injections intra-veineuses à huit jours de distance et douze injections intra-musculaires. Les vertiges disparaissent définitivement ainsi que la céphalée. Le nez se dégage et rend des mucosités abondantes. On commence les insufflations d'air dans les trompes qui améliorent encore l'audition.

En janvier 1912 l'audition s'est sérieusement améliorée.

En février l'écoulement des oreilles revient pendant six jours et ramène avec lui l'audition.

Puis, en mars, à la suite d'une violente contrariété, paraît-il, l'audition disparaît à nouveau.

État actuel (15 juin 1912). — La malade entend assez bien, surtout si on lui parle lentement. A noter que la malade est très nerveuse et qu'à la moindre contrariété elle éprouve beaucoup de bourdonnements.

Mais les mucosités nasales sont redevenues abondantes et croûteuses comme avant l'emploi du 606 (Brière).

Observation 5. Homme de 35 ans. A contracté la syphilis il y a deux ans (chancre de la verge). Un an après (janvier et février 1911), il est traité d'une roséole à l'hôpital Ricord où il reçoit trois injections de 606 ($0^{gr},40$, $0^{gr},40$, $0^{gr},45$), espacées de cinq jours en cinq jours. La dernière fut faite le 19 février 1911 et quinze jours après il ne restait plus trace de la roséole.

C'est en mai 1911 que le malade nota une disparition presque complète de l'acuité auditive à gauche. La vue était intacte.

Je l'examine le 26 avril 1912, dans le service du Professeur Gaucher, à l'hôpital Saint-Louis.

Les deux membranes tympaniques sont légèrement opalescentes, les manches du marteau relevés.

La surdité est complète à gauche, non seulement pour la voix, mais même pour les diapasons.

L'oreille droite est indemne. Le malade m'a dit qu'il avait mis six semaines à perdre complètement cette oreille.

Les détails de l'examen me firent conclure à une surdité cochléaire de l'oreille interne toxique.

Observation 6. (Service du Professeur Gaucher.) Jeune homme de 20 ans. A un chancre à la fin d'août 1911. Six piqûres de biodure. Roséole deux mois après. Il reçoit alors trois piqûres intra-veineuses de 606, espacées d'une semaine : la première de $0^{gr},60$ et les deux autres de $0^{gr},30$.

Vers le 15 novembre 1911, il est pris de névralgie faciale gauche et de paralysie faciale périphérique complète (front, orbiculaire, facial inférieur), du même côté.

Huit jours après, bourdonnements, sifflements et surdité à l'oreille gauche.

Le Wassermann était négatif.

Enfin, l'hectine, qui passe pour être le moins toxique, mais qui n'est peut-être pas le plus efficace des produits arsénicaux organiques, n'avait pas encore donné lieu à des accidents neurotoxiques, quand MM. le Professeur Gaucher et H. Guggenheim eurent l'occasion d'en observer trois cas dont voici l'observation résumée :

Observation 7. Homme de 64 ans, syphilitique secondaire, atteint de sclérose otique ancienne. Traitement par l'hectine. Cinq mois après le début du traitement, alors que le malade a reçu quatorze grammes d'hectine administrés en quatre séries d'injections quotidiennes séparées par trois intervalles de repos (deux intervalles de quinze jours, un intervalle d'un mois) apparition de bourdonnements d'oreilles. A une diminution unilatérale légère de l'acuité auditive qui existait depuis des années succède brusquement une surdité bilatérale très marquée. L'examen otologique montre l'existence d'une otite interne double (limitée au nerf cochléaire). Sous l'influence de sept autres injections de 0,40 d'hectine, la surdité et les bourdonnements ne font qu'augmenter. Après cessation de tout traitement, les troubles de l'ouïe rétrocèdent.

Observation 8. Homme de 51 ans ayant eu un chancre syphilitique en novembre 1910. Quelques jours plus tard apparition d'une syphilide papuleuse généralisée qui disparaît après quatorze injections de 0,02 de benzoate.

Le 1er janvier 1911 on constate des syphilides papulo-hypertrophiques périanales et on commence une série d'injections d'hectine de 0,20. Les accidents ne cessent qu'après cinquante-sept piqûres d'hectine et le 19 mars le malade étant guéri depuis huit jours, se plaint de bourdonnements d'oreille intenses. L'acuité auditive est un peu diminuée des deux côtés. On cesse tout traitement et les accidents auriculaires disparaissent.

Observation 9. Homme de 55 ans. Syphilis nasale tertiaire. Il reçoit trente-deux injections d'hectine de 0gr,20.

Une surdité grave bilatérale s'installe progressivement, accompagnée de bourdonnements violents. L'examen otologique, pratiqué par M. Castex, permet de constater l'origine labyrinthique de ces troubles de l'ouïe. On cesse l'hectine. Trois mois après, la surdité s'est un peu atténuée, mais les bourdonnements persistent avec quelques vertiges.

Observation 10. Homme de 53 ans. Surdité syphilitique et néo-salvarsan. Surdité complète de sorte que j'ai pu avoir les renseignements seulement en causant avec lui par l'écriture. Chancre de la verge en 1904. Trois mois après, plaques muqueuses dans l'arrière bouche. Pilules de mercure et Kl. Puis sur un espace de six ans, un 10e de piqûre d'huile grise.

A partir de ce moment, n'a plus eu d'accidents.

En septembre 1911, devient un peu sourd, puis brusquement dans une nuit du commencement de décembre 1911 devient complètement sourd. N'avait rien remarqué dans cette nuit.

Dans un hôpital de Paris (Broca), on vient de lui faire six piqûres de néo-salvarsan en huit jours, les premières de 0,45, les autres de 0,60.

Il déclare que depuis ces piqûres il entend moins encore, mais que ses bourdonnements sont devenus moins forts quoique mués en bouillonnements et cascades. Pas de vertiges, mais plus fatigué par l'attention, par exemple, s'il joue aux cartes.

Examen des oreilles montre les deux tympans scléreux. Diapasons perçus seulement en leur vibration et non en sons.

Au total, le Salvarsan paraît avoir aggravé une sclérose diffuse et une labyrinthite syphilitique.

7 novembre 1912. On a arrêté les injections et cependant toujours aussi sourd avec bouillonnements.

Observation 11. *Un cas de labyrinthite à forme grave après injection de Salvarsan.* M. Guisez rapporte le cas d'un malade qui, à la période primaire, reçut deux injections de 20 et de 30 centigrammes de Salvarsan. Quatre semaines après, survinrent de la surdité, des bourdonnements, des vertiges, et un état de malaise spécial qui rendait la situation tout à fait critique.

Dans les surdités spécifiques de la période primaire, on n'observe

jamais des accidents de pareille intensité ; aussi doit-on les attribuer au Salvarsan injecté un mois auparavant. Les phénomènes se sont amendés progressivement par la cessation de la médication et l'administration d'un traitement mercuriel au calomel. Ce malade avait eu, antérieurement déjà, des poussées de labyrinthite légère, et ceci doit nous rendre assez circonspects dans l'emploi du 606 lorsqu'il y a un passé pathologique, même léger, du côté des oreilles.

(Soc. d'O.-R.-L. de Paris, 11 mars 1912.)

Observation 12. Atrophie du biceps droit et surdité gauche, consécutives à des injections de 606.

(Service du Professeur Gaucher, Hôpital Saint-Louis.)

Homme de 42 ans. En mai 1911, chancres spécifiques du sillon, traité par le Salvarsan à l'hôpital de Rennes. Six injections intraveineuses, faites à trois jours d'intervalle. A chaque séance, on injectait dans les deux bras. Ne se souvient pas des doses.

Quatre à cinq jours après les injections, le malade s'aperçoit que son bras manque de force.

Examen otologique par le Docteur Antoine Courtade le 10 septembre 1912 : — Légère opalescence de la membrane tympanique gauche, et rétraction du manche du marteau, indiquant une obstruction tubaire ancienne. Altération semblable à droite, mais moins accentuée. Au point de vue fonctionnel, le diapason est entendu à droite (la 3) les $\frac{4}{5}$ du temps normal. Il est entendu à gauche la moitié du temps normal. La diminution de l'ouïe à gauche est due à une altération du nerf auditif, la lésion de l'oreille moyenne étant trop légère pour expliquer la surdité. Il est incontestable qu'il s'agit d'une névrite toxique. La surdité s'est établie graduellement, à la suite de bourdonnements qui persistent encore. Ces bourdonnements ont débuté cinq jours après, ils étaient intenses et généralisés. Avant le traitement par le 606, le malade n'avait rien présenté de semblable et entendait fort bien.

Un examen oculaire, fait par le Docteur Antonelli, a été négatif.

Observation 13. Femme de 20 ans qui j'ai examiné à l'hôpital Saint-Louis (Service du Professeur Gaucher), le 1er octobre 1911.

Elle a contracté la syphilis en juin 1911 (chancre vulvaire, roséole, plaques muqueuses dans le pharynx, adénopathies inguinales).

Vers la fin de juin 1911, une piqûre intra-veineuse de 606 faite à Londres. Les accidents disparaissent, mais reviennent au bout de quinze jours, céphalées intenses.

Quinze jours après l'injection se déclarait une surdité à gauche avec paralysie faciale du même côté.

Un premier examen de cette oreille par le Docteur Maurice Bloch. Le tympan est normal. La malade entend la voix haute à un mètre de distance.

Elle accuse aussi des troubles oculaires de l'œil gauche survenus quinze jours après le 606. L'examen pratiqué par le Docteur Antonelli indique de la névrite optique à forme rétro-oculaire, presque guérie.

Elle fut réexaminée par le Docteur Courtade le 5 décembre 1911. Il y a toujours à gauche une diminution de l'ouïe aux trois quarts. La perte existe surtout pour les tons graves.

Le Docteur Courtade concluait que sur cette malade des lésions au niveau de l'oreille moyenne avaient préparé l'effet nocif du 606.

Observation 14. Femme de 38 ans, qui se présente à ma clinique en avril 1912. Chancre buccal en juin 1910, quatre mois après accidents cérébraux, neurasthénie et quelques semaines après symptômes démentiels. Une fausse couche au mois de décembre 1910.

Au mois d'octobre 1911, on commence les piqûres de 606, quand déjà depuis trois mois elle avait des bourdonnements dans les deux oreilles avec diminution de l'ouïe.

Elle reçut quinze piqûres de 606 faites dans une autre clinique. Six semaines après le commencement du 606, l'état général et l'état cérébral étaient bien meilleurs. On pouvait de nouveau causer avec la malade.

Mais peu à peu et depuis deux mois environ, l'ouïe a disparu de nouveau.

Quand je l'examine (27 avril 1912), je constate une double otite adhésive ancienne avec vastes perforations. Le diapason n'était aucunement perçu.

Observation 15. Homme de 28 ans que j'examine en octobre 1912 dans le service du Professeur Gaucher. Syphilis contractée il y a huit ans. Un an après il avait un écoulement de l'oreille droite. En mai 1912 on lui fait deux piqûres de 606. Depuis, il entend moins surtout de l'oreille droite et il a des sifflements qu'il ne ressentait pas avant les piqûres de 606.

Je reproduis maintenant un certain nombre d'observations recueillies dans les divers périodiques de notre spécialité et qui serviront avec les miennes à établir mes conclusions.

Ce sont d'abord trois faits publiés par mon collègue Georges Gellé (Société française d'O.-R.-L., mai 1912), et dont je ne reproduis que les résumés, renvoyant le lecteur pour plus de détails à l'excellent mémoire original.

Observation 16. Syphilis récente traitée par le 606. Cinquante jours après la première injection, surdité gauche. Nouvelle injection ; trois jours après, surdité droite ; la surdité droite disparaissait totalement au bout de sept jours, la gauche persiste.

Observation 17. Chancre traité par le 606. Soixante-trois jours après la première injection et quarante-six jours après la dernière, paralysie faciale droite périphérique, puis paralysie du cochléaire et du vestibulaire droits.

Observation 18. Syphilis datant de décembre 1910. En 1911, Salvarsan. Quarante-deux jours après la dernière injection, surdité bilatérale (nerfs cochléaire et vestibulaire). A droite, la surdité disparaît au bout de quinze jours. Elle persiste à gauche.

Observation 19. Homme de 25 ans, syphilitique depuis décembre 1909 Le 20 juillet 1910, surdité progressive de l'oreille droite et trois semaines

après surdité de l'oreille gauche. Lors de son admission surdité totale. Un peu de vertiges, bourdonnements intenses. Pas de manifestations cutanées.

Tympans normaux des deux côtés.

Traitement mercuriel intense.

Huit jours après légère amélioration de l'oreille gauche.

14 septembre, une injection de Salvarsan. Le malade a l'air d'entendre un peu mieux surtout à droite.

Puis injections au calomel, et le 24 octobre il pouvait tenir une conversation avec les autres malades, quoique entendant encore très mal.

Cette observation est plutôt en faveur du mercure que du 606.

(Jörgen Möller, *Archiv für Ohrenheilkunde*, 1911, 97.)

Observation 20. H. Neumann (Lésion syphilitique de l'oreille interne). Tout au début d'une syphilis, lésion isolée du nerf vestibulaire. Injections de mercure sans effet. 0,45 de 606 aucun effet non plus.

(*Arch. f. Ohrenh.*, 1911, 184.)

Observation 21. O. Beck (Deux cas de syphilis héréditaire traités par le 606).

(*a*) Garçon de 13 ans, surdité progressive depuis deux ans. L'audition monte deux jours après l'injection, pour la voix haute à l'une des deux oreilles, de 0,25 à 2½ m., et pour l'autre oreille de 4 m. à 9 mètres.

Observation 22. (*b*) Surdité complète et inexcitabilité complète du nerf vestibulaire. Aucun résultat par le 606.

(*Arch. f. Ohrenh.*, 184 ; Société autrichienne d'Otologie ; *Arch. f. Ohrenh.*, 1911, 174 et suiv.)

Observation 23. O. Beck (Abolition transitoire isolée du nerf vestibulaire chez des syphilitiques traités par le 606. Réaction d'Herscheimer dans le domaine de la huitième paire).

Dans le premier cas décrit par Ruttin, il s'agit d'une lésion rétro-labyrinthique du nerf vestibulaire qui, l'audition étant conservée, présente les symptômes de l'abolition aiguë des fonctions du labyrinthe : vertiges subjectifs, troubles de l'équilibre, quelquefois vomissements, nystagmus vestibulaire vers le côté sain. Inexcitabilité totale de l'appareil vestibulaire aux excitations caloriques, rotatoires et galvaniques.

Dans quatre cas, ce syndrôme fut consécutif à une injection de 606, chez des malades qui n'avaient pas été traités spécialement pour leur affection auriculaire, mais dont les oreilles n'étaient pas normales.

Une explication de ces quatre cas où les symptômes disparurent après douze ou quatorze jours est difficile.

Plusieurs interprétations possibles :

1° Névrite toxique transitoire, un cas analogue de névrite toxique au cours d'un rhumatisme observé par Ruttin. Un autre cas de Leidler dans empoisonnement par des saucisses.

Un troisième cas analogue a été observé par Barany au cours d'une syphilis non-traitée par le 606.

2° Urbantschitz admet des phénomènes inflammatoires dans le domaine du nerf.

Observation 24. O. Beck (syphilis papuleuse du pavillon de l'oreille avec infiltration molle du pavillon). Papules dans le conduit auditif externe. Otite moyenne suppurée subaiguë. Injection du 606.

Au cinquième jour après l'injection de $0^{gr},50$ cent. de 606, toutes les manifestations ont disparu.

L'oreille moyenne est tarie et on voit une perforation sèche dans le quadrant antéro-inférieur du tympan.

Observation 25. O. Beck (troubles de surdité labyrinthique avec vertige de Menière, traités par le 606). Sur ces trois cas, deux malades syphilitiques — amélioration de l'audition des deux oreilles chez le premier. Chez le second, amélioration seule de l'oreille la moins malade, aucun effet sur l'autre.

Chez le troisième malade (une syphilitique) aucune amélioration par le 606.

Dans tous ces cas, employer seulement petites doses, soit $0^{gr},40$ cent. en tout.

(*Arch. f. Ohrenh.*, 1911.)

O. Beck (Lésion tardive de l'appareil cochléaire et vestibulaire consécutive au 606). Chez deux syphilitiques ayant avant les oreilles normales.

Observation 26. (*a*) Jeune fille de 20 ans, une injection de 606 pour un chancre. Soixante-cinq jours après, subitement, surdité, vertiges, Rinne positif des deux côtés, raccourcissement de la durée de la conduction osseuse et aéro-tympanale, nystagmus. Voix haute à un mètre. Réactions caloriques faibles, réaction galvanique de 56 milli ampères. La malade est restée quatre semaines dans le même état.

Observation 27. (*b*) Cas intéressant de polynévrite cérébrale meniériforme (type Frankel-Hochwart). Jeune femme de 22 ans. Injection pour un chancre. Le Frankel-Hochwart a paru sept semaines après l'injection quand l'ensemble de la syphilis avait été enrayé. Pas d'accidents secondaires. Or, quand F.H. a paru, réaction de Wassermann est devenue positive, seulement alors. La surdité est donc bien ici syphilitique.

(*Arch. f. Ohrenh.*, 1911.)

Haike et Wechselmann. (Action curative du 606 sur l'oreille.)

Dans 5 cas d'affection syphilitique de l'oreille ils emploient le 606.

Observation 28. (*a*) Syphilis datant de 18 ans, diminution de l'audition et bourdonnements. Otite interne à gauche. Amélioration de l'audition par 0,60 cent. de Salvarsan sous-cutané.

Observation 29. (*b*) Malade de 33 ans. Syphilis datant de 7 ans. Otites moyennes et internes depuis 2 ans. Amélioration insignifiante par le Salvarsan.

Observation 30. (*c*) Malade de 33 ans. Syphilis de 1908. Depuis, bourdonnements et vertiges, diminution des fonctions vestibulaires. Injections de Salvarsan sans résultat.

Observation 31. (*d*) Homme de 55 ans. Syphilis ancienne. Surdité double. Amélioration de l'audition par le Salvarsan.

Observation 32. (*e*) Garçon de 14 ans. Hérédo-syphilis, surdité, vertiges, non-influencé par le mercure. Amélioration de l'audition. Cessation des vertiges après une injection de 606.

(*Arch. f. Ohrenh.*, 1911, 213.)

Haike et Wechselmann (Phénomènes accessoires du 606 sur les oreilles). (*Arch. f. Ohrenh.*, 19.)

Sur 3,000 malades traités par le 606, H. et W. ont observé sept fois une affection auriculaire.

(*a*) Affection cochléaire gauche guérie par le mercure.

(*b*) Affection cochléaire droite guérie par le 606.

(*c*) Labyrinthites et otites moyennes des deux côtés, paralysie faciale à droite. Amélioration par le 606 et le Hg.

(*d*) Bourdonnements qui ont guéri sans traitement.

(*e*) Labyrinthites et otites moyennes, guéries par le Salvarsan.

(*f*) Otite moyenne double guérie sans traitement.

(*g*) Affection cochléaire guérie par le Salvarsan.

Conclusions : Les affections de l'acoustique consécutives aux injections de 606 sont imputables seulement à des lésions de syphilis.

Observation 33. Névrite cochléaire double consécutive au Salvarsan dans une syphilis récente.

Forgeron de 33 ans. Chancre. Injection de 0,70 cent. de Salvarsan. Quatre mois et demi après le chancre et l'injection, surdité double avec tintements de cloches. Affection cochléaire pure sans rien au vestibule. Krick admet une action toxique du 606 sur l'oreille.

Être prudent sur le Salvarsan dans les professions à marteau.

Knick (*Arch. f. Ohrenh.*, 1911, 277).

Observation 34. (*a*) Syphilis congénitale, névrite cochléaire double syphilitique chez jeune homme de 19 ans. Sept jours après une injection de Salvarsan, amélioration de l'ouïe :

Voix chuchotée à droite (du contact à 0m,75).

Voix chuchotée à gauche (de 0,30 à 12 mètres).

Observation 35. (*b*) Petite fille de 11 ans, surdité complète à droite, à gauche elle entend la voix parlée à 0m,40. Vertiges — quatre jours après une injection de Salvarsan. A gauche entend à 1m,50, quatre jours après à 3 mètres. Quatre jours après encore à 0m,40. N'avait rien donné à droite.

Lang dit de ne pas en faire chez les gens qui ont un passé auriculaire.

Lang (*Arch. f. Ohrenh.*, 1911, 203).

Observation 36. O. Beck. Exclusion passagère du nerf vestibulaire (2 cas) après injection de Salvarsan.

1° Une malade. Stade secondaire de syphilis. Deux semaines après injection de Salvarsan, vertiges, nystagmus vestibulaire vers la gauche. Audition et oreille moyenne intactes des deux côtés. Quinze jours après l'apparition des vertiges, quelques vertiges rotatoires, la malade ne

pouvant pas garder l'équilibre. Nerf vestibulaire droit absolument inexcitable. Guérison spontanée en quinze jours. L'injection avait été sous-cutanée.

Observation 37. Injection intra-veineuse. Six heures après, symptômes subjectifs et objectifs de l'exclusion de la huitième paire ; symptômes qui disparaissent en quelques heures.

Observation 38. O. Beck. Surdité intense chez une fille de 6 ou 7 ans, atteinte de syphilis héréditaire, améliorée sensiblement par le Salvarsan. Beck dit que ce cas confirme le fait que le Salvarsan peut agir favorablement dans les syphilis héréditaires où il y a encore des restes auditifs.

(Soc. autrichienne d'Otologie, 30 janvier 1911.)

Observation 39. O. Beck. Exclusion bilatérale de l'appareil vestibulaire. A droite surdité complète ; à gauche surdité intense, après Salvarsan. Chez un chauffeur, cinq semaines après une injection. Pendant les cinq semaines que les symptômes mirent à se développer, la réaction de Wassermann resta négative. Donc, c'est bien le fait du Salvarsan.

(*Arch. f. Ohrenh.*, 1912, 29.)

Observation 40. Braun. Un cas de syphilis d'oreille interne guérie par le Salvarsan. Homme de 31 ans, syphilitique depuis 6 ans.

(*Arch. f. Ohrenh.*, 1912, 32.)

Observation 41. O. Beck. Homme, cinq semaines après Salvarsan, surdité complète et exclusion des labyrinthes. Wassermann négatif. Frictions mercurielles sans résultat. On aurait trouvé arsenic dans les urines et quand l'arsenic a commencé à diminuer dans les urines, l'ouïe est revenue progressivement.

Observation 42. Sugàr (Société royale de médecine de Budapest, 19 novembre 1910) parle d'un malade atteint d'oto-sclérose pour lequel il avait déconseillé le Salvarsan. Néanmoins le sujet se fait faire une injection. Aggravation considérable (vertiges).

Il conclut qu'avant le Salvarsan, on devrait toujours examiner les oreilles.

(*Arch. f. Ohrenh.*, 1912, 101.)

Ehrler (Hôpital d'Eppendorfer) dit que le Salvarsan dans vieilles affections osseuses et cartilagineuses syphilitiques du crâne et du larynx risque de produire une mobilisation des spirochètes qui se traduit par de nouveaux accidents syphilitiques.

Observation 43. (*a*) Infiltration gommeuse de l'omoplate — Salvarsan — Augmentation menaçante de l'œdème et de la dyspnée.

Observation 44. (*b*) Érosions à la base de la langue et de la luette — Salvarsan — petite perforation de la voûte.

Observation 45. (*c*) Périostite du dos du nez — Salvarsan — kératite double.

Observation 46. (*d*) Affection grave du squelette nasal et sphénoïde avec cécité double — Salvarsan — méningite mortelle.

(*Arch. f. Ohrenh.*, 1912, 102.)

Observation 47. Sainton, à la Société médicale des hôpitaux de Paris (15 mars 1912) citait le cas d'une femme de 27 ans qui, trois mois avant, contractait un chancre et avait reçu cinq injections de 606 (quatre de $0^{gr},30$ cent., et une de 0,25 cent.).

Un mois après la dernière injection la malade présentait tout à coup une paralysie faciale totale, des phénomènes d'anesthésie dans la sphère du trijumeau, une surdité totale à droite et une énorme quantité d'albumine alors qu'avant le 606 on n'en constatait que les traces.

Sous l'influence d'une seule injection de 1 cc. de biodure, l'albuminurie a disparu et la paralysie rétrocédé.

Sainton fait remarquer combien ici il est difficile de dire ce qui appartient à la syphilis ou au 606.

Observation 48. Homme de 25 ans, chancre pénien en septembre 1910. Plaques muqueuses du pharynx et syphilides sur la peau trois mois après. Quatre mois après le chancre un peu de surdité progressive double avec quelques bourdonnements rares. Six mois après le chancre trois piqûres de 606 à deux jours d'intervalle. Les syphilides cutanées pharyngiennes ont été améliorées mais les oreilles n'ont été ni aggravées ni améliorées par le 606.

Mon examen du malade m'a montré une sclérose tympanique double légère qui semblait antérieure au chancre.

En tout cas, ici le 606 n'a pas aggravé un état otopathique antérieur.

Deux autres observations récemment recueillies m'ont montré que le Salvarsan n'est pas toujours nuisible même quand il y a un passé auriculaire.

Observation 49. Malade : homme, syphilitique depuis 7 ans, avec syphilome diffus du larynx depuis un an. A l'apparition de cette laryngopathie, il a reçu quatre piqûres de 606. Elles n'ont en rien aggravé une otite adhésive droite antérieure.

Observation 50. Homme ayant reçu deux injections de Salvarsan. Aucun inconvénient pour ses oreilles bien qu'il eut une otorrhée à gauche avec perforation au centre de la membrane.

Observation 51. Mon collègue Maurice Bloch m'a communiqué l'observation suivante, recueillie par lui dans le service du Professeur Gaucher, à l'hôpital Saint-Louis. Elle s'ajoute à celles qui montrent la nocivité du Salvarsan pour les nerfs crâniens.

Homme de 35 ans, ayant contracté la syphilis il y a quinze mois. Au moment du chancre initial, il reçut une injection de 606. Vingt jours après paralysie faciale gauche et labyrinthite du même côté. La conduction osseuse est complètement abolie à gauche et la conduction aérienne y est très écourtée.

Il s'agit donc de polynévrite acoustique et faciale.

Pour Imhofer (de Prague) (le Salvarsan en oto-rhino-laryngologie, 1911), le 606 est contre-indiqué partout où avant l'infection syphilitique il existait une affection quelconque de l'oreille, à l'exception des cas où il y a surdité complète antérieure. En tout cas, il faut prévenir les

intéressés que le Salvarsan peut aggraver leurs oreilles et au besoin leur faire signer un papier d'acceptation, comme le conseille le Professeur Gaucher.

Si, après avoir étudié la question, d'après les faits observés, nous passons en revue ce qui a été publié, nous voyons :

O. Beck déclare que depuis l'ère du Salvarsan on voit beaucoup plus de lésions nerveuses graves dans la domaine de la dix-huitième paire. A l'appui de son assertion, il consigne trois cas de surdité unilatérale complète consécutifs à une seule injection de Salvarsan.

(*Arch. f. Ohrenh.*, 1912, 54 ; Société autrichienne d'Otologie, 26 juin 1911.)

D'après Lang, les lésions de l'oreille interne viennent sûrement du 606 et la rétrocession des accidents par une nouvelle injection ne peut pas l'innocenter, car ces accidents disparaissent d'eux-mêmes à la longue.

Dans un cas de Wanner ou le 606 avait été employé contre un lichen ruber, il y eut des accidents d'oreilles.

M. Milian, d'après sa pratique personnelle évalue à 58 pour cent les cas où la surdité des syphilitiques peut être imputée au Salvarsan. Les accidents se montrent vingt-quatre ou trente-six heures après l'injection et consistent en surdité incomplète, généralement unilatérale qui dure quelques jours et disparaît sans laisser de reliquats.

M. Milian fait remarquer d'ailleurs qu'on peut se prémunir contre les accidents en graduant les doses et s'abstenant de faire d'emblée des doses fortes. Il admet que ces lésions auditives sont superficielles, ce qui lui fait supposer qu'il s'agit simplement de phénomènes congestifs et non de lésions toxiques.

(Société médicale des Hôpitaux, 15 mars 1912.)

Lévy considère comme typiques les manifestations auriculaires qui surviennent même cinq ou six semaines après le Salvarsan. Il a observé un cas où la surdité après Salvarsan est restée stationnaire.

(Société d'Otologie de Berlin, 23 juin 1911.)

Il n'est pas d'ailleurs possible d'expliquer toujours les accidents après le Salvarsan par une récidive de syphilis.

Par contre, nous pouvons enregistrer les documents suivants :

Haike, après une première injection de Salvarsan, a constaté une surdité avec bourdonnements et paralysie faciale, mais après la deuxième injection, la paralysie du facial a disparu et après la troisième les symptômes auriculaires. Il admet que, dans ce cas, il y avait participation de l'oreille moyenne. Il a vu des cas de succès ou d'échec du 606 dans la syphilis de l'oreille.

(Société berlinoise d'Otologie, 17 février 1911.)

Bernhardt mentionne l'amélioration par le 606 d'une otite interne syphilitique. Krumbein a traité 6 cas d'otite syphilitique par le Salvarsan. Quatre fois il a noté une grande amélioration.

(Société d'Otologie de Berlin, 23 juin 1911.)

Ballenger d'Atlanta a employé le 606 dans 110 cas, sans accidents auriculaires.

La question se pose souvent de savoir si les accidents otiques sont dus au 606 ou à la syphilis elle-même. Je crois que lorsque les accidents sont rapides, graves et tenaces, ils relèvent plutôt de la syphilis.

2° *Nez*

Les observations concernant le nez sont bien moins nombreuses que celles de l'oreille.

Ehrler (*Monatschr. f. Ohrenh.*, 1911, n° 9) met en garde contre les effets nuisibles que peut présenter le Salvarsan dans certaines rhinopathies syphilitiques, à cause de l'irritation locale produite par le médicament et la mobilisation des spirochètes dans les organes avoisinants :

Observation 52. Un cas d'affection grave du squelette nasal, participation du sinus sphénoïdal. Injection de 606. Cécité rapide bilatérale. Mort par méningite.

Observation 53. Périostite du dos du nez. 606. Kératite grave de l'œil droit.

Observation 54. Par contre O. Beck chez un malade de 25 ans, hérédo-syphilitique à Wassermann positif et présentant une perforation de la cloison nasale, avec ozène fait une injection intra-musculaire de 0 gr. 70 cent. Dix jours après, amélioration de l'ozène, grande diminution des croûtes, épidermisation des bords de la perforation.

(*Arch. f. Ohrenh.* 1911.)

Observation 55. De même je viens d'observer un homme de 44 ans, vieux syphilitique, qui a reçu 6 piqûres de 606 sans qu'une perforation ancienne de sa cloison ait subi la moindre aggravation.

3° *Pharynx et Larynx*

Pour le pharynx et le larynx, les observations n'abondent pas non plus.

Dans bien des cas, le 606 a fait merveille encore que pour le larynx on ait noté des infiltrations dangereuses avec dyspnée (Ehrler).

M. Milian (des Hôpitaux de Paris) l'a très heureusement employé contre la stomatite mercurielle.

On savait depuis les travaux de Galippe que la stomatite mercurielle est d'origine microbienne. Il est démontré aujourd'hui qu'elle est une stomatite microbienne spécifique constituée par l'association fuso-spirillaire. Le mercure amène la stomatite en exaltant la virulence des microbes normaux de la bouche, c'est-à-dire l'association fuso-spirillaire qui est fréquente dans la bouche surtout si les dents ne sont pas en bon état. Se basant sur ces notions, le Dr Milian a pensé que le 606, qui détruit non seulement le triponème mais aussi la plupart des micro-organismes spirolés, serait préventif et même curatif de la stomatite mercurielle et de fait, le médicament lui a réussi dans les deux observations suivantes :

Observation 52. Malade traité par l'huile grise qui avait des poussées de stomatite telles qu'il fallait interrompre le traitement à la cinquième ou sixième injection. M. Milian fait cinq injections intra-veineuses de 606 et immédiatement après cette cure une série d'injections d'huile grise. Le malade n'a pas eu la moindre trace de stomatite, le 606 ayant agi préventivement comme un excellent désinfectant de la bouche.

Observation 53. Ici le 606 a agi comme curatif.

Une malade du service de M. Balzer présentait une stomatite très intense due à plusieurs injections d'huile grise. Elle ne pouvait ouvrir la bouche. La langue et les gencives étaient ulcérées, saignantes, dégageant une odeur putride, avec trismus. Le traitement local classique était resté sans résultats. M. Milian fait une injection de 0,20 cent. de 606. Dès le lendemain la malade pouvait écarter les mâchoires de 4 à 5 centimètres et la fétidité de l'haleine avait disparu. L'amélioration dura seulement quatre ou cinq jours, car il y avait toujours du mercure qui renouvelait les accidents. Alors nouvelle injection en ajoutant au traitement général des lavages au 606 très dilué. La malade guérit en une quinzaine de jours, alors que ces stomatites graves durent quelquefois deux mois.

Troisième Partie : Conclusions

Les sels arsénicaux nouvellement employés dans le traitement de la syphilis sont :

Le cacodylate iodohydrargyrique, l'énésol, l'atoxyl, l'hectine, l'arséno-phénylglycine, le Salvarsan, le néo-salvarsan.

Si cette médication a donné de bons résultats, elle a déterminé d'autre part des accidents graves qui ont été observés notamment sur les oreilles.

La préparation la plus récemment employée, le néo-salvarsan, a sur le Salvarsan l'avantage de n'être pas précipité par les sels acides du sang et de pénétrer ainsi dans les organes les moins vascularisés. Ses réactions tardives sont à peu près celles du Salvarsan, mais ses accidents sont moins intenses et moins prolongés. Son emploi est plus facile et mieux toléré.

Sur les nouveau-nés, il vaux mieux recourir à la voie sous-cutanée ou intra-musculaire, on emploiera au plus 15 milligrammes de néo salvarsan par kilogramme du poids de l'enfant.

Ces produits arsénicaux trouvent une contre-indication générale dans les maladies du foie et du rein. Ils sont dangereux chez les nouveau-nés quand les accidents sont confluents.

Ils paraissent produire souvent une fixation du poison syphilitique sur le système nerveux.

On ne doit pas en réitérer l'emploi sans s'assurer qu'il y a eu élimination de l'arsenic employé antérieurement.

Les conclusions que je présente ici sont tirées de ma pratique personnelle et des observations que j'ai pu recueillir un peu partout. Sur un total de cinquante-trois faits, j'en compte sept que j'ai pu suivre

personnellement. J'envisage successivement les effets aux oreilles, au nez et à la gorge.

En suivant l'ordre où j'ai énuméré les divers agents arsénicaux, nous trouvons d'abord l'atoxyl qui passe pour exposer plus aux névrites les nerfs crâniens que le Salvarsan et néanmoins nos médecins coloniaux qui l'emploient fréquemment pour divers cas dont la maladie du sommeil, ne signalent pas les accidents auriculaires.

L'hectine, que j'ai employée dans nombre de cas, ne m'a pas donné non plus de complications aux oreilles, nez, ou larynx, mais, d'autre part, mes malades en ont eu peu de bénéfice. L'amélioration rapide au début se ralentit vite. Les récidives ne sont pas rares et il faut en venir au mercure. Pourtant si je m'en rapporte à certaines observations publiées (Voir observations 7, 8 et 9), l'hectine se serait montrée particulièrement nuisible et quand on suspendait son emploi les troubles disparaissaient.

Mais, ce sont principalement les effets du Salvarsan qui ont été étudiés.

Une première question se pose : Pendant quelle période de temps après son emploi le 606 peut-il être tenu pour responsable des accidents signalés ? Un mois, me semble-t-il, d'après l'ensemble des observations, car souvent les accidents éclatent quelques jours après et ceux qui se déclarent deux ou trois mois après me paraissent bien plutôt imputables à la syphilis que le 606 n'a pas guéri. Il se montre alors seulement inutile et le cas n'est pas rare (Voir observation 2). D'après mes relevés sur un ensemble de 20 cas où des accidents d'oreilles ont été notés à la suite de l'emploi du 606, 9 fois ils étaient imputables à la syphilis même, 11 fois au Salvarsan.

A un autre point de vue, je note sur vingt-cinq faits bien étudiés :

Amélioration, 8 cas.
Aggravation, 17 cas.

L'amélioration des troubles auriculaires par l'arsénobenzol, quand elle se produit est assez rapide, en quinze jours par exemple (Observation 4), par contre elle n'est parfois que temporaire. Elle a été notée surtout chez les hérédo-syphilitiques (Observations 21, 32, 34, 35, 38), et, d'une manière générale, je crois à l'amélioration possible par le Salvarsan quand le malade n'a pas de passé auriculaire (Otorrhée tarie, sclérose otique, etc.) et même quand il n'a pas exercé de métier à marteau qui sclérose souvent le labyrinthe.

Les accidents produits aux oreilles par le Salvarsan consistent en douleurs, phénomènes congestifs, qu'on voit même sur le tympan et pouvant aller jusqu'à l'hémorragie et par-dessus tout, surdité (Observation 1).

Cette surdité peut n'être que transitoire (Observation 37), mais elle peut rester très grave si le sujet a déjà de la sclérose otique et de la labyrinthite syphilitique (Observation 10).

Or le 606 paraît surtout provoquer des lésions aux oreilles internes et peut-être même aux centres auditifs.

Depuis l'ère du Salvarsan les lésions graves de la huitième paire semblent augmenter beaucoup (O. Beck). M. Milian évalue à 58 pour cent les cas où la surdité des syphilitiques peut être imputée au Salvarsan. Je n'irai pas jusqu'à admettre avec Haike que tous les accidents d'oreilles observés après le 606 sont dus à la récidive de la syphilis traitée. En tout cas cette opinion tendrait à établir l'insuffisance de l'arsénobenzol puisqu'il ne préviendrait pas les effets de la syphilis sur l'oreille.

Lorsque, suspendant l'emploi du 606, on note de l'amélioration des troubles otiques, nul doute que cet agent n'en soit responsable.

Et si nous mettons en parallèle dans le traitement de la syphilis auriculaire le 606 et le mercure, nous voyons que quand on emploie alternativement les deux, le mercure semble le plus efficace (Observation 19) et que là où le mercure a échoué le Salvarsan n'a pas fait mieux (Observation 20).

D'ailleurs les partisans les plus convaincus de la nouvelle méthode et le Professeur Ehrlich lui-même admettent que le mercure et l'iodure de potassium restent un complément très important du traitement par le Salvarsan.

Pour le nez on a enregistré une méningite mortelle, mais c'est une exception sujette à discussion, et l'ensemble des observations est plutôt favorable au 606.

Ballenger (d'Atlanta) a vu disparaître des ulcérations du Cavum en 48 heures.

Pour le pharynx et le larynx, on s'en trouve bien aussi de façon générale. Je signale pourtant un cas d'infiltration œdémateuse du larynx (Observation 43), mais l'iodure de potassium nous expose au même danger. Le Docteur Milian s'est bien trouvé du 606 dans un cas de stomatite mercurielle.

En Résumé

Il me paraît résulter de mon enquête :

1° Que le Salvarsan et néo-salvarsan sont souvent inutiles et parfois nuisibles pour les oreilles, car ils semblent fixer le poison syphilitique sur le système nerveux.

2° Que dans les cas où l'on a employé le mercure chez le même sujet, celui-ci s'est montré plus efficace.

3° Que d'autre part le 606 est utile dans les affections syphilitiques du nez, du pharynx et du larynx.

4° Que l'atoxyl expose aux névrites et que l'hectine se montre peu utile.

L'emploi de ces produits arsénicaux me paraît donc d'une importance médiocre en oto-rhino-laryngologie.

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SECTION XV

RHINOLOGY AND LARYNGOLOGY

DISCUSSION No. 2

(Jointly with Section XVI, Otology, Discussion No. 3)

TREATMENT OF DISEASES OF THE THROAT, NOSE, AND EAR BY SALVARSAN AND OTHER ARSENICAL COMPOUNDS

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M. H. Vor einem Aerztekreise wie dem hier versammelten erübrigt es sich wahrlich, auf die Bedeutung hinzuweisen, die die Pathologie und Therapie der Syphilis gerade für den Rhino-Laryngologen hat. Muss bei dem furchtbaren proteusartigen Charakter dieser Krankheit eigentlich *jeder* Arzt auch etwas Syphilidologe sein, so besteht für den Hals- und Nasenarzt diese Nötigung mehr wie für jeden andern. Denn nächst den Genitalien wird kaum *ein* Organ des menschlichen Körpers so häufig von dieser Krankheit befallen wie die *Mund- und Rachenhöhle*, und zwar in *allen* Stadien ihrer oft über Lebensdezennien und das ganze individuelle Leben sich erstreckenden Dauer. Die Mund- und Rachenhöhle bildet nächst den Genitalien die häufigste Eingangspforte der Spirochaeta pallida, — etwa 40 Prozent aller extragenitalen Primäraffekte entfallen allein auf sie, Sekundäreffloreszenzen etablieren sich in ihr kaum seltener wie auf der Haut, bisweilen schon früher wie da und werden nicht selten hier allein empfunden und entdeckt. Und schliesslich bilden Mundrachen, *Nase* und *Kehlkopf* noch die Lieblingsstätten jener gefürchteten tertiären Zerstörungen, die Organfunktion und Leben gefährden können, mindestens aber ihre Opfer — Verbrechern gleich — mit einer Art von Zuchthausstempel aus der Haft entlassen, den sie als ein κτῆμα ἐς ἀεί unveräusserlich mit sich davon tragen.

So haben wir Rhino-Laryngologen denn Grund genug, allen Fortschritten der Syphilistherapie getreulich zu folgen und sorgsam jedes neue Mittel zu prüfen, das eine wirkliche Bereicherung unseres Könnens verheisst. Selbst *wenn* wir nun in den uralten erprobten Syphilis-Mitteln den Zauberstab hätten, immer und so rasch wie nötig allen bösen Folgen vorzubeugen und selbst Heilung zu garantieren, — selbst *dann* wäre der Erwerb eines neuen wirksamen Mittels froh zu begrüssen. Welcher Arzt aber wüsste nicht, wie oft Quecksilber und Jod, diese an sich so glänzenden Mittel, uns im Stich lassen. Sei es, dass es sich um refraktäre

Formen handelt, sei es um Patienten mit Idiosynkrasien gegen diese Mittel, sei es, dass der Erfolg nicht rasch und nicht vollständig genug eintritt.

So hat man sich denn in der neueren Zeit, besonders nachdem die Syphilisforschung durch die Entdeckung der Spirochaeta pallida, der Verimpfbarkeit der Syphilis und der Serumdiagnostik in ganz neue Bahnen getreten war, auch nach neuen Mitteln umgesehen und geglaubt, solche besonders im *Arsen* und in Verbindungen von Arsen und Quecksilber gefunden zu haben. Allerdings hat man Arsen schon seit Beginn des vorigen Jahrhunderts wiederholt gegen hartnäckige Syphilis ins Feld zu führen versucht (vgl. bei Naegeli, Ackerblom u. Vernier [269]). Unter den Arsenderivaten hat besonders das *Atoxyl* und das atoxylsaure Quecksilber von sich reden gemacht. Nachdem dieses Anilid der Metaarsensäure von dem englischen Kollegen Thomas [330] — Liverpool 1905 — gegen Trypanosomenkrankheiten empfohlen worden war, stellte zuerst Uhlenhut seine Wirkung auf *Tierspirochaeten*, besonders auf die Hühnerspirillose fest, und nach sehr günstigen Erfolgen bei der Affensyphilis wandte man es nach dem Vorgang von Uhlenhut, E. Hoffmann, Metschnikoff, Neisser und des Franzosen Salmon auch gegen die menschliche Syphilis an. Es konnten ganz entschiedene Erfolge verzeichnet werden, und zwar besonders bei den späten Formen der Lues, ebenso mit dem von Blumenthal [65-6] hergestellten *atoxylsauren Quecksilber*. Unter den von verschiedenen Instituten, so den von Lesser, Lassar, Blaschko, Neisser, Hallopeau u. a. mit dem Atoxyl erzielten Resultaten finden sich auch Heilungen von Affektionen der oberen Luft- und Verdauungswege, sowohl von Lippen- und Tonsillenschankern, wie von spezifischen Anginen, von Gaumenulcerationen etc.

Sehr bald aber wurde von den verschiedenen Seiten auf die Nachteile und Schädigungen der Atoxylbehandlung hingewiesen, so auf Stomatitiden, Albuminurie, auf die Idiosynkrasie vieler Patienten und vor allem auf die verhängnisvolle Wirkung des Mittels auf den *Sehnerv*. Dadurch wurde die anfängliche Begeisterung sehr rasch abgekühlt und das Mittel sehr bald von den meisten Aerzten wieder aufgegeben.

Nicht viel anders ist es mit den andern Arsen- und Arsenquecksilberverbindungen gegangen. So mit dem *Arsacetin*, dem *Arsenophenylglycin*, den Präparaten, die Ehrlich — vom Atoxyl ausgehend — zuerst entdeckte, dem *Asurol*, *Enesol* = Hg + methylarsensaures Natrium (Coignet, Bonau, C. Fränkel, Kahn), dem *Hektin* — Benzosulfo-Atoxyl u. a. Die Heilwirkungen aller dieser Mittel, — meist weit hinter denen des Quecksilbers zurückbleibend — standen in gar keinem Verhältnis zu den bisweilen verhängnisvollen Schädigungen, die sie den Patienten brachten; ihre '*Organotropie*' war im Verhältnis zu ihrer '*Spirillotropie*' viel zu gross. Auch an meiner Klinik sind mit einigen dieser Mittel Versuche gemacht und auch lokale Erfolge in den oberen Luft- und Verdauungswegen erzielt worden. Hallopeau lobte und lobt noch heute das Hektin. Aber die ganze ärztliche Welt wandte sich — mit wenigen Ausnahmen — von

diesen Medikamenten ab, als Ehrlich mit seinem *Dioxydiamidoarsenobenzol* alle andern Arsenpräparate so zu sagen über den Haufen rannte, — derart, dass genügende vielseitige Erfahrungen mit andern arsenhaltigen Mitteln speziell bei den Hals-, Nasen- und Ohrenkrankheiten meines Wissens gar nicht vorliegen und gar nicht zu erörtern sind. Die *Kakodylsäure* (Dimethylarsensäure) und ihre Salze eignen sich vielleicht zur Nachbehandlung, aber wohl kaum zur direkten Kur der Syphilis. Dasselbe gilt von stärkeren arsenhaltigen Mineralwässern, wie der Dürkheimer Maxquelle, die in manchen Fällen zur Nachbehandlung wohl angezeigt sein dürften. Aber nicht wie die gepanzerte Minerva, mit einem Male fix und fertig dem Haupte des Zeus entsprungen, sondern bekanntlich als Früchte jahrelanger, ebenso genialer wie mühevoller chemotherapeutischer Versuche entstand — an Stelle des fünfwertigen — das dreiwertige Arsenpräparat mit seinen 31,5 Prozent Arsengehalt, das *Salvarsan* im Ehrlich'schen Laboratorium und späterhin das *Neosalvarsan* mit 21 Prozent. Was dem Salvarsan von vorneherein die Ueberlegenheit über alle früheren Arsenmittel gab, das war das von Ehrlich angestrebte und erreichte ganz *andere Verhältnis zwischen therapeutischer und toxischer Wirkung*. Hata hat dieses Verhältnis (Dosis curata : Dosis tolerata $= c : t$) der einzelnen Arsenmedikamente bei der Hühnerspirillose wie folgt angegeben :

Atoxyl : $c/t = \frac{1}{2}$;
Arsacetin : $c/t = \frac{1}{3,3}$;
Arsenophenylglycin : $c/t = \frac{1}{3,3}$;
Dioxydiamidoarsenobenzol : $c/t = \frac{1}{58}$;

d. h. also, während beim Atoxyl schon jenseits des Doppelten der Heildosis die Giftwirkung beginnt, geschieht das beim Salvarsan erst jenseits des 58 fachen. Bei der Kaninchensyphilis ist das Verhältnis $\frac{1}{10}$–$\frac{1}{7}$. Nun kann von der Kaninchensyphilis nicht ohne weiteres auf die menschliche Syphilis, vom Laboratorium nicht auf die Klinik geschlossen werden. Hier hat die klinische Erprobung und Erfahrung das letzte Wort zu sprechen ; und diese Feuerprobe hat auch das Salvarsan bestehen müssen, besteht es jetzt noch und wird es noch weiterhin zu bestehen haben, wie es bei einer Krankheit von der Natur der Syphilis nicht anders sein kann.

Nun, Ihnen brauche ich nicht das Aufsehen ins Gedächtnis zurückzurufen, das die verblüffenden Erfolge des Mittels von Anfang an gemacht haben, nicht auch die Dämpfung des Enthusiasmus, als die Sterilisatio magna mit einem Schlage nicht gelang und die Ernüchterung, als Misserfolge und die anscheinend durch das Mittel gesetzten Schädigungen bekannt wurden, — Erscheinungen, wie sie beim Beschreiten jedes neuen therapeutischen Weges unausbleiblich sind, und doppelt, wenn der Weg ein so ganz neuer und unerhört kühner ist. Jetzt, nachdem die Salvarsantherapie ihre schlimmsten Kinderkrankheiten hinter sich hat, nachdem richtige Dosierung und beste Anwendungsart, Indikation und Kontraindikation festgestellt, wissen wir, dass die meisten schädlichen Neben-

wirkungen Fehler in der Anwendung waren, die zu vermeiden jetzt in unserer Hand liegt, oder Erscheinungen der Syphilis selbst, auf die früher nicht in dem Masse wie jetzt geachtet worden ist. Und schliesslich haben wir erkennen gelernt, dass, wenn gewisse Kategorien von Syphiliskranken dem Salvarsan widerstehen, — wie andere dem Hg widerstanden haben, — wir in Zukunft wohl am erfolgreichsten und sichersten zugleich vorgehen werden, wenn wir die zwei klassischen Luesmittel *kombinieren* und somit von vorneherein die Quecksilber- wie die arsenfesten Spirochaetenstämme zu treffen versuchen.

Noch kann das letzte Wort über das, was Salvarsan und Neosalvarsan für die völlige *Ausheilung der Syphilis* zu leisten vermögen, nicht ausgesprochen werden. Das ist auch gar nicht meine Aufgabe. Ueber das aber, was die Ehrlich'schen Mittel bei den spezifischen und gewissen nicht spezifischen Erkrankungen des Halses, der Nase und des Ohres leisten, über das sind wir — und *gerade wir* — in der Lage, schon heute ein *abschliessendes* Urteil abgeben zu können.

I

Syphilis des Halses und der Nase

M. H. Schon in den ersten grossen allgemeinen Mitteilungen über die Salvarsanbehandlung der Syphilis, — von Schreiber, Wechselmann [347-9], Alt, Iversen, Weintraud u. a. — fielen die hervorragenden guten Resultate gerade bei den *Schleimhautaffektionen der Mundhöhle* besonders ins Auge. Diese wurden nach dem Vortrag von Ehrlich auf dem Königsberger Kongress 1910 auch von Neisser, Lesser, Unna, Spiethoff, weiterhin von Bingel [54], Ballenger [11], Treupel und Levi [333], Kopp [230] u. a., dann auch in den Mitteilungen vieler deutschen Spezialgesellschaften nicht nur, sondern auch in der französischen Gesellschaft für Dermatologie von Brocq und le Blaye, Bizard [56], Darier et Cottenot [88], Hudelo u. Thibaut [208] und Renault (bei Félix [127]) voll bestätigt und seither ist kaum eine grössere Publikation über die Salvarsantherapie erschienen, in der nicht derartige Fälle, — vor allem natürlich die Wirkungen auf die Angina syphilitica, auf die Schleimhautpapeln der Mund- und Rachenhöhle mitgeteilt worden sind. Sehr bald erfolgten denn auch Mitteilungen aus den Spezialkliniken, und es ist heutzutage ebenso unmöglich wie unnötig, diese gesamte Kasuistik hier aufzuführen. Was mir zugängig war, findet sich in den Literaturangaben aufgeführt. Unsere spezielle Fachliteratur brachte zunächst die mündlichen Auslassungen in der Berliner Laryngol. Gesellschaft im Oktober 1910 von Halle [187], Scheier, Finder, Heymann und Kuttner, die sämtlich über ausgezeichnete Resultate berichteten, wogegen nur von Echtermeyer und Lennhoff einige Versager angeführt wurden, die späterhin auch bei grossem Material immer nur als *vereinzelt* aufgeführt wurden. Heymann konnte gleich über 40 Fälle berichten, die bis dato ohne Recidive geblieben waren. Von Publikationen erschienen zunächst die des

Referenten[154], die von Avellis[9], Safranek[302] aus der Onodi'schen Klinik, von Herzfeld[199], Chiari[80] und anderen. Grössere Beobachtungsreihen brachten dann noch Gurwitsch[176], Fabry[112–15], Bilancioni[51], Mangioli[248], Gereda[155–60], Kanasugi[220], Nakano[272], und Imhofer[213] und Félix[127] fassten die bisherigen Erfahrungen in guten Sammelreferaten zusammen.

1. *Mundrachenhöhle*

Unter den Mitteilungen über die Erfolge bei Mundrachenhöhlen-Affektionen, die im Ganzen natürlich weitaus am zahlreichsten sind, figuriert auch eine ganze Menge von *Primäraffekten* und legt — ganz nebenher — Zeugnis von der noch immer nicht genügend beachteten Häufigkeit dieser Lokalisationen ab. Wir werden später sehen, dass sie in der Salvarsanliteratur eine ganz besondere Rolle spielen. So sind — um nur einige Beispiele herauszugreifen — Schanker der *Lippen* von Fabry und Jerzycki[116], Safranek[302], Parker, Wechselmann[348], Stern[317], Freund[13a], und Werther[355], der *Zunge* von Schmidt, Wechselmann[348], Swiatkiewitz[327], und dem Referenten (Henke[195a]), der *Tonsille* von Werther[355], Arning[4], Weiler, Braun, Avellis[9], Munch[267], Sack[299], dem Ref.[154] u. a. mit Salvarsan sowohl wie mit Neosalvarsan geheilt worden. Die Heilungsdauer betrug 3–10 Tage, die bei diesen Sklerosen bisweilen so erheblichen Beschwerden wichen oft schon nach Stunden, bei Avellis nach einem halben Tage. Munch hatte noch 2 Monate, Stern und Freund noch 5 Monate nach der Behandlung keine Sekundärerscheinungen, ebenso Werther, der auch negativen Wassermann erzielte. In verschiedenster Hinsicht bemerkenswert ist unser Fall von Primäraffekt der Zungentonsille bei einer 60 jährigen Frau, die sich von ihrem hereditär-luetischen Pflegekind infiziert hatte. Die Sklerose sass auf der rechten Hälfte der Zungenbasis, schmieriger Belag auf induriertem Grunde, die Umgebung inclusive der Epiglottis ödematös geschwollen. Im Abstrich typische Pallidae unter zahlreichen Mundspirochaeten. Wassermann positiv. Die Patientin äusserst elend und herabgekommen. Wegen des Alters und des Allgemeinzustandes erhielt Patientin nur 0,3 Neosalvarsan, trotzdem war das Ulcus nach 8 Tagen fast verschwunden. Am neunten Tage 0,5 Neosalvarsan, nach 3 Wochen nochmals 0,5 Neosalvarsan. Als Patientin sich dann zur Kontrolle vorstellte, war nicht nur alles verschwunden, sondern wir hatten eine blühend gesund aussehende Frau vor uns mit offenbar sehr erheblicher Gewichtszunahme. Wassermann +.

In wie vielen von diesen Fällen, — und ob überhaupt mit der Heilung dieser extragenitalen Primäraffekte eine wirkliche *Abortivkur* gelungen war, — lässt sich bei dieser Art des Krankenmaterials schwer kontrollieren. Tatsache ist, dass viele Autoren hier ebenso wie bei den gewöhnlichen Genitalsklerosen noch nach Monaten keine Sekundärerscheinungen konstatieren konnten, so Fabry und Jerzycki[116] (Werther sah bei einer Sklerose der Lippe nach Monaten eine Reerosion ohne Sekundärerscheinungen). In dieser Hinsicht werden sich die extragenitalen Primäraffekte

wohl nicht anders verhalten wie die genitalen, bei denen Gennerich, Géronne, Gutmann u. a. eine Abortivkur ja als durchaus möglich bezeichnen, während eine Kommission der Medizinischen Gesellschaft in Paris die Möglichkeit einer solchen zur Zeit bestreitet.

Wie natürlich und schon vorher bemerkt, betreffen die meisten Beobachtungen in der Mundrachenhöhle: *Sekundäre Efflorescenzen.* Auch ist das Studium der Salvarsanwirkung bei diesen am lehrreichsten, von klinischen Tatsachen wie den häufigen Recidiven abgesehen, schon wegen ihres Spirochaetenreichtums. Frische oder ältere Papeln auf den Tonsillen, der Lippen- und Wangenschleimhaut, der Zunge verschwinden durchschnittlich in 12 bis 36 Stunden — am raschesten nach intravenöser Injektion von durchschnittlich 0,5 bis 0,6 Salvarsan oder den entsprechenden Dosen von Neosalvarsan. Ja, wir haben das auch schon nach 0,2 Salvarsan beobachtet. Hat man noch Zeit zur Beobachtung, so kann man in der Zwischenzeit eine Art von *Herxheimer'scher Reaktion* konstatieren, bestehend in starker Schwellung und Rötung am Rande, von woher dann auch die Reinigung und Epithelisierung erfolgt, besonders am Isthmus faucium. Meistens zeigt sich schon am nächsten Tage eine Reinigung und Abstossung der Papeln, oft sind sie bis auf etwas Rötung schon jetzt verschwunden. Hals- und Schluckschmerzen, ebenso etwa bestehende Begleiterscheinungen, besonders Kopfschmerzen sind meist auch am nächsten Tage verschwunden oder mindestens gebessert, bisweilen schon nach einigen Stunden. Hand in Hand hiermit zeigt sich eine Verminderung, ein Absterben und Verschwinden der Spirochaeten im Dunkelfelde von einem zum andern Tage. Meine klinisch-mikroskopischen Untersuchungen decken sich hierin völlig mit den Laboratoriumresultaten von Salmon und Browne [302a]. Langsamer geht die Adenitis zurück. Wassermann haben wir mehrfach in 9 bis 14 Tagen negativ werden sehen; viele Fälle haben wir allerdings auch nach 4 bis 6 Wochen zunächst noch mit positivem Wassermann entlassen müssen. Die Umkehrung eines anfangs negativen Wassermann nach der ersten Injektion wurde wiederholt beobachtet. Von den wenigen Patienten, die sich später wieder vorgestellt haben, waren die meisten recidivfrei: einer nach 6, einer nach 8, einer nach 9 Monaten.

Sehr wichtig für den Wert des Salvarsans sind natürlich jene Fälle von '*recidivierenden Plaques*', die schon vielfach und ohne Erfolg zuvor behandelt worden sind. So hatten wir einen Patienten, der 30 Sublimatinjektionen ohne jeden Erfolg bekommen hatte und bei dem die Papeln nach einer einzigen Injektion von 0,5 nach 9 Tagen völlig geschwunden waren. Ein anderer Patient zeigte Tonsillenpapeln nach 2 Injektionskuren und einer Schmierkur und wurde sie schon nach einer einmaligen Injektion von 0,6 Salvarsan los. Diesen Beobachtungen analoge finden sich massenhaft in der Literatur, wie sie mir z. B. von Avellis [9], Fallas [120], Llubes [245] und Schnarbachowski [303] vorliegen. Der Fall von Avellis war seit 6½ Jahren vergeblich mit Quecksilber und Jod behandelt worden und heilte nach 0,8 Salvarsan in 3 Wochen! Friedländer (bei Avellis)

sah ein junges Mädchen, das gegen ihre Schluckbeschwerden vergeblich Schmier- und Spritzkuren, Jod und Zittmann gebraucht hatte und nach 24 Stunden schlucken konnte!

Solche verblüffende Erfolge bei vorher nutzlos behandelten Fällen zeigen sich nun umsomehr, je mehr wir uns den eigentlichen Spätformen zuwenden, wie das schon früher von Iversen betont worden ist. So bekam ich eine 27 jährige Patientin mit einem grossen, schmierig belegten Defekt der hintern Rachenwand in Behandlung, die 1904, 1906 und 1909 Injektionskuren ohne Erfolg gebraucht hatte. Am 9. Nov. 1910 0,6 Salvarsan. *Schon 5 Stunden nach der Einspritzung* gibt sie spontan an, dass die Schluckbeschwerden, die sie jahrelang gequält, völlig geschwunden seien. 14. Nov.: Füllung des Defektes mit gesunden Granulationen. 29. Nov.: Geheilt entlassen. — In einem andern Falle war ein Ulcus des Epipharynx in 6 Tagen geheilt, eines der hintern Rachenwand mit Uebergreifen auf den Aryknorpel in 11 Tagen gereinigt, bei einem hereditär-luetischen, vom Rachendach bis zur Epiglottis reichenden Geschwür wurde mit 0,5 Reinigung in 4 Tagen erzielt. Ebenso erzielten Iversen, Safranek, Chiari, Zieler u. a. bei den *Ulcera des Pharynx* Heilungen in durchschnittlich 4 bis 10 Tagen. Ebenso gingen die eigentlichen Gummen in neoplastischer Form zurück, so bei M. v. Zeissl [365], Kahler [219], Piolti [285] u. a. ein Gumma des Segels nach 0,5 Salvarsan, bei Fabry [112] 7 Fälle in 4 bis 10 Tagen, bis auf einen mit Perforation des Segels, die nach 10 Tagen sich zurückbildete und nach 6 Wochen nicht mehr bemerkbar war. Wechselmann [347–52], Fabry und Kretzmer [117] haben eine ganze Reihe schwerster tertiärer Rachen-, Nasen- und Kehlkopffälle mit schönsten Erfolgen behandelt. Cherzelitzer heilte eine Segelperforation in 5 Tagen, eine andere in 6 Wochen mit 2 Injektionen.

Ueber weitere Fälle *maligner Syphilis* mit destruktiven Prozessen am Segel, Zäpfchen und Isthmus berichten unter andern Gurwitsch [170], bei dem 3 Fälle nach Ablauf von 7 Tagen vernarbten, Réthi, der zwei Fälle mit Segelperforation und tiefen Rachengeschwüren (neben andern schweren Erscheinungen) behandelte, die bisher allen merkuriellen Behandlungen widerstanden hatten und nach Salvarsan vernarbten. Recidive nach 2 und 3 Wochen wichen wiederum der Injektion. Sack [301] sah eine frische Perforation des Gaumens nebst andern Affektionen im Pharynx und an den Choanen bei gleichzeitiger Nephritis und schwerer Allgemeininfektion nach 2 Injektionen von 0,4 und 0,3 vernarben, die Nephritis zurückgehen und eine Gewichtszunahme von 15 Pfund in 14 Tagen. Die unerträglichen Schluckschmerzen schwanden schon 2 Stunden nach der Injektion! Leredde [241] heilte ein Gumma des Segels mit hochgradiger Zerstörung, nach erfolgter Hg Behandlung mit 606.

Auch die Affektionen der Zunge (Glossitis gummosa-ulcerosa) leisten dem Salvarsan nicht mehr Widerstand wie die andern Teile der Mundrachenhöhle (Fabry [112-15], Bayet [14], Hudelo u. Thibaut; Darier u. Cottenot [88], Dind [92], u. a.) Mikley [256] sah einen Fall schwerster ulceröser Glossitis, der nur gerade flüssige Nahrung nehmen konnte, am Tage

nach der Injektion feste Nahrung kauen und nach 24 Stunden völlig geheilt.

Ehe wir uns den andern Organen zuwenden, sei auch noch der teilweise sehr guten Erfahrungen bei der so überaus hartnäckigen Leukoplakie gedacht, wie sie Rasch [290], Milian [260] u. a. mitteilen. Letzterer sah eine solche, die 7 Jahre lang der Merkurbehandlung getrotzt, nach 0,5 Salvarsan in 12 Tagen kleiner werden.

2. *Nase*

In der Nase sind Primäraffekte sehr selten. Immerhin sind auch solche schon, wie im Falle von Fahrguahrson [122], Loaec et Rollet (b. Félix [127]) mit Salvarsan erfolgreich behandelt worden. Hauptsächlich haben wir es bekanntlich hier mit spät-sekundären oder *tertiären* Affektionen zu tun, seien es nun ulceröse Schleimhautprozesse oder osteoperiostitische des Nasengerüstes. So behandelten wir mehrere Patienten mit *Septumulcerationen* — meist mit Pharynxaffektionen zusammen, die durchschnittlich ebenso rasch wie diese letzteren heilten. Ein Septumulcus verschwand bei einer 13 Jahre bestehenden Lues nach 0,5 Salvarsan in 8 Tagen, ein anderes kombiniert mit perforierendem periostitischen Prozess am Nasenboden, der nach der Raphe des harten Gaumens durchgebrochen war, nach im Ganzen 2,3 Neosalvarsan mit Hg kombiniert in etwa 4 Wochen. Kontrolle nach weiteren 4 Wochen zeigt völlige Abheilung. Ein Gumma des Septums heilt nach 3 mal 0,6 Neosalvarsan in 9 Tagen, ein Ulcus im Bereich der mittleren Muschel nach 5 Tagen. Pat. erhält im Ganzen 2,6 Neosalvarsan.

Entsprechende Erfahrungen bei der Nasenlues teilten schon Wechselmann [348], dann Safranek [302] mit, der ein Ulcus septi in 7 Tagen, eine Periostitis gummosa ossis nasal sin. in 10 Tagen abheilen sah, ein luetisches Geschwür des Epipharynx in 12 Tagen. Bei diesen Fällen war die Herxheimer'sche Reaktion auch von ziemlich starken subjektiven Symptomen begleitet. Bilancioni [51] hatte bei 9 Fällen tertiärer Nasenlues ausgezeichnete Resultate, ebenso Gurwitsch [176], Biehl, v. Zeissl [365], E. Hoffmann [205], A. v. Sokolowski [314], Borden [58], Fabry [112], Hecht [195] und viele andere, bei congenitaler ebenso wie aquirierter Lues. Costiniu [86] brachte ein kolossales die Nase obstruierendes Gumma durch 1,0 Salvarsan zur Heilung. Bei Hoffmann vernarbte ein ausgedehntes tertiäres ulceröses Syphilid des Gesichts, ein *tuberoserpiginöses* der Nase, Wange, ganzen Ober- und Unterlippe und der Umgebung nach 0,36 in 11 Tagen; bei Stroscher [321] eine maligne Lues mit ausgedehnten, die ganze Nasenhöhle und hintere Rachenwand überziehenden Ulcerationen, in äusserst desolatem anämischem Zustand mit Hg, Jod und Joha vergeblich behandelt, mit Neosalvarsan in 2 Tagen. Hecht heilte einen ausserordentlich schweren Fall in 10 Tagen. Hervorgehoben wird mehrfach auch der gute Einfluss auf die postluetische (oder sogar die 'genuine') *Ozaena*, — so von Ledermann [240], Beck [27], Safranek [302], Hoffmann, Hamel [190], Aisenberg,

Touton [332], Kannengiesser [221]. Hiervon konnte ich selbst mich bisher nicht recht überzeugen, wenn es auch klar ist, dass nach Abstossen eines Sequesters, wie im Falle von Port [287], die den nekrotischen Prozess als solchen begleitende symptomatische 'Ozaena' schwinden kann. Bemerkenswert ist der durch Hg und J unbeeinflusste, nach 0,5 Salvarsan geheilte Fall von *Syphiloma hypertrophicum diffusum* der Nase, Ober-, Unterlippe und Zunge, den Bindermann [49a] mitteilt.

Geradezu als Reklamefälle für die Salvarsanerfolge bei der Nasenlues können die von Heuck und Jaffé und von Sack mitgeteilten Beobachtungen gelten. Die ersteren berichten aus der Hautklinik in Bonn über einen Fall von Lues congenita tarda mit völliger Zerstörung des Naseninnern bis zur Schädelbasis, die bereits aufgegeben und 2 Tage nach der Injektion gerettet war. Sack [301] teilt den Fall eines 16jährigen, lange Zeit vergeblich mit Hg und J behandelten Mädchens mit, die einen 'geradezu grauenerregenden Anblick' darbot. 'Von der eigentlichen Nase keine Spur. Nur die zackigen retrahierten Lappen am Rand der gähnend blosgelegten Apertura pyriformis zeigen noch die Reste der fleischigen Nase an. Bis auf die Nasenwurzelknochen war das ganze knorpelige und knöcherne Skelett der Nase einschliesslich des ganzen Vomers, eines Teils des Ethmoidalsinus, etc. . . . zerstört.' Hier nun bildete sich nach einer Gesamtdosis von im Ganzen 1,7 Salvarsan innerhalb eines Monats der ganze Prozess zurück. 'Schon nach 8 Tagen hat der nekrotische Zerfall gänzlich aufgehört und die Natur vollzog nun unter der eminent inzitierenden Regenerationstätigkeit des Arsenobenzols innerhalb einiger Wochen eine Plastik der Nase, wie sie einem geschickten Chirurgen wohl kaum besser gelungen wäre.' Hätte S. die Patientin nicht vor und nach der Behandlung vorgestellt, so könnte man, wie er selbst sagt, den Bericht bezweifeln, der uns aber auch durch die beigegebenen Bilder bestätigt wird.

Auch *Nebenhöhlenaffektionen* auf spezifischer Basis sind mit Salvarsan behandelt worden. Mangioli [249] sah in einem Falle von 'Sinusitis frontomaxillaris' keinen Erfolg, im andern, in dem der Abgang von Sequestern zu starken, das Leben gefährdenden Blutungen führte, tat das Ehrlich'sche Mittel Wunder, die meningitischen Symptome und eine Lähmung des M. rectus externus verschwanden und Pat. wurde geheilt. Sharp [309] erzielte eine sofortige Besserung bei einem 'Gumma des Antrum Highmori' mit Nekrose der untern Muschel, der nasalen Antrumwand etc. Aehnlich ist der von mir mitgeteilte Fall mit Fisteln des rechten oberen Alveolarfortsatzes und nekrotischem Prozess in der rechten Nasenhöhle. In einem Falle Finders von Syphilis der Stirnhöhlen und des Siebbeins blieb Salvarsan ebenso erfolglos wie Hg, und J. Ehrler [101] beschreibt einen Fall aus der Thost'schen Abteilung mit Sattelnase, Optikusatrophie und Keilbeinaffektion. 0,6 Salvarsan. 4 Wochen danach Exitus unter meningitischen Erscheinungen. Sie warnen vor der Anwendung des Mittels bei Knochenprozessen des Schädels, da die starke Reaktion von Krankheitsherden in der Nähe lebenswichtiger Organe gefährlich werden

könne. Das kann gelegentlich vielleicht einmal zutreffen ; ob hier die Meningitis nicht auch ohne das Salvarsan eingesetzt hätte, ist aber recht zweifelhaft ; denn erstens war es ein von Anfang an besonders schwerer, teils vernachlässigter, teils falsch behandelter Fall, ausserdem hatte er eine Keilbeinaffektion ! Dass übrigens nicht alle Knochenprozesse in der Nachbarschaft der Nebenhöhlen von Syphilitikern eigentliche Nebenhöhlenaffektionen sind, darauf habe ich schon früher hingewiesen. So haben wir auch jüngst wieder einen Patienten mit typischer Lorgnettenase gehabt, der mit Auftreibung und Druckpunkt am rechten medialen Augenbrauenrande zu uns kam, bei dem aber weder rhinoskopisch noch röntgenologisch eine Stirnhöhlenaffektion festgestellt werden konnte. Diagnose : *Osteo-Periostitis spezifica ossis frontis.* Da Patient vor langen Jahren und ganz ungenügend behandelt war, wurde hier erst eine vorbereitende Hg- und J-Kur gegeben und dann 2,8 Neosalvarsan im Ganzen. Heilung.

3. *Kehlkopf, Trachea und Bronchien*

Sind die spezifischen Erkrankungen des *Kehlkopfs, der Luftröhre und Bronchien* auch seltener wie die der Mundrachenhöhle und der Nase, so können sie um so gefährlicher werden und erfordern oft um so mehr *rasche* und radikale Hilfe. Dass das Salvarsan die *sekundären* Kehlkopfaffektionen nicht minder beeinflussen würde wie die der andern Organe, war anzunehmen, und solche Beobachtungen wurden denn auch zahlreich gemacht, meist da, wo es sich neben Mundrachenaffektionen um Erythem, spezifische Katarrhe und um die selteneren Papeln handelte. So sah ich eine spezifische Laryngitis sogar schon nach 0,2 Salvarsan in 10 Tagen schwinden, typische Papeln an den Stimmbändern nach 0,6 in 8 Tagen — *allerdings sehr viel später wie die gleichzeitigen Rachenpapeln,* die schon am Tage nach der Injektion kaum noch zu sehen waren. Auch bei Safranek [302] scheinen die *Larynxerytheme* etwas zögernder zu heilen als die sekundären Rachenprozesse. Sehr viel zahlreichere Mitteilungen liegen natürlich über die *tertiären Kehlkopfprozesse* vor. Glas [167] sah Ulcerationen der Epiglottis und der Stimmbänder, die allen andern Methoden getrotzt hatten, nach *einer* Injektion in wenigen Tagen heilen, ebenso v. Sokolowski [314], Biehl, Ledermann [240], Bilancioni [51], Safranek [302] und der Ref. Gummata des Stimmbandes, Ulcerationen der Epiglottis, Sack [300] ulcerierte Gummata in einem Monat. Seine Patientin bekam 5 Wochen später ein Recidiv, das durch eine kombinierte Behandlung in 14 Tagen heilte. Interessant und relativ zahlreich sind gerade die Larynxfälle bei Chiari [80] : Von 7 Fällen wurden 5 geheilt, mit Ausnahme einer Ringknorpelnekrose, die nicht zurückging. In den restierenden beiden Fällen handelte es sich um alte Infiltrate und Wucherungen, die nur teilweise zurückgingen, *aber doch in viel höherem Masse als durch alle andern Behandlungsarten.*

Damit kommen wir zu dem therapeutisch schwierigsten Kapitel der

Kehlkopfsyphilis, den *stenosierenden Prozessen*, unter denen wir zwei verschiedene Kategorien zu unterscheiden haben. Es können sowohl frische entzündliche, gummöse und perichondritische Prozesse die Glottis verlegen, als auch die aus solchen Affektionen hervorgegangenen Narbenstenosen, also eigentlich syphilitische und metasyphilitische. Beiden gegenüber haben wir bisher einen schweren Stand gehabt, denn bei den ersteren war Hg meist von zu langsamer Wirkung und das Jod wegen seiner auflockernden und Oedemwirkung gefürchtet, von vielen geradezu perhorresziert. Bei den letzteren aber versprach man sich von der spezifischen Behandlung überhaupt nichts mehr und wandte sich der chirurgischen zu — oft ohne jeden Erfolg. A priori nun konnte man sich auch vom Salvarsan bei spezifischer Kehlkopfstenose nicht gerade viel versprechen. Denn bei der ersten Kategorie der Stenosenfälle konnte ja — ähnlich wie das Jodödem — die Herxheimer'sche Reaktion für die Glottis geradezu verhängnisvoll werden, wie das auch von Roth, Kahler u. a. direkt ausgesprochen worden ist, und bei den Narbenstenosen, bei denen doch ein spirochaetenloses Bindegewebe vorauszusetzen war, war auf eine *erweiternde* Wirkung des Mittels gewiss nicht zu rechnen.

Nun, m. H., es ist anders gekommen! Gerade auf diesem verlornen Posten, wenn ich mich so ausdrücken darf — hat sich das Ehrlich'sche Mittel in seiner ganzen therapeutischen Macht gezeigt, gerade hier hat es ganz ungeahnte, zauberhafte Wirkungen hervorgebracht!

So beobachteten wir eine 27 jährige, vor 3 Jahren infizierte Frau, seit 2 Monaten gravid und angeblich eben so lange heiser. In der letzten Woche zunehmende Atemnot und hochgradige Dysphagie, so dass nur flüssige Nahrung mit Mühe genommen werden konnte. Pat. hat die ganze Zeit hindurch bis jetzt Jod in hohen Dosen genommen; vorher mit Hg behandelt. Die ganze hintere Rachenwand in ein grosses, schmierig belegtes, bis in den Hypopharynx hineinreichendes Ulcus verwandelt. An der Zungenbasis markstückgrosses Geschwür. Epiglottis stark verdickt, gerötet, ulceriert, rechte aryepiglottische Falte, Aryknorpel und Taschenband von grossem, in oberflächlichem Zerfall begriffenen gummösen Geschwür eingenommen; starke Infiltration der ganzen Umgebung, erhebliche Verengerung des Kehlkopflumens. Linke Larynxhälfte infiltriert, nicht ulceriert. Wassermann +. Zahlreiche Mundspirochaeten, keine Pallidae. Nach Injektion von 0,45 Salvarsan intragluteal schon am nächsten Tage Atemnot und Schluckschmerz geringer; beginnende Reinigung des Geschwürs und Abschwellung. Keine Herxheimer'sche Reaktion. Völlige Heilung nach 10 Tagen. Kontrolle nach 4 Monaten: Kein Recidiv. Wassermann +. Bei einem Patienten in den 30 ger Jahren, der etwa 14 Tage hindurch wegen Atemnot nicht geschlafen hatte, bei dem die Glottis einen kaum noch sichtbaren Spalt zeigte und bei dem wir — ebenso wie im ersten Falle — schon die Tracheotomie vorbereitet hatten, stellte sich nach 1,3 Neosalvarsan in 2 Dosen wieder völlig normale Atmung ein.

Diesen ganz typischen Fällen ähnliche Beobachtungen liegen noch

zahlreich vor, von Chiari [80], Herzfeld, Scheier, der auf diese Weise die Tracheotomie vermied, Bilancioni [51], Alexander [2] (Frankfurt), Poyet [288], Piolti [285], Gereda [156], Bingel [54], Schmidt, Werther [355] u. a. — Chiari [80] hatte unter anderem einen ausgezeichneten Effekt bei einer Stenose durch starke subglottische Infiltrate — die allerdings schon vorher tracheotomiert war, Bilancioni [51] bei einem ulcerierenden Gumma, das zu Erstickungsanfällen geführt hatte, in 2–3 Tagen. Bingel [54] erzielte Verschwinden der Stenosenerscheinungen in wenigen Tagen, wodurch er die drohende Tracheotomie vermied, ein normales Kehlkopfbild in 8, Huguet [209–10] bei Adduktionsfixation des (ulcerierten) Stimm- und Taschenbandes völlige Heilung in 3 Wochen, Schmerzlosigkeit nach 4 Tagen. Darier und Cottenot [88] beseitigten eine Stenose, die vorher vergeblich, teils mit Merkur, teils operativ behandelt worden war. Ob in dem Falle von Huguet mit Erstickungsanfällen, bei dem *nach* der Injektion wegen eines erneuten Erstickungsanfalles die Tracheotomie gemacht werden musste — eine Herxheimer'sche Reaktion vorlag, ist nicht klar. Uebrigens konnte die Kanüle hier nach 3 Tagen entfernt werden und nach 8 Tagen trat Heilung ein. Aehnlich war es in einem Fall von Fabry und Kretzmer. Chiari konstatierte die Herxheimer'sche Reaktion ' in einem Falle am 11. Tage sehr stark, in einem andern am 3. deutlich, in einem weiteren von 1–3 Tage mässig '. Von einer direkten Verschlimmerung einer gummösen Larynxstenose durch Salvarsan berichtet nur Ehrler [101] (Thost).

Dass wir nun in solchen Fällen mit gummösen und spezifischen Granulationsprozessen des Larynx, in denen Quecksilber und Jod so oft zu spät, so oft gar nicht wirksam waren, am Salvarsan einen fast immer zuverlässigen Helfer gefunden, ist — wie dankenswert immer auch — so doch noch erklärlich und nach seinen günstigen Leistungen nicht so wunderbar. Das Wunderbarste aber bleibt seine jetzt unbestreitbare Wirkung bei *alten Schrumpfungsprozessen und narbigen* Stenosen. Ich hatte schon frühe Gelegenheit, einen derartigen Fall zu beobachten, bei dem ich das Salvarsan eigentlich ganz ohne Hoffnung anwendete und dessen Ausgang mich in das höchste Erstaunen versetzte.

Die 63 jährige Frau weiss nichts von einer luetischen Infektion. Ihre mehrjährige Atemnot datiert sie von dem Steckenbleiben eines Knochens. Der laryngoskopische Befund 1908 zeigt eine schon im Aditus beginnende Stenose durch starres, gewulstetes, anscheinend sehr derbes Gewebe, das sich nach unten trichterförmig verschmälert und eine kaum erbsengrosse Oeffnung über der Glottis frei lässt. Nichts von Ulcerationen. 1907, 1909, 1910 Jodkali und Injektionskuren. Beschwerden bald geringer, bald stärker. 1910 Dehnungen mit Schrötter'schen Kathetern und O'Dwyer'schen Tuben. Zunächst Besserung, dann vermehrte Beschwerden. Patientin wird wegen wahrscheinlich notwendig werdender Tracheotomie in die Klinik aufgenommen. An der laryngealen Epiglottisfläche jetzt ein — erst nach der mechanischen Behandlung, und wohl durch diese entstandenes Geschwür. Dysphagie und Dyspnoe.

Im Abstrich des Geschwürs nur Mundspirochaeten, keine Pallidae. 15. XII, 1910: 0,6 Salvarsan intragluteal. 17. XII: Epiglottis abgeschwollen, Glottis etwas weiter. 19. XII: Völlig normale Atmung. Laryngoskopisch: *Der ganze Narbentrichter ist verschwunden, Glottis normal weit, ungehinderter* Einblick in die Trachea. 24. XII: Patientin wird mit *normalem Kehlkopf* nach Hause entlassen. Letzte Kontrolle Februar 1913: Kehlkopf normal, die früher elende Frau sieht blühend gesund aus und ist unglaublich stark geworden. Aehnlich sind wohl auch die Fälle von Alexander [2] (Frankfurt), von Gereda [156] (Madrid), von Casati [75] u. a. gewesen. Letzterer sagt, *er sei zu dem Grundsatz gekommen, Syphilitiker nicht zu tracheotomieren, ohne vorher eine Salvarsankur anzuwenden.* Und auch Poyet [288] meint, dass die Tracheotomie durch das Salvarsan in vielen Fällen vermeidbar geworden sei.

Der Wirkung auf die Kehlkopfaffektionen entspricht die auf die Prozesse der *Trachea* und *Bronchien*. Ein 16 jähriger Patient sucht wegen Luftbeschwerden, die seit 1 Jahr bestehen und sich letzthin gesteigert haben, die Poliklinik auf. Anamnese negativ. Sprache rein aber nicht kräftig, deutlicher, besonders inspiratorischer Stridor. Kehlkopf normal. Etwa 3 cm unterhalb der Glottis von rechts und hinten sich vorwölbender, derber fester Tumor von glatter Oberfläche. — In den andern Organen nichts pathologisches. Wassermann +. Drei Wochen hindurch Jodkali und Sublimatinjektionen. Keine Besserung. 16. Febr. 1912 0,6 Salvarsan und 28. März 0,6 Neosalvarsan. Luftbeschwerden ganz beseitigt. An der Stelle des Gummas leicht ovale Vorwölbung. Wassermann negativ. Halle [187] befreite eine Frau von schweren asthmatischen Beschwerden, veranlasst durch Gummata, die die ganze Trachea einnahmen und die 4 Schmierkuren und Jodkali ohne Erfolg gebraucht hatte, durch eine Injektion von 0,6 in 14 Tagen von ihrer Krankheit. Glas [168] sah Heilung bei tiefer Geschwürsbildung im Gebiet der Bifurkation, ohne Herxheimer'sche Reaktion; Jurasz [218] beseitigte eine Stenose des rechten Bronchus durch Granulationen mit 2 Injektionen und nachfolgender kombinierter Behandlung, Encina [111] sah deutliche Besserung einer Stenose am Eingang der Bronchien. Chiari [80] sah die Rückbildung gummöser Infiltrate der Trachea und des Unterlappenbronchus in kurzer Zeit in 2 Fällen. Gaston heilte ein Syphilom des Mediastinums mit Trachealkompression durch 0,5 Sa.

M. H. Aus diesem objektiv mitgeteilten Tatsachenmaterial werden wir entnehmen, dass die Ehrlich'schen Mittel bei der Behandlung der spezifischen Erkrankungen des Halses und der Nase geradezu glänzende Resultate gezeitigt haben. Gegenüber der jetzt schon nicht mehr übersehbaren ungeheuern Zahl dieser Resultate können die wenigen Versager nichts bedeuten und sehr wenig auch die ganz vereinzelt mitgeteilten schädlichen Wirkungen durch lokale oder entfernte Reaktion. Verchère [342a] sah bei einem 5 Wochen alten Schanker der Oberlippe nach einer Injektion von 40 cg, die ein Tag vor der Menstruation gemacht wurde, am Abend kolossale Blutungen; ähnlich wie Kannengiesser [221]. Von den

drei Ehrler'schen Fällen ist der eine schon früher kritisiert worden. Auch in dem zweiten handelt es sich augenscheinlich um eine so schwere hereditäre (?) Lues, dass ich das Hinzutreten einer *Keratitis* (!) gerade hier, zu Gummen des Schädels und der Arme, der Rachenwand und Periostitis der Nase kaum auf Rechnung des Salvarsans setzen würde. Dass die Herxheimer'sche Reaktion eine Larynxstenose verschlimmern *kann*, soll nicht geleugnet werden; tatsächlich scheint es doch recht selten vorzukommen. In dem Falle von Basch[13] ist, wie Ref. selbst auseinander setzte, der Tod nicht dem Salvarsan, sondern der im Leben nicht erkannten Tracheo-Oesophagealfistel zuzuschreiben, durch die es zu putrider Bronchitis und Lungengangräne kam. Dass ein Mittel von der therapeutischen Mächtigkeit des Salvarsans 'verschiedene Organsysteme toxisch anzusprechen *vermag*' (Stümpke und Brückmann) ist plausibel. Auf dieses im Allgemeinen einzugehen, ist nicht meine Aufgabe; auf die speziellen Erscheinungen am Acusticus komme ich im letzten Teil meines Referats zurück.

II

Lokale Spirochaetosen und andere Erkrankungen der oberen Luftwege

Es ist Ihnen, m. H., aus einer grossen Reihe von Publikationen bekannt, dass das Salvarsan und Neosalvarsan auch eine Zahl von nicht luetischen Erkrankungen günstig beeinflusst oder direkt heilt. Unter diesen Krankheiten haben wir — wenn wir nach einer Erklärung für diese Wirkung suchen — zwei Gruppen zu unterscheiden: 1) die durch *Spirochaeten* hervorgerufenen, 2) die durch andere (bekannte oder unbekannte) Erreger veranlassten. Dass ein Mittel, das die Spirochaeta pallida so mächtig beeinflusst, auch die andern Spirochaeten nicht gleichgültig lassen würde, das lag ja von vorneherein nahe. Als ich aber im November 1910 im Verein für wissenschaftliche Heilkunde in Königsberg über die Spirochaeten der *Mundrachenhöhle* und ihre Beeinflussung durch das Salvarsan die ersten Mitteilungen machte, erntete ich teils Verwunderung und Zweifel, teils direkten Widerspruch. Seither nun haben sich die bezüglichen Beobachtungen so gemehrt, dass schon eine ganze Literatur für sich hierüber vorliegt und diese Frage als entschieden betrachtet werden kann. Da diese Spirochaeten hauptsächlich die Mundrachenhöhle bevölkern, so haben gerade wir Halsärzte allen Anlass, uns mit ihnen und den durch sie veranlassten *lokalen Spirochaetosen* (Gerber) zu beschäftigen. Denn so werden wir diese Krankheiten am besten zusammenfassen, im Gegensatz zu den *generalisierten Spirochaetosen*, z. B. der Syphilis, der Recurrens. Allerdings sind Fälle von *Lungengangräne* und *Appendicitis*, die durch Spirochaeten veranlasst und mit 606 geheilt sind, mitgeteilt worden, deren Ausgangspunkt möglicherweise auch in den oberen Luft- und Verdauungswegen zu suchen ist. Bisher aber sind das nur ganz

vereinzelte Fälle. Als sicher oder höchstwahrscheinlich durch Spirochaeten in Gemeinschaft mit fusiformen Bazillen *bedingt* können wir jetzt wohl ansehen:

1. *Die Plaut-Vincent'sche Angina.*
2. *Die Gingivitis simplex.*
3. *Die Gingivitis mercurialis.*
4. *Die Stomatitis simplex.*
5. *Die Stomatitis mercurialis.*
6. *Gewisse Formen von Glossitis.*
7. *Manche skorbutische Geschwüre.*

Vielleicht auch gewisse Fälle von

8. *Noma.*

Ausserdem

9. *Periodontitische Abscesse, Pulpitis, Alveolarpyorrhoe* (Miller, Zilz).

Auf alle diese ist denn auch bereits die Wirkung des Ehrlich'schen Mittels erprobt worden. Unklarer aber ist die Wirkung auf eine Reihe anderer Prozesse, wie den *Pemphigus, Tuberculose* und *Lupus, Sklerom, Rotz* und *maligne Tumoren.* Erklärlich ist eine bessernde Wirkung bei diesen Prozessen nur da, wo sie zu *geschwürigem Zerfall* geführt haben, da wir jetzt wissen, dass sich auf den meisten Geschwüren der Mundrachenhöhle und des Kehlkopfs, welcher Provenienz auch immer, *sekundär* Spirochaeten ansiedeln, die die weitere Nekrose, Verjauchung, Gangränescenz und Phagädenismus herbeiführen oder unterstützen können und deren Beseitigung durch Salvarsan — auch ohne Heilung des Grundleidens — in der Tat objektive und subjektive Besserung herbeizuführen vermag. In den andern Fällen wird man eventuell auf die schon früher bewährte Wirkung des *Arsen* als solchen auf gewisse Ex- und Enantheme wie bisweilen auch Tumoren zurückgreifen müssen.

Die erste Beobachtung über die Beeinflussung von Mundspirochaeten durch Salvarsan überhaupt wurde 1910 von mir mitgeteilt.[379] Die erste Heilung einer Plaut-Vincent'schen Angina durch Salvarsan-Injektion stammt von Ehrlich selbst[375]. Ihr folgten sehr rasch die entsprechenden Beobachtungen von Rumpel[399], der 7 Fälle, teils in wenigen Tagen, heilte, und vom Ref.[380–1]. Späterhin die Fälle von Plaut[397], der sehr schwere Fall von Achard et Flandin[370], der allen andern Behandlungsmethoden getrotzt hatte und auf Salvarsan prompt heilte, und die Mitteilungen von Lubliner[393], Sourdel[402], und Roger[398], Schreiber (mit Neosalvarsan 0,15)[401], Gutmann[384], Citron u. a.

Dass diese ersten Beobachtungen sich ausschliesslich auf die Plaut-Vincent'sche Angina bezogen, ist erklärlich, da diese ja allein als Repräsentantin einer durch 'fuso-spirilläre Symbiose' hervorgerufenen Affektion auch weiteren ärztlichen Kreisen bekannt war. Wie manchen Forschern aber schon lange der Zusammenhang gewisser anderer Affektionen der Mundrachenhöhle, besonders des *Zahnfleisches* mit ulceromembranösen Erkrankungen der Tonsillen aufgefallen war, so konnten Beobachtungen über die Wirkung der Salvarsanpräparate auch auf

Gingivitis, Stomatitis u. a. nicht ausbleiben. Auch ich hatte nicht nur regelmässig bei diesen Erkrankungen Spirochaeten und Spiessbazillen, oft in Reinkultur gefunden, sondern auch ihre prompte Abheilung unter Salvarsan, sei es nun, dass sie mit Plaut-Vincent'scher Angina zusammen oder allein aufgetreten waren.[380–1] Dieselben Beobachtungen machten Plaut[397], der in Hamburg über die Heilung von zwei schweren Fällen mit *Stomatitis ulcero-membranosa* berichtete, Lenzmann [391] bei *' Stomacace '*, Stühmer [404] bei *' bullöser Stomatitis '*, le Blaye und Roger [398] bei merkurieller Stomatitis, Zilz [410–12] bei verschiedenen Formen ulceröser Stomatitis, ebenso Schreiber [401] mit Neosalvarsan und Roger [398], der in seiner interessanten Dissertation 8 neue Fälle von Spirochaetosen mit Salvarsan geheilt, mitteilt. Diese Autoren haben, ebenso wie ich, das Salvarsan bei leichten Fällen vielfach *lokal*, in Lösung oder Pulverform angewendet. Demgegenüber stehen nur einige Mitteilungen, wie die von Gutmann, die bei kombinierter Hg- und Neosalvarsanbehandlung mehr oder minder schwere Stomatitiden auftreten sahen — Beobachtungen, die also als nicht ' rein ' nicht recht zu verwerten sind. Auffallend ist es ja, dass in solchen Fällen das Salvarsan nicht die schädigende Wirkung des Hg koupiert hat. Bei chronischer, 12 Jahre bestehender *Glossitis* hatten Allport [371] und Roger [398], Joannides [389] und Laiguel-Lavastine [390] bei angeblich nicht luetischer *Leucoplasie* ausgezeichnete Erfolge. Eine ganz besonders interessante merkwürdige Beobachtung veröffentlichte Gerlach [383] aus der Richard Hoffmann'schen Abteilung in Dresden. Hier schloss sich an ein *Otit. extern. ulceromembranosa* eine Stomatitis, Glossitis und Angina ulceromembranacea. In allen Wundsekreten, auch im Ohreiter die typischen Spirochaeten und fusiformen Bazillen, Heilung nach zweimaliger, intravenöser Salvarsaninjektion. Hier, wie auch sonst, möchte ich annehmen, dass der primäre, wenn auch latente Herd am Zahnfleisch oder jedenfalls in Zahnnähe gesessen hat.

Zilz [409–12] hat sich in mehreren sehr eingehenden und vortrefflichen Arbeiten mit der Wirkung der Salvarsanpräparate nicht nur auf verschiedene Gingivo-Stomatiden, sondern speziell auch bei einer Reihe von eigentlichen Zahnaffektionen beschäftigt, von denen schon Miller seiner Zeit einige auf Rechnung der Spirochaetenwirkung zurückführen zu können glaubte, so bei *paradentalen Abcessen* (' Wunschheim-Abscessen ') und der *Pyorrhoea alveolaris*.

Auch die Dermatologen haben eine Reaktion der Mundspirochaeten bei Salvarsaninjektionen zugegeben. Nämlich, — was Neisser als ' Kuriosum ' anführt, starke Schmerzen an den Zähnen nach den Einspritzungen. Diese Beobachtung ist von E. Hoffmann, Kannengiesser [221] und von Zimmern [368] bestätigt worden, welch letzterer sie besonders bei Patienten mit Stomatitis mercurialis konstatiert hat und sie auf den Zerfall der Mundspirochaeten und das Freiwerden von Toxinen unter dem Einfluss des Salvarsans zurückführt. Auch Berger [45] erwähnt diese Erscheinung, erklärt sie aber für eine angioneurotische. Meine aprioristische Annahme, dass vielleicht auch die *Noma* eine Fuso-spirochaetose

sein könnte, ist inzwischen wenigstens an *einem* Falle von Nicoll [396] bestätigt worden. Er gab zwei Injektionen und erzielte 24 Stunden nach der ersten Injektion eine deutliche Lokalreaktion, dann Demarkation und Heilung. In einem zweiten, mit schwerer Scharlachnephritis kombinierten Falle trat allerdings Exitus ein. Auch ich selbst hatte in einem Falle, bei dem die Wahrscheinlichkeits-Diagnose Noma gestellt wurde, keinen Erfolg mit der Injektion. Allerdings zeigte die mikroskopische Untersuchung hier nicht den für eine typische Spirochaetose charakteristischen, unverkennbaren Befund. Auch hatte der Patient schon die Zeichen schwerster Allgemeininfektion. Mir selbst [381-2] überraschend war dagegen die rasche und vollkommene Heilung *skorbutischer Geschwüre* an Zahnfleisch und Mundschleimhaut bei zwei Matrosen. Diese Beobachtung ist durch Tuschinsky und Iwaschenzow [406] bestätigt worden, die in fünf Fällen von Skorbut eine deutliche Besserung der Lokalsymptome sahen.

Diese letzten Beobachtungen nun leiten schon zu der zweiten Kategorie von Fällen über, die wir nicht mehr als eigentliche Spirochaetosen zu betrachten haben, bei denen vielmehr die Spirochaeten und fusiformen Bazillen nur als sekundär hinzukommende Schmarotzer aufzufassen sind. Wie sehr solche im Allgemeinen aber den ursprünglichen Zustand vieler, sonst in sich bestimmter Affektionen zu verändern vermögen, wie manche objektiven Erscheinungen und subjektiven Symptome gerade durch sie bedingt sein können, ist ja bekannt. Davon sind auch die *spezifischen* Geschwüre nicht ausgenommen, bei denen zur Wirkung der Pallida die sekundäre der andern Mundspirochaeten hinzutreten kann, wie das an meinem Institut wiederholt beobachtet wurde (Henke[195a]).

Leider ist in vielen der hierher gehörigen Beobachtungen der mikroskopische Befund nicht angegeben worden. Wir werden aber wohl nicht fehl gehen, wenn wir annehmen, dass es sich in den meisten Fällen, wo wirklich Besserungen oder Heilungen eines Pemphigus, Reinigung und Besserung eines Zungenkrebses, eines Skleromgeschwürs konstatiert wurden, um sekundäre Infektion mit Spirochaeten handelt. Bei den Tumoren kommt natürlich die erweichende und nekrotisierende Wirkung des Salvarsans in Betracht.

Schreiber [401] sah günstige Wirkung des Neosalvarsans auf Streptotrix Angina. Ueber die rasche Heilung von Pemphigus, der auf Zunge und Wangenschleimhaut begonnen oder sich besonders lokalisiert hatte, berichten Wolff [408], Grouven [385] und Stümpke [405], über schnellen Rückgang der Erscheinungen bei einem wahrscheinlichen Fall von menschlicher Maul- und Klauenseuche Stühmer [404], bei Rotz Zilz [412], über erhebliche oder völlige Beseitigung der subjektiven Beschwerden bei Zungenkrebs Czerny u. Caan [374], Bodin [372].

Bekanntlich gibt man *Arsen* in verschiedenen Formen bei malignen Tumoren ebenso wie bei Exanthemen seit langer Zeit, und manche Beobachter haben über weitgehende Besserungen, einige sogar über

Heilungen, besonders beim *Sarkom*, berichtet. Ich selbst habe niemals eine Heilung eines wirklich bösartigen Tumors durch Arsen konstatieren können. Auch hier erweisen sich *Atoxyl* (Blumenthal, Fränkel, Czerny, u. a.), sowie *Salvarsan und Neosalvarsan*, sei es nun intratumoral, sei es subkutan und intravenös injiziert, weitaus am wirksamsten.

Zahlreich sind die Salvarsanversuche beim *Sklerom*, — sehr verständlich bei einer Krankheit, der gegenüber man in vorgeschrittenen Fällen so gut wie machtlos ist. Ich selbst habe nur einen Fall damit behandelt [382], der nach einer scheinbaren, vorübergehenden Besserung keine deutliche Reaktion zeigte. Allerdings war die Dosis klein und ich will ein abschliessendes Urteil danach nicht abgeben, wenn ich mir auch — nach dem oben gesagten — einen kurativen Einfluss a priori nur auf die *Zerfallsprodukte* denken könnte. So sind denn auch die Erfahrungen Nagys [395], Feins [376] und Liecks [392] teils negativ, ebenso die von Wieser [407]. Gerade die Beobachtungen Wiesers bestätigen meine Ansicht, denn bei seinen drei Fällen blieben die *Infiltrate* alle unbeeinflusst, und nur in einem Falle heisst es: Reinigung der *Ulcerationen!* In einem andern ist Besserung des subjektiven Befindens notiert. Um so überraschender ist das gute Resultat von Canepele [373], ferner in einem Fall der Abteilung von Spiess [403] und die vollkommene Heilung in einem Falle Hölschers [387]. Leider fehlen hier klinische und mikroskopische Befunde. Ueber die Wirkung des Salvarsans auf lupöse und tuberkulöse Prozesse wird verschieden geurteilt, sei es nun, dass sie allein oder mit Lues zusammen beobachtet wurden. Safranek [302] sah eine Verschlimmerung, Alexander (Frankfurt) [2], Fleischmann [377], R. Hoffmann [388] Heilung, Troisfontaines Besserung des Allgemeinzustandes. Herxheimer und Altmann hatten gute Erfolge bei Lupus mit einer kombinierten Tuberkulin-Salvarsanbehandlung.

Zieler sah in einem Fall von *sekundärer Syphilis mit Tuberkulose* nach 2 Tagen zweimal Hämoptoe eintreten, in einem andern Fall keinen Einfluss auf den Lungenbefund.

Die in diesem Kapitel mitgeteilten Beobachtungen lassen sich wohl dahin zusammenfassen: *Auf die eigentlichen lokalen Spirochaetosen wirkt Alt- und Neosalvarsan — sei es in intravenöser, sei es in lokaler Applikation — wie ein spezifisches Mittel.* Bei den andern Erkrankungen dürfte die Wirkung wohl von der Ansiedlung der Mundspirochaeten auf den Zerfallsprodukten und deren möglicher Beeinflussung abhängen.

III

Syphilis des Ohres

M. H. Lange nicht so häufig wie Hals und Nase wird bekanntlich das Ohr von der Syphilis affiziert, immerhin wohl häufiger, wie bisher von den meisten angenommen worden ist. Die weiteren Ausführungen werden zeigen, warum das hier von vorneherein besonders betont werden muss. Scheint es doch fast, als ob die Einführung des Salvarsans berufen ist,

unsere bisherigen Anschauungen, besonders über die *frühzeitigen* spezifischen Ohrläsionen, gleichwie über die des Centralnervensystems einer Revision zu unterziehen, wenn auch einige Autoren, unter ihnen der Ref. schon früher auf das häufig frühzeitige Vorkommen gewisser spezifischer Ohraffektionen hingewiesen haben. Ob aber häufiger oder nicht, in *einer* Beziehung hat über die Syphilis des eigentlichen schallperzipierenden Apparats nie eine Meinungsverschiedenheit geherrscht: über ihr refraktäres Verhalten gegen Quecksilber und Jod, über ihre schlechte Prognose überhaupt. In dieser Beziehung nahm das Gehörorgan offenbar gegenüber allen andern Organen eine so besondere, — so besonders ungünstige Stellung ein, dass man wohl bei der spezifischen Affektion des nervösen Apparates auch auf ganz besondere Verhältnisse schliessen durfte. Ob ein neues, sonst noch so mächtiges Mittel hier eine Aenderung herbeiführen würde, war von vorneherein mindestens zweifelhaft, — seine Anwendung aber um so mehr des grössten Interesses sicher.

Ueber die kurative Wirkung des Salvarsans und des Neosalvarsans liegt — ebenso wie über ihre schädigenden Einflüsse — heute schon eine so grosse Menge von Beobachtungen vor, dass eine erschöpfende Uebersicht schwierig ist. Von den andern Arsenmitteln sind mir nur vereinzelte Mitteilungen, meistens über ihren ohrschädigenden Einfluss, so z. B. vom Hektin bekannt geworden (Gaucher-Gougenheim[144-8], Milian[261], Gilbert[165] u. a.), das aber immer noch in Frankreich seine Anhänger hat (Hallopeau). Filaretopoulo hat mit Hektin eben so gute Erfolge wie mit Salvarsan zu verzeichnen. Die Manifestationen der Syphilis am *äussern Ohr*, ob sie nun primärer, sekundärer oder tertiärer Natur sein mögen, zeigen natürlich dem Salvarsan gegenüber dasselbe Verhalten wie alle andern Affektionen des äussern Integuments und werden uns hier wenig interessieren. So heilten Plaques der Ohrmuschel bei Beck[24] in 5 Tagen nach der Injektion, luetische Ulcerationen der Muschel bei Ballenger[11], ein nässendes Exanthem bei Michaelis[255].

Im *Mittelohr* ist ebenso wie im *innern Ohr* eine scharfe Grenze zwischen sogenannten sekundären und tertiären Formen nicht zu ziehen. Die hier im Verlaufe einer Syphilis auftretenden Katarrhe und Eiterungen schliessen sich meist an die entsprechenden Affektionen im Meso- und Epipharynx, noch häufiger aber an die hier so oft lokalisierten gummösulcerösen Prozesse an. An sich unterscheiden sie sich kaum von den *nicht* spezifischen Mittelohraffektionen, bis auf die *häufige Mitbeteiligung des Labyrinths*, und sind deshalb relativ selten beobachtet und beschrieben. Anders ist es schon, wenn man Spirochaeten im Ohreiter nachweisen kann.

Kommen nun die Infiltrationen und ulcerösen Prozesse im Epipharynx unter Salvarsan prompt zur Abheilung, so heilt damit sehr oft auch die consecutive Mittelohraffektion und weiterhin die schon gleichzeitig bestehende labyrinthäre oder retrolabyrinthäre Läsion des Gehörorgans, wenn sie noch jungen Datums ist. Ref. heilte einen Patienten, der nur wegen Hörstörung und Ohrensausen Hilfe suchte, von seinen Papeln

im Rachen nichts wusste und eine Infektion leugnete, mit zwei Injektionen von 0,5 und 0,6 Salvarsan, danach Innunktionskur. Braun [68] heilte einen Fall mit luetischer Otit. interna und gleichzeitigen gummösen Rachenaffektionen durch eine Salvarsaninjektion in etwa 10 Tagen. In der sich an diese Mitteilung anschliessenden Diskussion in der Oesterreichischen Otolog. Gesellschaft (27. II, 1911) wurde von verschiedenen Seiten (M. v. Zeissl, O. Beck, H. Frey, Otto Mayer) darauf hingewiesen — worauf wir später noch näher eingehen müssen — dass *Störungen des innern Ohrs auch für sich allein im Frühstadium der Lues* nicht so selten seien, wie man früher angenommen, und O. Beck hat auf der 20sten Versammlung der Deutschen Otologischen Gesellschaft ausdrücklich betont, dass zuerst in der Monographie des Ref. diesen Tatsachen Rechnung getragen ist. Erst jetzt in der Aera des Salvarsans häufen sich derartige Beobachtungen und unter ihnen finden wir auch schon eine ganze Reihe, in denen die Ehrlich'schen Mittel Besserungen, Heilungen, ja oft ganz verblüffende Erfolge erzielt haben. Das gilt natürlich besonders von den *frischen* Fällen, von denen wir annehmen dürfen, dass es sich bei ihnen im nervösen Apparat noch nicht um degenerativ-atrophische Prozesse handelt, sondern erst um die primären perivaskulären und Gefässveränderungen selbst. Benario [44-8] stellte 10 Fälle von Ohrlues im Sekundärstadium zusammen, von denen 9 wesentlich gebessert oder geheilt wurden. Westler berichtet über eine Heilung von frühzeitiger Neuritis acustica. Haike und Wechselmann [183] erzielten normales Gehör bei einer 2 Wochen alten Labyrinthaffektion. So berichtete auch Siebenmann [312] in der erwähnten Versammlung über 2 Fälle von Taubheit bei sekundärer Lues, von denen der eine vorher eine Hg-Kur gebraucht hatte. Gerade bei diesem trat die Heilung so rasch und so schön auf, dass S. die Kombinationskur als besonders wirksam anzusehen geneigt ist. Durch eine solche heilte auch Zinsser [369] einen frisch-syphilitischen Ohrschwindel. Kohan [228] sah günstigsten Einfluss des Salvarsans bei Ohrensausen und Schwindel auf frisch-luetischer Basis, wo Hg absolut erfolglos geblieben war. Bilancioni [51-2] heilte Schwerhörigkeit und Schwindel und Ohrgeräusche, die einige Monate bestanden und erzielte in einem andern Fall erhebliche subjektive Besserung. Wichtige Fälle teilen Knick und Zaloziecki mit: 1) Exanthem seit 3 Wochen, Affektion des N. vestibularis und cochlearis auf spezifisch-meningitischer Basis — laut Lumbalpunktion — seit 14 Tagen, Taubheit links. Nach 0,4 Salvarsan subjektive und objektive Besserung. 2) Infektion vor 4 Monaten, papulöses Syphilid, Iritis specif. Seit 1 Monat nervöse Schwerhörigkeit, Liquor entzündlich verändert. 0,5 Salvarsan. Vorübergehende Besserung, später Recidiv, Lähmung des Vestibularis. 3) Initialsklerose seit 3-4 Wochen, maculo-papulöses Exanthem. Doppelseitige Neuritis cochlearis, Schwindel. Zweimal 0,5 Salvarsan. Flüsterzahlen von 0,3 und 0,4 auf 2,0 und 2,3 gebessert; kein Schwindel. Werther [355-6] beobachtete: 1) Labyrinthäre Schwerhörigkeit im 4. Monat der Syphilis.

Salvarsan, Besserung aller Beschwerden. 2) Infektion vor 6 Monaten, Ohrensausen, totale Facialisparese. Nach Salvarsan Zurückgehen der Acusticus-Symptome, Besserung des Facialis. 3) Syphilis seit 9 Monaten, Taubheit links, Nystagmus, Schwindel, Geräusche. Nach 0,5 Heilung. Flüsterzahlen von $1\frac{1}{2}$ auf 7 Meter. Hervorragende Erfolge hatte Botey [64]: 1) Infektion vor 9 Monaten, Labyrinthitis seit 2 Monaten; Flüstersprache = 0. Laute Sprache rechts = 20 cm, links = 10 cm. Nach 0,3 Salvarsan keine Besserung. Nach 4 Injektionen von im Ganzen 2,5 Neosalvarsan Fl. r. = 6 m, l. = 8 m! 2) Infektion vor 14 Monaten, Ohrsymptome seit 5 Wochen. Fl. = 0; laute Sprache = 15 und 18 cm. Nach Behandlung mit Salvarsan und Neosalvarsan laute Sprache = 9 m beiderseits! Sehr günstige Resultate hatte auch Denker [89] in 3 frischen Fällen. Im ersten eine Besserung für Flüstersprache von 5 cm bis $5\frac{1}{2}$ m, im zweiten von 0,25 und 0,5 m bis $1\frac{1}{2}$ m, im dritten von 1 bis 6 Meter! Girmonski [66] heilte eine plötzliche totale Ertaubung, vergeblich mit Hg und J behandelt, mit Sa.

Nicht minder nützlich erwies sich das Salvarsan in 3 Fällen rezenter Neuro-Labyrinthitis Citellis [83]. Krumbein [232] erreichte in einem noch relativ frischen Falle Besserung der Cochlear- und Verschlechterung der Vestibularfunktion. Besserung oder Heilung erzielten ferner Okunew [279], Sugàr [325], Perkins [282], Encina [111], Voorhees [345] u. a. Einen Erfolg neben zwei Misserfolgen Ehrler-Thost [101–2]. In den 2 Fällen von Okunew verschwanden in kurzer Zeit alle Symptome sowohl des Cochlear- wie auch des Vestibularapparates.

In einer zweiten Kategorie von Fällen handelt es sich um *alte*, zum Teil sehr alte Lues, aber um mehr oder weniger frische Affektion des innern Ohres. Hierher gehören besonders viele Fälle von *hereditärer Lues* mit plötzlichem Auftreten Menière'scher Symptome. Siebenmann [312] heilte 5 Fälle hereditärluetischer Otit. interna, darunter der auffallendste Erfolg bei dem frischesten, erst einige Monate alten Ohrleiden. — Kander erzielte Heilung bei einem 10jährigen Mädchen, das plötzlich mit schweren Menière'schen Symptomen erkrankte, Krumbein [232] hatte in einem Falle von 3 Monaten keine Besserung, in einem von 8 Monaten Beseitigung der subjektiven Beschwerden und Besserung des Gehörs, in einem 1 Jahr bestehenden eine offensichtliche Besserung des Gehörs. In einem Fall Botellas [60–2] bestand die Syphilis 8 Jahre, seit 8 Monaten eine gummöse Osteo-Periostitis des Labyrinths mit Schwindel, Ohrensausen, Nystagmus, die nach 5 Injektionen verschwanden. Kopp [229], Engelmann, Lang [237], Piolti [284–6] erreichten schnelle Besserung bei Lues herd. tarda, Ernö [112] erzielte bei einem vor 3 Tagen plötzlich einsetzenden spezifischen Menière'schen Symptomenkomplex eine Heilung in 7 Tagen, die nach 5 Monaten noch recidivfrei war. Bei vorher vergeblich mit Hg und Jod behandelten spezifischen Labyrinthstörungen erzielten teils wesentliche Besserung teils Heilung Emery, Sellei [306 308], Neisser [274] (3 Fälle) u. a.

Am verblüffendsten aber und mit unseren bisherigen Vorstellungen

gar nicht recht vereinbar sind die Salvarsanerfolge *bei alten inveterierten Ohraffektionen*, und wir werden sie nur mit den entsprechenden Erfahrungen bei den alten Larynx- und Trachealstenosen vergleichen können. Solche Fälle sind zunächst von Urbantschitsch und Beck[38] mitgeteilt worden. 1) 13 jähriger Knabe, hereditär-luetisch, seit 2 Jahren progredient schwerhörig. Zwei Tage nach der Injektion steigt die Hörweite von 25 cm und 4 m auf $2\frac{1}{2}$ und 9 Meter! 2) 29 jährige Pat. mit hereditärer Lues, seit 2 Jahren vollständig taub, Vestibularis ausgeschaltet. Kein Erfolg. 3) Hochgradige Schwerhörigkeit mit Menière'schen Symptomen bei 26 Jahre bestehender Syphilis. Nach der Injektion Menière günstig beeinflusst, Hörweite von 15 cm und 1 m auf 2 und 4 m. 4) Ein ähnlicher Fall bei 20 jähriger Lues. Nach Salvarsan Besserung des Menière, das Hörvermögen auf der einen Seite gebessert, auf der andern unbeeinflusst. 5) Alte Lues. Nach Injektion Flüstersprache von 5 auf 9, von $1\frac{1}{2}$ auf 8 Meter. In einem sechsten Fall keine Besserung. Werther[355] sah Besserung auch in einigen alten Fällen von Neuritis acustica. Haike und Wechselmann[183] machten folgende Beobachtungen: 1) Linksseitige Labyrinthaffektion bei 18 Jahre bestehender Lues. Nach der Injektion Verbesserung des Hörvermögens von 20 cm in Etappen bis zum normalen Befunde! 2) Lues seit 7 Jahren, Otit. med. et interna seit 2 Jahren. Flüstersprache von 15 auf 45 cm, von 0 auf 15 cm gehoben. 3) Spezifischer Mittelohrkatarrh seit 2 Jahren bei inveterierter Lues. Besserung des Gehörs von $\frac{1}{3}$ und $\frac{3}{4}$ m auf 1 und 3 Meter. 4) Hereditäre Lues, Schwerhörigkeit und Schwindel seit 2 Jahren, Hg ohne Erfolg. Besserung des Hörens rechts auf 4 Meter, links keine Aenderung, Aufhören des Schwindels. In einem fünften Falle von reiner Vestibularerkrankung wird keine Besserung erzielt. Fachini[118] an der Muri'schen Klinik erzielte Besserung resp. Heilung bei doppelseitiger Labyrinthitis, in einem Fall mit Basalmeningitis kombiniert. Besserungen oder Heilungen des Hörvermögens bei oft sehr lange zurückreichenden Affektionen erzielten ferner Donath, Swiatkiewitsch[327], Botella[59-62] in mehreren Fällen u. a.

Der Eintritt der Besserung bei diesen alten Fällen ist ein sehr verschiedener, oft nach Tagen, oft erst nach Wochen. In einem Falle Geredas[155, 160] mit doppelseitiger Labyrinthaffektion (wie alt?) trat die Besserung schon nach 24 Stunden ein. Jedenfalls besteht aber die Ansicht Boteys[64a], dass Salvarsan und Neosalvarsan *nur frische* Labyrinthitis heilt, und nach 3 Monaten jede syphilitische Taubheit unheilbar ist, nach diesen Beobachtungen nicht mehr zu recht. Halten wir uns nun die frühere fast absolute Ohnmacht der Therapie bei der spezifischen Otit. interna vor Augen, so werden wir zugeben müssen, dass das Salvarsan auch hier eine Reihe bemerkenswerter Erfolge aufweisen kann. Auch hier sind — und zwar natürlich sehr viel mehr wie bei der Lues des Halses und der Nase — *Versager* zu notieren. Einige sind schon im vorhergehenden *neben* den günstigen Erfahrungen erwähnt. Andere Autoren haben grösstenteils oder ausschliesslich *negative Resultate* erzielt, so z. B. Wanner[346] bei 4 Fällen *congenitaler* Hörstörungen. Jedes Kind

erhielt allerdings nur einmal 0,2 *Salvarsan*. Er resümiert: ' Ich möchte nicht sagen, dass durch die Injektion mit Salvarsan eine Verschlimmerung eingetreten ist, vielmehr glaube ich, dass die Misserfolge damit zu deuten sind, dass eben auch das Salvarsan ebensowenig, wie eine Schmierkur in der Lage ist, in schwereren Fällen das Fortschreiten dieser tückischen Ohrerkrankung aufzuhalten.' Ohne Einfluss blieb es auch bei Herzog, Imhofer[213], Mangioli, Buys und anderen. Jörgen Möller[263-4], Weintraud[354], Herzfeld, Bilancioni hatten neben guten Resultaten auch negative.

Aber nicht die Salvarsanerfolge und Misserfolge bei spezifischen Gehörerkrankungen beherrschen die Literatur, so weit sie sich mit den Ehrlichschen Mitteln befasst, sondern die *Gehörschädigungen nach anderweitigen mit Salvarsan behandelten luetischen Erkrankungen*, beherrschen sie derart, dass die ersteren fast ganz übersehen werden. Das liegt nun zum Teil daran, dass wohl alle Schädigungen — und diese oft in wiederholten Publikationen (Beck!) — aber nicht alle guten Erfahrungen mitgeteilt worden sind. Auch manche literarischen Lapsus sind dabei im Spiele. Hierfür nur ein Beispiel. So heisst es in einem Referat über den Fall von Berlstein[47]: ' Surdité totale après Salvarsan.' Dass dieser Fall dann geheilt ist, — davon erfahren viele Leser nichts! Schien es aber doch eine Zeitlang, als ob der *Acusticus* dem *Salvarsan* denselben Streich spielen wollte, wie der Optikus dem Atoxyl. Das Atoxyl hat in diesem Kampf weichen müssen; wird das Salvarsan das gleiche Schicksal haben? Um diese Frage objektiv beantworten zu können, müssten wir uns eigentlich zunächst folgende freilich in sich zusammenhängende Vorfragen stellen: 1) Wie viele Schädigungen sind durch *Applikationsfehler* bedingt und in Zukunft vermeidbar? 2) Welche Schädigungen sind dem Salvarsan resp. dem Neosalvarsan als solchem zuzuschreiben? 3) Wie viele Schädigungen sind nur vorübergehende gewesen, spontan oder durch erneute spezifische Behandlung geschwundene, wie viele persistieren? —

Zahlenmässig lassen sich diese Vorfragen nicht beantworten, denn die grosse Teilnahme der gesammten Aerztewelt an der neuen Therapie hat schon in diesen ersten zwei Jahren ein kaum noch übersehbares Material zusammengebracht. Aber eben deshalb und Dank besonders der zentralisierten Sammelforschung Ehrlichs und seines Schülers Benario können diese Fragen grösstenteils schon jetzt im Prinzip beantwortet werden.

Dass bei einem ganz neuen, so differenten Mittel wie dem Ehrlich'schen erst grosse klinische Erfahrungen gesammelt werden müssen, bis die quantitativ und qualitativ beste Anwendungsweise festgestellt ist, ist selbstverständlich und selbstverständlich daher auch, *dass die üblen Folgen aus der ersten Aera der Versuchszeit dem Mittel als solchem nicht in Rechnung gestellt werden können.* Nun sind wir doch aber — und das kann nicht genügend betont werden — über diese Aera kaum noch heraus. Kaum noch mit dem Salvarsan vertraut, wurde uns das *Neosalvarsan* übergeben und spezialistische Beobachtungen hierüber liegen noch sehr wenige vor. Ich brauche auf diese allgemein bekannten Tatsachen nicht einzugehen. Wir wissen jetzt, dass die *intravenöse* Injektion die wenigsten Gefahren mit

sich bringt, wir wissen, wieviel Unheil der *Wasserfehler* (Wechselmann) angerichtet hat, wir wissen, dass zu kleine und verzettelte Dosen sich oft als schädlich erwiesen und sogenannte ' neurotrope ' Wirkungen des Mittels vorgetäuscht haben. Umgekehrt hat man beim Neosalvarsan anfangs zu grosse, zu schnell gehäufte Dosen verabfolgt. Diese Tatsachen werden wir auch bei der hier vorliegenden Frage zu berücksichtigen haben, immerhin ist nicht zu leugnen, dass nach der Einführung des Salvarsans auffallend viele Beobachtungen über Störungen des innern Ohrs im Verlauf der Syphilis, besonders im Frühstadium, veröffentlicht worden sind und dass diese zum Teil sehr schweren Schädigungen zunächst von den meisten Forschern, von manchen auch noch jetzt für rein toxische Salvarsanwirkungen gehalten worden sind, woraus denn die entsprechenden Konsequenzen zu ziehen wären. Andererseits musste es aber von vorneherein von allen Beobachtern zugegeben werden, dass diese nach den Salvarsan-Injektionen aufgetretenen Erscheinungen nichts an sich hatten was sie als Arsen-Vergiftungen charakterisierte, *dass sie an sich im Gegenteil sich in nichts von spezifisch-luetischen Innen-Ohraffektionen unterschieden.* Auch hatte Ehrlich bei einer grossen Reihe von Tierversuchen — bei entsprechender Dosierung (die Hinsberg'schen Dosen waren augenscheinlich viel zu gross) — keine Schädigungen des Gehörs durch Salvarsan nachweisen können, im Gegensatz z. B. zum Arsacetin. Erhlich [104–9] erklärte deshalb die beobachteten Nervenschädigungen als spezifisch-luetische, als ' *Neurorecidive* ', und ihre Entstehung dadurch, dass bei der relativen schwachen Gefässversorgung der Nerven (Wechselmann : bei verstopften Gefässen) hier lokalisierte Spirochaetenherde der Sterilisation entgehen. Ausserdem seien gerade die in engen Knochenkanälen verlaufenden Nerven, also eben Acusticus, Opticus, Facialis, durch die reaktive Schwellung besonders gefährdet.

Auf die an die Mitteilungen Ehrlichs und Benarios sich anschliessende lebhafte Kontroverse brauche ich des näheren hier nicht einzugehen, sie gehört der allgemeinen Medizin an, und es ist Ihnen bekannt, dass die meisten Forscher heute die Ehrlich'sche Ansicht so weit akzeptiert haben, dass sie die nach Salvarsangebrauch aufgetretenen Nervenerscheinungen als luetische, nicht als arsen-toxische ansehen. Bestimmend hierfür war nicht allein die Tatsache, dass viele dieser Erscheinungen durch eine erneute spezifische Behandlung, sei es nun Salvarsan allein, Quecksilber oder beides zusammen, zurückgingen, — denn manche gingen auch spontan zurück, — als viel mehr noch der in letzter Zeit nach dem Vorgange von Assmann [7], Dreyfus [97], Zaloziecki und Frühwald [363] u. a. *fast in allen Fällen von ' Neurorecidiv ' durch die Liquor-Diagnostik erbrachte Beweis, dass es sich im Grunde um Fälle von mehr oder minder ausgesprochener luetischer Meningitis handele.* Der Zellgehalt des Liquors war vermehrt, und zwar meist durch Lymphozyten, der Eiweissgehalt, besonders der Globulingehalt, erhöht (= positive Nonne-Apelt'sche Reaktion), der Wassermann positiv. Fälle mit normalem Liquorbefund sind dagegen nur ganz vereinzelt beobachtet worden. Da nun die luetische Meningitis

sich hauptsächlich an der *Basis* abspielt, so müssen vor allem auch die *basalen Hirnnerven*, und — gemäss der Ehrlich'schen plausibeln Annahme — die Nervi II, VII, und VIII affiziert werden. So werden wir denn die Fälle, in denen Hirn- und Gleichgewichtsstörungen zusammen vorkommen, als primäre Affektionen des *Acusticusstammes*, nicht eigentlich des Labyrinths anzusehen haben, während die vielbesprochene isolierte Läsion oder Ausschaltung des *N. vestibularis* allein — deren Feststellung ja überhaupt erst neuerdings möglich geworden ist (Ruttin) — nur durch besondere anatomische Verhältnisse (Neumann) oder eine besondere Hinfälligkeit dieses Nervenzweiges erklärt werden könnte, wie sie Beck auch stärkeren Hg-Dosen gegenüber annimmt, und wofür wir ein Analogon etwa in der grösseren Hinfälligkeit des Posticusastes des Recurrens haben. — Dass in diesen Fällen meist sonst keine klinisch ausgesprochenen Meningitis-Symptome vorlagen, darf nicht Wunder nehmen, da diese Meningitis im Sekundärstatum meist mehr oder minder latent verläuft und andererseits leichte meningeale Reizungen, wie Kopfschmerzen und dergl., vielfach notiert worden sind. Geht somit heute die Ansicht der meisten Forscher — Wechselmann [352–3], Haike [183], Frey [135], Geronne-Gutmann [162], Assmann [7], Zaloziecki-Frühwald [363], Gennerich [156–7], Neisser [272], v. Zeissl [364–6], Knick [227], Desneux-Dujardin [91], Milian [257, 261], Levine, Voss [342], Hoffmann [204], Jadassohn, Lipsztat [244], Stepanoff [318], Sänger, Schwenkenbacher, Dreyfus [97–100], Fehr u. a. — dahin, dass auch die Affektionen des innern Ohrs resp. des N. acusticus nach Salvarsangebrauch gleich denen der andern Nerven spezifisch-syphilitische sind, so bleibt doch auch hier die Frage zu beantworten, *warum denn diese Nervenerscheinungen im Frühstadium der Syphilis jetzt so häufig und vor der Salvarsanaera gar nicht oder doch höchst selten beobachtet worden sind*? Diese Frage nun schliesst eine Behauptung ein, die nach unserer Ansicht erst für sich eines Beweises bedarf: dass Erkrankungen des innern Ohrs resp. des Acusticus im Frühstadium der Syphilis vor der Salvarsanaera überaus seltene Erscheinungen waren. Von den spezifischen Nervenerscheinungen wissen wir erst in der neueren Zeit, dass viele von ihnen in das erste Jahr — bei *Naunyn* 20 Prozent — ja, in die ersten Monate nach der Infektion fallen. Was speziell das Gehörorgan betrifft, so ist darüber vor allem zu sagen, dass dessen spezifischen Erkrankungen — auch in den Lehrbüchern der Ohrenheilkunde — bis vor kurzem überhaupt nicht genügend Beachtung geschenkt ist (Frey, Beck), und es daher eigentlich niemanden Wunder nehmen darf, wenn er jetzt, wo plötzlich die Aufmerksamkeit der ganzen ärztlichen Welt auf die 'Neurorecidive' gelenkt ist, seine früheren irrigen Anschauungen revidieren muss. Zustände, die man nicht kennt, kann man nicht buchen. Ich darf wohl das Verdienst für mich in Anspruch nehmen, dass ich in meinem Buche in allgemeinerer Darstellung zuerst den wirklichen Verhältnissen entsprechend Rechnung getragen habe, wie denn auch Beck, Imhofer u. a. bei der Erörterung der Neurorecidivfrage meine Ausführungen zum Ausgangspunkt ihrer Erörterungen gemacht haben. Es heisst dort:

'Eine Mitbeteiligung des Labyrinths bei einer grossen Reihe von spezifischen sekundären Mittelohrerkrankungen haben wir soeben als ein gewissermassen besonderes Merkzeichen dieser letzteren kennen gelernt. (In den Haike-Wechselmann'schen Neurorecidiv-Fällen war neben dem Labyrinth immer auch das Mittelohr beteiligt, — und in vielen andern Fällen gewiss nur nicht bemerkbar geworden.) Syphilitische Labyrintherkrankungen *können aber auch allein in jedem Stadium, zu jeder Zeit auftreten*, wenn auch freilich die Spätformen am innern Ohr die häufigsten sind.' Ich habe dann auf die früheren Fälle Politzer, Schwartze, Habermann und Baratoux hingewiesen, welch letzterer die Labyrintherkrankungen am häufigsten am Ende der sekundären und am Anfang der tertiären Periode auftreten lässt. 'Den Beginn der sekundären Labyrinthaffektionen konnte ich meistens gleichzeitig mit dem Ausbruch sekundärer Nasen- und Rachenaffektionen konstatieren,' — was Beck neuerdings für die Vestibularisaffektionen speziell bestätigt. 'Die Veränderungen können natürlich den Vorhofs- wie den Schneckennerven selbst und auch den Acusticusstamm resp. sein *Perineurium* betreffen.' Ich erwähne dann Jung, der die Hörstörungen in der sekundären Periode vorwiegend oder ausschliesslich auf Rechnung des Acusticus setzt, — was ich damals irrigerweise nicht anerkannte, und wofür Jung schon die Fälle von Bockhart, de Horne und Pearsson anführt. Es wird weiterhin dann der sehr verdienstvollen Arbeit von Rosenstein gedacht, die auch eine Reihe von Frühfällen des Acusticus bringt, so die von Barr, Ostino und Santamaria, Oedemann und Hermet und zwei eigene Fälle, bei denen allen wenige Monate nach der Infektion Schwerhörigkeit oder Taubheit, Schwindel und Erbrechen, mit oder ohne nachfolgende Facialisparese auftrat. Aehnliches beobachtete Lannois u. a.

Diese und eigene Beobachtungen sind also schon in meinem Buche mitgeteilt und ihre Kenntnis hätte allein schon genügen können, die Mitteilungen über die 'Salvarsanschädigungen des Gehörorgans', die sich ihrer Natur nach so gar nicht von jenen unterscheiden, wenigstens mit Skepsis aufzunehmen. Solche Beobachtungen aus dem sekundären Stadium sind aber seither noch sehr vermehrt worden. So führt Frey [135] allein weitere Fälle von Barr, Buck, Charazac (Gerber), Heermann, Hoffmann, Jansen, Lanceraux, Jörgen Möller, Sexton, K. Stein, Roosa (3 Fälle), Crouzillac, Freytag, Grünberg, Magnus Möller (2 Fälle) und Stümpke (2 Fälle) auf. Aehnliche Mitteilungen folgen jetzt sehr zahlreich, so die von Kreibich [231] (4 Fälle in einem Jahr), Beck, Hintze [203], Werther [356] in 2 Jahren 7 Fälle, die im ersten Jahr der Syphilis Hirnnervenaffekte zeigten, 3 unbehandelt, 4 nach Hg-Kuren. v. Zeissl [364] in einer Woche 3 Fälle von Facialislähmung, Ohrensausen und Schwerhörigkeit, ohne jede vorangehende Behandlung. Otto Mayer [253] führt die Erhebungen von Habermann an der Grazer Klinik fort und hat seitdem 65 Acusticusstörungen (!) im Sekundärstadium zusammenstellen können, von denen 6: 3–6 Wochen, 7: 6–10, 8: 10 bis 16 Wochen nach dem Primäraffekt auftraten. Allerdings waren keine isolierte

Vestibulariserkrankungen darunter, die er eher als toxische aufzufassen geneigt ist. Knick [227a] beobachtete an der Leipziger Klinik 29 Acusticusstörungen im Frühstadium, darunter 18 mit Salvarsan, 2 mit Hg, 9 *ohne jede Behandlung*! Nur ein einziger Ausnahmefall schien daneben zu bestehen und toxischer Natur zu sein, bis er sich bei erneuter Untersuchung als spezifisch luetischer Fall entpuppte! Nimmt man hierzu noch die Tatsache, dass ein grosser, ja vielleicht der grösste Teil von Fällen mit leichterer Neuritis acustica, weil nur mit geringen Beschwerden oder ganz ohne sie einhergehend, gar nicht zur ärztlichen Beobachtung gekommen ist (Rosenstein) — Dreyfus [97–100] hat unter 11 teils gar nicht, teils mit Salvarsan, teils mit Hg behandelte Fällen 3 schwere Acusticusläsionen ohne alle Symptome konstatieren können — so wird man den Hinweis, dass man in der Vor-Salvarsanzeit im rezenten Stadium der Syphilis Läsionen des innern Ohres nicht, oder fast nicht gekannt hätte (Alexander u. a.), als Argument für die neurotrope Wirkung des Salvarsans, besonders auf den Acusticus, nicht mehr verwenden können (Frey).

Ist es also einerseits nicht mehr zu bestreiten, dass auch vor der Salvarsan-Aera Acusticusaffektionen im Frühstadium der Syphilis zahlreich vorgekommen sind, dass sie überhaupt als spezifische Erkrankungserscheinungen auftraten, so ist andererseits nicht zu verkennen, dass zahlreiche vorher ohrgesunde, oder doch nicht nachweislich ohrkranke Syphilitiker nach Salvarsaninjektionen in ziemlich typischem Zeitablauf am Acusticus erkranken. Halten wir diese Erkrankung auch für eine luetische, so ist die *provokatorische* Wirkung des Salvarsans dabei doch kaum zu verkennen.

Die in Rede stehenden Erscheinungen treten bekanntlich nach zwei verschiedenen Zeitintervallen auf und sind danach in zwei verschiedene Kategorien einzuteilen: In einer kleineren Gruppe wenige Stunden oder Tage nach der Injektion; und diese werden wir nach Ehrlich und Urbantschitsch durchaus als *Herxheimer'sche Reaktion* auffassen, die meist schwindet wie sie gekommen (Fälle von Urbantschitsch, Beck, Biehl, Sellei, Port, Müller (Mainz), Hahn (Bremen), Ehrmann b. Benario, der bemerkenswerte Fall von Peritz [281]). In der zweiten, weitaus grösseren Zahl aber durchschnittlich 6–9 Wochen nach der Injektion — die eigentlichen Neurorecidive, deren Erklärung durch Ehrlich ja oben erwähnt ist.

Wir haben uns aber nun die weitere Frage zu stellen, ob diese auch von Ehrlich nicht geleugnete provokatorische Wirkung nur der Salvarsan-*oder auch der Hg-Therapie zukommt*? Bekanntlich glaubt Benario [41], den Beweis für letzteres erbracht zu haben, indem er den Neurorecidiven bei Salvarsanbehandlung die im gleichen Zeitraum gesammelten Neurorecidive nach Hg-Behandlung an die Seite stellt und zeigt, dass die erhaltenen Zahlen eine fast absolute Uebereinstimmung — für den Acusticus geradezu eine absolute — bieten. Schon von den oben mitgeteilten Acusticusstörungen der Frühperiode waren viele während Hg-

Behandlung aufgetreten, so die Fälle von Stümpke [319], einige von Knick [227], Werther [356] u. a. Benario aber konnte unter 210 Salvarsan-Neurorecidiven 79 Acusticusaffektionen-, unter 121 Hg-Neurorecidiven 46 notieren. Des näheren sind bei den ersteren betroffen: beide Aeste 33 mal, der Cochlearis allein 23 mal, der Vestibularis 6 mal. Der Acusticus mit andern Hirnnerven zusammen 17 mal. Seine Tabellen sind freilich, wie Finger mit Recht sagt, ' nicht ganz leicht zu entziffern '. Danach kann übrigens von einer — von Beck u. a. — behaupteten charakteristischen Bevorzugung des Vestibularis auch nicht die Rede sein. Auch in dem Sammelreferat von Valentin [341] ist das nicht der Fall. Er stellte 45 Fälle von Acusticusläsionen zusammen, von denen 9 den Cochlearis allein, 7 den Vestibularis und 20 beide Aeste betrafen.

Die Tabellen und daraus gezogene Schlüsse Benarios sind nun von vielen Seiten angezweifelt und kritisiert worden, besonders scharf von Finger [128–32], der lange der Hauptverfechter der Neurotropie des Salvarsans gewesen ist. Die beiden Haupteinwände, die Finger gegen die Statistik Benarios anführt, sind: 1) dass B. die Zahl der Syphilitiker, unter denen die 121 Hg-Neurorecidive beobachtet sind, nicht wissen kann, womit F. sagen will, dass diese gewiss im Verhältnis zu der Zahl, aus denen die Salvarsanrecidive geschöpft sind, eine sehr grosse ist. Dagegen ist nun zu sagen, dass in den Jahren 1910 und 1911, aus denen die Zahlenreihen stammen, die Zahl der mit Hg behandelten eo ipso gegenüber der Zahl der Salvarsanbehandelten relativ nicht eben gross gewesen sein wird. Zudem ist die Tatsache, dass in kurzer Zeit ohne besondere Mühe überhaupt 121 Hg-Neurorecidive festgestellt werden konnten, das für die Frage der Salvarsanwirkung wichtige.

Der zweite Einwand ist der, dass Benario für seine Berechnung der Salvarsan-Neurorecidive das *gesamte* Material von Herxheimer, Arning, Weintraud und Wechselmann benutzt und nicht nur — wie verlangt werden muss — die genügend, d. h. mindestens 4 Monate nach der Injektion kontrollierten Fälle. Dieses Postulat ist im strengwissenschaftlichen Sinne durchaus berechtigt. Wenn wir aber bedenken, dass die Patienten mit so schweren Störungen wie sie die Neurorecidive doch meistens darstellen, höchstwahrscheinlich wiederum ihren oder einen andern Arzt aufsuchen würden, dass sie so oder so doch wohl kaum spurlos aus der Beobachtung und der in dieser Hinsicht doch durchaus nicht zurückhaltenden Literatur verschwunden sein würden, *so werden wir nicht annehmen können, dass ein grösserer Prozentsatz der nicht lange genug in Evidenz gehaltenen Fälle späterhin noch Neurorecidive bekommen haben wird.* Fingers Vermutungen widersprechen auch den speziellen Erfahrungen der Militärlazarette. Damit verlieren beide Einwände Fingers gegen die Statistiken Benarios — was man auch sonst an ihnen aussetzen mag — für die Praxis ihre Bedeutung und können den Kernpunkt der Ehrlich-Benario'schen Theorie nicht erschüttern.

In Uebereinstimmung hingegen befinde ich mich mit Finger hinsichtlich der Rolle, die die *extragenitalen* ' Kopfschanker ' bei Benario

spielen und auf die auch Rille als möglich hinweist. Auch ich glaube nicht, dass die Hypothese einer besonderen Bösartigkeit der extragenitalen Sklerosen aufrecht erhalten werden kann. Wenn manche solcher Fälle vielleicht schlechter verlaufen, so ist das wohl nur deshalb, weil ihre Diagnose bisweilen schwieriger, — und ihre Therapie deswegen nicht rechtzeitig eingeleitet worden ist.

Es soll nicht verschwiegen werden, dass neben Finger auch noch andere Autoren an eine neurotrope Wirkung des Salvarsans glauben, so Stern, Wiltenhöfer, Levy Port, Bethmann, Buschke und Rille, welch letzterer die Summierung des Arsen- und Syphilisgiftes für möglich hält, und auch noch nach den Benario'schen Statistiken sind viele Fälle von Neurorecidiven veröffentlicht worden, sowohl von Beobachtern, die sie für toxischer, wie auch von solchen, die sie für spezifischer Natur halten. Von ersteren wären z. B. zu erwähnen Marzochhi, Tommasi, Sicard, Castex, Ehrler, Salzer und Faye, Kerl, Segmann, Neumann (Wien), Marschalko, Guisez, Gelle v. Bergmann, Brinitzer, Cimbal u. a. Nur in einem *einzigen* dieser Fälle ist aber die normale Beschaffenheit des Liquor nachgewiesen oder notiert worden und somit können die andern nach unserer heutigen Anschauung nicht die Annahme ihrer Autoren stützen, denn bei ihnen allen kann es sich eben um spezifische basalmeningitische Affektionen handeln, wie z. B. in den beiden Fällen von Gellé [149], und in dem einen von Sicard [310 11], wo ja ausdrücklich Lymphozytose des Liquors angegeben ist. Andere Autoren lassen die Frage, ob eine Salvarsanwirkung bei den Neurorecidiven vorliegt oder nicht, auch jetzt noch unentschieden, so Closier, Freystadtl u. a., indess die neueren Untersuchungen, gestützt *auf vergleichende Ohruntersuchungen bei gar nicht behandelten, bei mit Hg und Sa. behandelten, die ganz übereinstimmende Resultate ergaben, gestützt ferner auf die Heilung der Erscheinungen durch weitere spezifische Behandlung und schliesslich auf die Liquorbefunde, eine direkte Neurotropie des Ehrlich'schen Mittels in Abrede stellen.* Hierzu kommt die wichtige Erfahrungstatsache, einmal dass Salvarsan gerade bei *Lues cerebrospinalis* keine Neurorecidive macht, und ferner, dass es auch hier vielfach glänzende Heilwirkungen entfaltete (Gennerich, Becker u. viele andere), wie es auch den ohne klinische Erscheinungen in der Sekundärperiode veränderten Liquor günstig beeinflusste (Dreyfus u. a.). Auch die Finger'sche Klinik hat wohl, wie aus der Mucha'schen Arbeit zu schliessen, ihre Ansicht hinsichtlich der Neurorecidive inzwischen etwas gewandelt. Die wichtigen Arbeiten über die Liquorbefunde von Assmann, Dreyfus, Zaloziecki und Frühwald u. a. sind ja schon oben zitiert worden, — in ersterer Hinsicht sind die Untersuchungen von Stepanoff [316], Denker [89] Botella u. a. von grosser Bedeutung. Stepanoff stellte 100 Untersuchungen bei 33 Syphilitikern im Sekundärstadium an, und zwar alle zwei Wochen, mehrere Monate hindurch, mittels Konversations- und Flüstersprache, Lucae'scher Stimmgabel und Galtonpfeife und kalorischer Prüfung. Am Vestibularapparat wurde häufiger eine Herabsetzung, seltener eine Steigerung — meist ein normaler Befund —

erhoben, ob die Patienten nun mit Salvarsan oder mit Hg behandelt waren. Am Cochlearapparat häufiger eine Besserung, seltener eine Herabsetzung und status quo. Die Störungen sind persistenter bei Hg. Bei Prüfung mit C_4 zeigt sich nach Salvarsan eine Herabsetzung in $\frac{2}{3}$ der Fälle; mit Merkur die analogen Resultate. Bei Prüfung mit dem Monochord zeigt sich nach Salvarsan eine bedeutende Heraufsetzung der oberen Tongrenze.

Denker führte gleichfalls 100 Untersuchungen bei 22 Patienten aus. Es wurde geprüft: Weber'scher Versuch mit A, Schwabach'scher Versuch mit A, Rinne mit a! Ferner wurde geprüft die Hördauer der Stimmgabel C,c,c^1 $C^1c^3c^4$, bei sämtlichen Versuchen mit dem Dölger'schen Stimmgabelspanner in derselben Stärke gespannt. Der Nystagmus wurde bei jedem Patienten mit dem Drehstuhl geprüft. Die erste Untersuchung erfolgte stets vor der ersten Einspritzung des Salvarsans, die weiteren Untersuchungen begannen 3 bis 8 Tage nach der Anwendung des Mittels und erstreckten sich auf eine Dauer von 1 bis 4 Monaten nach der ersten Injektion. — Das Ergebnis dieser Untersuchungen lässt sich dahin zusammenfassen, *dass sich nicht die geringste Einwirkung des Salvarsans auf den Cochlear- und Vestibularapparat feststellen liess.* Botella untersuchte 81 Fälle vor und nach der Injektion. Er konstatierte 38 Ohrstörungen *vor* jeder Behandlung! Von diesen blieben 24 nichtluetische bestehen, 7 luetische wurden gebessert und geheilt, bei 7 war der Ausgang unbekannt. Vor und nachher normal waren 35. Neurorecidive 4 mal, 2 geheilt, 1 geblieben, 1 unbekannt. 4 vorübergehende Störungen. Zu ähnlichen Schlüssen kommen Canestro (Genua) u. a. Ich selbst habe bei meinen wegen Lues des Halses oder der Nase einer Salvarsankur unterzogenen Patienten den Cochlearapparat regelmässig, wiederholt auch den Vestibularapparat kalorisch geprüft, — und bei keinem ohrgesunden Patienten eine Aenderung konstatieren können.

Was nun die Heilung der Salvarsan-Neurorecidive betrifft, so ist der *weitaus grösste Teil* bekanntlich geheilt, bei Benario 83 Prozent, gebessert 8 Prozent. Selbst bei Beck, der das Wiener Material von Finger, Ehrmann, etc., zusammengefasst hat, von 25 Ohraffektionen 13 geheilt oder gebessert. Freilich sind einige Störungen, so bei Haicke in einem Fall geblieben. Goldbach (Lesser'sche Klinik), der unter 442 injizierten Fällen 5 Neurorecidive hat, — also dem Benario'schen Prozentsatz ziemlich entsprechend, — sah 4 davon heilen, Strandberg von 10 Fällen 8, Heden alle 3 Fälle, und ähnlich bei den meisten Autoren, *so dass die Zahl der dauernden Schädigungen überhaupt relativ eine äusserst geringe ist.* Am lautesten aber sprechen für das Salvarsan seine Leistungen bei den Hg-Neurorecidiven. *Von 121 Quecksilber-Neurorecidiven sind 39 geheilt, 29 gebessert, in Summa 68 durch Salvarsan,* gegenüber 12 Heilungen und 4 Besserungen durch Quecksilber. Dass man nach alledem bei den Neurorecidiven — wie immer man sie sich sonst erklären mag — von eigentlichen Salvarsanschädigungen nicht mehr sprechen kann, ist selbstverständlich.

Immerhin — und daran dürfen wir nicht stillschweigend vorübergehen — bleiben manche Rätsel noch ungelöst. So lässt sich der Einwand von Ehrlich, Benario u. a., dass das Salvarsan, bei nicht luetischen Prozessen angewandt, noch nie Acusticusläsionen ausgelöst habe, nicht mehr aufrecht erhalten, nachdem solche Störungen in einem Malariafalle Sicards [310] und einem Falle einer Leistendrüsenerkrankung von Neubauer [275] beobachtet worden sind, während die Verschlechterung in dem Otosklerosefall von Sugar [323-4] natürlich hier nicht herangezogen werden kann. Wie erklären wir nun diese — an Zahl allerdings so bescheidenen — Fälle? Wie erklären wir ferner die wenigen Neurorecidivfälle mit *normalem* Liquorbefund? Wie vor allem die für viele Beobachter fortbestehende Tatsache, dass die Neurorecidivfälle seit der Einführung des Salvarsans häufiger und frühzeitiger auftreten?

Für die nicht spezifischen Fälle und die mit normalem Liquorbefund werden wir wohl auf eine Arsenschädigung zurückgreifen müssen. Ich möchte da an die — schon so lange vor der Salvarsan-Aera — von Politzer betonte *besondere Hinfälligkeit des Acusticus gegen alle möglichen Noxen* erinnern, die es wohl verständlich machen kann, dass ein Mittel von so mächtiger Wirkung wie das Salvarsan bei besonderer Disposition die in Rede stehenden Schädigungen auszulösen vermag. Im übrigen handelt es sich hier um so verschwindend wenige Fälle, dass sie für die Bewertung des Salvarsans als Heilmittel eigentlich nur einen theoretischen Wert besitzen.

Unendlich viel wichtiger ist die Frage nach der Frühzeitigkeit und Häufigkeit der Neurorecidive bei der Salvarsantherapie. Die Ansichten Ehrlichs (= sterilisatio fere completa und daher stärkeres Wachstum der restierenden Spirochaeten) und seiner Anhänger, sowohl wie die seiner sich anscheinend immer mehr vermindernden Gegner sind Ihnen bekannt und brauchen hier nicht ausführlich wiederholt zu werden. Die ersteren geben hauptsächlich einer zu wenig intensiven Behandlung die Schuld, nach der natürlich die überlebenden Spirochaeten ein um so leichteres Spiel haben dürften. Dreyfus [99] hat dies durch fortlaufende Liquoruntersuchungen während der Injektionen bewiesen. Wir haben oben gesehen, dass schon in der Sekundärperiode meningitische und neuritische Affektionen *latent* wohl in sehr viel mehr Fällen vorhanden sind, als man früher angenommen; dass diese durch den mächtigen therapeutischen Iktus des Salvarsans plötzlich manifest werden (Ledermann), ist eine naheliegende Annahme, dass sie ohne diesen in *späterer* Zeit manifest geworden wären, sehr wahrscheinlich. Haike und Wechselmann kommen zu dem Schluss, dass die nach Salvarsaninjektion auftretenden Affektionen des Acusticus durch das Mittel indirekt veranlasst werden, aber nur an Stellen, an denen Spirochaeten lagern, die ohne Anregung durch das Arsenobenzol wahrscheinlich zu einer späteren Zeit die gleichen Erscheinungen hervorgerufen hätten. Eine solche, durch das Salvarsan lediglich bedingte *Verfrühung* der Erscheinungen würde aus naheliegenden Gründen therapeutisch in der Tat nur einen Vorteil

bedeuten. Mit Recht betonen Géronne und Gutmann, dass die so ganz anders geartete Sa.-Therapie der Syphilis fortan überhaupt, besonders hinsichtlich des zeitlichen Ablaufs, ein ganz anderes Gesicht geben könnte. Wir müssen hierbei ferner auch an die mächtige Einwirkung des Salvarsans auf die gesamten Immunisierungsvorgänge im Körper denken (Benario, Gennerich, Assmann u. a.), die in den wiederholt nach Salvarsanbehandlung beobachteten *Reinfektionen* ihren beredten Ausdruck finden. Bei der oft blitzartig erfolgenden Abtötung der Spirochaeten können nicht genügend Immunstoffe gebildet werden und die gerade an den Nerven vom Salvarsan nicht erreichten kleinen Spirochaetendepots können nun neu wuchern. Hierdurch wird auch der auch bei den Neurorecidiven konstatierte Antagonismus zwischen Nerven- und Integumentsyphilis, sowie der so häufig negative Wassermann erklärt. Ob wir uns nun diesen oder jenen Erklärungsversuch zu eigen machen wollen — über deren Richtigkeit ja erst, wie über so manche anderen Salvarsanfragen, die Zukunft entscheiden kann, — wir haben uns nun zum Schlusse mit den für die Praxis weitaus wichtigsten Fragen zu beschäftigen:

1. *Wie häufig kommen denn diese Neurorecidive vor? Müssen wir sie in der Praxis berücksichtigen?*

2. *Wenn das letztere der Fall ist, können wir sie vermeiden und auf welche Weise?*

Wenn man die medizinische Literatur des Jahres 1911 und 1912 durchsieht, so könnte man glauben dass die Neurorecidive alltägliche Erscheinungen seien. Ein klareres Bild bekam man zuerst aus der Zusammenstellung Benarios in der *Münch. med. Woch.*, die damals aus ca. 14000 Fällen 126 Neurorecidive, d. h. einen Prozentsatz von 0,9 berechnet hatte. In seinem Buche war es ihm dann nicht mehr möglich, die absolute Zahl der Fälle zu ermitteln, aus der die neue Zahl von 194 Neurorecidiven gewonnen war. Er bringt dann eine Zusammenstellung des Materials und der Neurorecidive einer grösseren Zahl von Beobachtern, bei denen — mit Ausnahme von Fingers 9 Prozent — die Prozente zwischen 0,09 und 2,7 schwanken, im grossen und ganzen auch den Durchschnitt von noch nicht 1 Prozent kaum überschreiten. Die Kontroverse von Finger und Benario ist schon früher kritisch beleuchtet worden. Auffallend ist es ja immerhin, dass auch der einzige Fall von *Amaurose* unter (damals) 30000 Fällen bei Finger beobachtet worden ist. Der Genius loci Wiens scheint dem Salvarsan ungünstig zu sein! Deshalb, und weil diese Frage für die Praxis die von allem wichtigste ist, bringe ich in nachfolgendem noch 2 Tabellen, eine aus Syphilido-Innern und Nervenkliniken etc., deren Beobachter bei Benario gar nicht vertreten sind (mit † bezeichnet) oder mit kleineren Zahlen, und einer zweiten Liste aus oto-laryngologischen Kliniken, die in Folge mangelnder Antworten auf meine Rundfrage leider nur klein ist. Wegen ihrer besonderen Bedeutung sind die Beobachtungen aus einigen Militärlazaretten zusammengestellt.

1. Dermatologische und andere Kliniken

Autor.	*Zahl der Fälle.*	*Neurorecidive.*	*Bemerkungen.*
Arzt †	420	Nur vorübergehende Acusticussymptome	Von 130 nur mit Salvarsan beh. 58% Recidive. Von 100 mit Salv.+Hg kein Recidiv.
Arning	1000	1 Neur. opt. nach 0,6 1 sensibl. Störung im r. N. ulnaris	Heilung nach Salvarsan. „ „ „
Bingel	250	Keine.	
Browning †	300		Keine Nebenwirkungen.
Dössekker †	458	10 Neurorecidive	4 nach 1 Injekt.; 6 nach mehreren Injekt.
Fabry	2653		Seit kombinierter Behandlung keine Schädigungen.
Desneux-Dujardin †	350	6 Neurorecidive bei Anfangsdosen	Jetzt bei intensiver Behandlung keine Schädigungen.
Favento	2000 Salv.	Keine	Kein Versager. 1 Exitus b. luet. Meningitis mit falscher Behandlung.
	60 Neo-Salv.	Keine	Gute Erfolge.
Füresz †	273 intravenös	Keine.	
Frühauf †	238	1 Vestib.-Schädigung.	
Klemm †	250	Keine	Ausgezeichnete Resultate. Kombinierte Kur.
Isaak †	1800	Keine	Bester Erfolg.
Galewski	230	1 Cochleariserkrank.	Durch Salvarsan geheilt.
Goldbach	1000	5 Acusticus, davon 1 Herxheimer, 4 geheilt.	
Guttmann	56 Alt-Salv.; 175 Neo-Salv.; 5 beides	Keine	Seit 2 Jahren bei intensiver Behandlung.
Hoffmann	Mehrere Tausende	Keine dauernde Schädigung.	
Hesse	100	1 Acusticuslähmung, mit 0,3 Hg geheilt.	
v. Marschalko†	231 Neo-Salv.	Keine.	
Mucha u. Finger	500	40 z. T. schwerste unheilbare Nervenerkrankungen.	
Loewenberg †	165	Keine.	
Nonne †	129		
Sellei	Über 1000	4 Störungen, davon 1 geheilt.	
Simon †	170	Keine.	
Milian †	8000	Mehrmals Erytheme. 3 mal Icterus. 2 mal Venenthrombose.	
Stokar †	450 Alt-Salv.; 50 Neo-Salv.	Keine.	
Lipsztal †	32	2 Gehörstörungen	1 geheilt:
Wechselmann	Mehr als 12,000		Niemals richtige Vergiftung. Prognose des N.-R. ist günstig.
Werther	350	5	4 geheilt, 1 gebessert.
Riecke	Über 300	3 ganz leichte Störungen, einmal Schwindel, einmal Fieber u. Nackensteifkr.; später noch 2 leichte Acusticusstörung., einer nach weiteren Neo-Salv. geheilt, der andere nicht mitgeteilt.	

1. Dermatologische und andere Kliniken—*Forts.*

Autor.	*Zahl der Fälle.*	*Neurorecidive.*	*Bemerkungen.*
v. Zeissl (Frey)	200	Keine Störung.	
Schreiber	1323 Alt-Salv.	5 sichere N.-R.	Nach antiluet. Behandlung geheilt.
		1 fragl. des Gehörnervs	Geheilt.
	528 Neo-Salv.	1 sicher N.-R.; 1 fragl. N.-R.	Gebessert n. in Behandlung.
Beck, Finger, Ehrmann, etc.	1500	29 Gehöraffekt., davon 13 geheilt oder gebessert	Seit Befolgung der von ihm aufgestellten Indikationen wenige Affektionen.
Neisser	3419	Keine eigene N.-R.	2 aus anderer Behandl.
Strandberg	211	Wiederholte Gehörstörung. 15 Neurorec., davon 10 Ohrfälle, von diesen 8 geheilt	In Folge zu kleiner Dosen.
Lenzmann		Keine	In letzter Zeit.
Heuk	700 Injekt. v. Neo-Salv.	Keine	Auch nach Alt-Salvarsan in letzter Zeit keine mehr.
Kren-Riehl	600	(?)	'Das Neurorecidiv ist ein Symptom der Syphilis, das eine energische Salvarsanbehandlung erfordert.'
Nakano	300	4	Vorübergehende Schädig.
Donath	107	Keine	2 Besserungen des Gehörs u. d. Facialis bei Tabes.
Frieboes (Hautklinik Bonn)	Mehrere Tausende (Injektionen ?)	Keine.	
Dind	66	Keine.	
Ballenger	110	Keine.	
v. Petersen	200 Fälle und 200 weitere Injektionen	Keine	Sehr gute Erfolge.
Zinsser-Kannengiesser	ca. 1500 Fälle	7 vorübergehende, keine dauernden	Seit energischer kombinierter Behandlung überhaupt keine Neurorecidive.
		Militärlazarette	
Gennerich	850	6	3 mal syphilitiformer Anfall. 1 mal Facialisparese. 2 mal Kopfschmerzen mit patholog. Punktat. Keine gröberen Hörstörungen.
Berger	69	Keine	100 % ohne Recidive bei 7–10 monatlicher Beobachtung. *Kombinierte* Kur.
Nichols	1200	3.	
Gibbard	392	Eine Myelitis.	
Wiener Garnisonlazarett	2300	8 = 0,35 %	7 davon geheilt. Leichte Störungen nervöser Art: 22.

2. Hals-, Nasen- und Ohrenkliniken

Autor.	*Zahl der Fälle.*	*Neurorecidive.*	*Bemerkungen.*
Lubliner	50 Fälle behandelt	Drei vorübergehende Störungen, aber keine schweren Störungen.	
Hinsberg		Keine üblen Nebenwirkungen.	
Killian	Sehr viele Injektionen	Wenig Unangenehmes.	
Avellis	20 Fälle Salv.; 4 Fälle Neo-Salv.		Keine Ohrschädigungen; keine Ohrbesserungen.
Denker		Keine Störungen	Bei sekundärer u. tertiärer Rachen- u. Kehlkopf-Syphilis ausserordentlich prompte u. vollständige Abheilung der Ulzer.
Barth	350 Fälle Salv. u. Neo-Salv.		22 Acusticus-Erkrankungen. 4 einseitige Taubheit geblieben. 1 doppelseitige Taubh. geblieben; z. T. infolge zu später antiluetischer Behandlg. durch Liquor event. luet. Meningitis.
Piolti	15 Fälle	Keine Störungen.	
Gereda	Bei 614 Fällen	1 vorübergehende Störung.	
Brieger	(?)	Wiederholte Störungen des Gehörs.	In Folge anderwärts gegebener *unzureichender* Dosen.
Eschweiler	(?)	Keine üblen Dauererscheinungen.	
v. Sokolowsky	3 Fälle	Keine	Syphilis maligna, nach 2 Jahren kein Recidiv.
Botey	ca. 100 Fälle	Keine	
P. Heymann-Berlin mit K. W. Frank u. Th. Mayer	900 Injekt.		2 kleine vorübergehende Hörstörungen, einer von selbst, einer nach nochmaliger Salvarsaninjektion zurückgegangen.
Halle	215 Salv.; 48 Neo-Salv.	Keine	2 luetische Hörstörungen, 1 mit gutem, 1 mit frappant gutem Erfolge behandelt.
Gerber	95 Salv.; 35 Neo-Salv.	Keine	2 mal Heilung bei spezifischem Mittelohrkatarrh. Kein Erfolg bei chronischer Schwerhörigkeit eines Luetikers.
Safranek-Onodi	Ueber 200	Keine	

Hierdurch dürfte nicht nur die Richtigkeit der Benario'schen Angaben in Bezug auf die Zahl der Neurorecidive für die Zeit ihrer Aufstellung bestätigt sein, es geht vielmehr mit Klarheit daraus hervor — und ist von verschiedenen Autoren, unter ihnen selbst von Beck noch ausdrücklich hervorgehoben, — dass die Neurorecidive, wie auch alle andern üblen Nebenerscheinungen bei Anwendung des Salvarsans, *in letzter Zeit*

immer weniger und weniger geworden sind, nachdem wir die Fehlerquellen erkennen und zu vermeiden gelernt haben. Dass wir bei einem Arsenmittel von der therapeutischen Machtentfaltung des Salvarsans trotzdem immer an die Möglichkeit übler Zufälle denken und unser therapeutisches Handeln danach einrichten werden, ist ja selbstverständlich. Müssen wir das denn bei den andern Mitteln nicht auch? Ist denn — um nur bei der Syphilistherapie zu bleiben — das Calomel, das graue Oel ungefährlich, und muss nicht auch jede andere Hg-Kur, wenn man höchste Wirksamkeit und Unschädlichkeit paaren will, auf das sorgsamste überwacht werden? Werden wir den Segen des Chloroforms Tausenden und Tausenden von Leidenden vorenthalten wollen, weil hie und da ein Herz einmal dabei versagt?

Damit sind wir zur *Anwendungsweise* des Ehrlich'schen Mittels gekommen, die ja ärztliches Allgemeingut geworden ist und deren Methode, wie sie jetzt von den meisten Therapeuten geübt wird, hier nur in kurzen Leitsätzen skizziert sein mag. Vorausgeschickt sei noch, dass wir heute Salvarsan und Neosalvarsan mit gleich ruhigem Gewissen verabfolgen können. Ist das letztere bequemer und bei richtiger Dosierung noch freier von Nebenwirkungen wie das erstere, so scheint beim Salvarsan die Wirkung rascher einzutreten, für später aber bei beiden die gleich gute zu sein. Ich stimme hinsichtlich der Eigenschaften des Neosalvarsans ganz Touton zu, dessen Arbeit ich erst bei der Korrektur des Referates gelesen. Auch stimmen unsere Methoden sehr überein.[1]

1) *Allgemeine Untersuchung des Patienten,* besonders Herz, Augenhintergrund, Gehörapparat, Urin. Relative Kontraindikationen: Hochgradige Atheromatose, Aneurysma, Vitium cordis, Nephritis. Berufsarten wie Musiker, Maschinisten etc. Ohrkrankheiten, ausgebreitete Drüsenerkrankungen. (Leichte Albuminurie keine Kontraindikation.)

2) Wenn möglich, klinische Aufnahme, bei Neosalvarsan genügt kurze Beobachtung nach der Injektion.

3) Aseptik; frische Destillation des Wassers.

4) *Intravenöse Injektion* in eine Cubitalvene. Bei beständigem Ausweichen sehr dünner Venen: Freilegung, aber selten nötig, Blutauffangen zum Wassermann. (Doppelirrigator für Salvarsan und Kochsalz von Weintraud-Assmy, Strauss'sche Kanüle mit Griffplatte. Verdünnung ca. 0,5 : 150, Kochsalzlösung vor- und nachlaufen lassen.)[2]

5) *Dosierung*: (Bei Lues 1: Exzision des Primäraffekts.) Je nach Gewicht, Konstitution und Alter: *a*) bei mittelkräftigen Männern 0,5 Salvarsan oder 0,6–0,9 Neosalvarsan. (Eigentliches Verhältnis 2 : 3.)

[1] *Anmerkung bei der Korrektur.* Vergl. Touton, *Berl. klin. Woch.*, No. 2, 1913; Gutmann, 'Ueber Parallelversuche mit Alt- und Neosalvarsan,' *Berl. klin. Woch.*, No. 13, 1913; Dreyfus, 'Neosalvarsan,' *Münch. med. Woch.*, No. 12, 1913.

[2] *Anmerkung bei der Korrektur.* Duhot empfahl neuerdings die Injektion einer konzentrierteren Lösung in nur 10 ccm Wasser mit einer einfachen Injektionsspritze von entsprechendem Gehalt. Auch auf diese Weise haben wir wiederholt, bisher ohne störende Nebenerscheinungen, injiziert.

Will man kombinieren, so benützt man die Salvarsanintervalle sofort zur Hg-Behandlung. Also: Die nächsten 5 bis 6 Tage Quecksilberinjektionen oder Inunktionen. Dann:

b) 0,5 Salvarsan, resp. 0,6 Neosalvarsan. Zweite Quecksilberwoche.

c) 0,6 Salvarsan, resp. 0,8 Neosalvarsan. Dritte Quecksilberwoche.

d) 0,7 Salvarsan, resp. 0,9 Neosalvarsan. Vierte Quecksilberwoche.

Zum Schluss, — je nach der Höhe der vorausgegangenen Dosen und klinischem Verlauf — Wassermann etc., evt. noch 1 bis 2 Injektionen. Im ersten Turnus durchschnittlich im ganzen nicht über 2,2 Salvarsan oder 3,0 Neosalvarsan.

6) *Modifikationen.* Bei Lokalisation der Affekte in der Nähe lebenswichtiger Centren, wie oben genannt, ebenso bei spezifischen Gehöraffektionen und den relativen Kontraindikationen: Beginn mit Hg und J oder Einschleichen mit kleinen Dosen 0,2 Salvarsan, resp. 0,25 Neosalvarsan.

M. H. Dieses ist nur ein therapeutisches Schema und wie jedes derartige unter Umständen von Fall zu Fall zu modifizieren. Auch die von den meisten Autoren jetzt empfohlene Kombinationskur, wie sie auch an meiner Klinik zumeist angewandt wird, hat ihre Gegner (Wechselmann, Lenzmann). Werden wir doch schon nicht immer in der Lage sein, unsere Patienten — wie es so ideal in den Militärlazaretten geschehen kann (Gennerich) — so lange in Behandlung behalten zu können. In solchen Fällen werden wir getrost die Injektionen rascher verabfolgen und die Patienten dann mit Hg und Jod nach Hause entlassen können, — gerade solche unsichern Kantonisten mit viel grösserer Aussicht auf Erfolg und völlige Heilung wie ehedem, da wir nur das Quecksilber besassen.

M. H. So von den Schlacken der ersten Heilversuche und den mit ihnen notwendigerweise verbundenen Fehlern und irrtümlicher Auffassung gereinigt, besitzen wir heute in dem Ehrlich'schen Mittel einen therapeutischen Edelstein von alles überstrahlendem Glanze, die über alles Hoffen geglückte Erfüllung der alten Sehnsucht nach einem wirksamen Arsenpräparat gegen die Lues. Was es speziell auf dem Gebiet der Rhino-Laryngo-Otologie zu leisten imstande ist, haben wir soeben gesehen. Gerade bei den Erkrankungen der oberen Luft- und Verdauungswege wirken alle seine glänzenden Eigenschaften zusammen und steigern sich gegenseitig zu den bewunderswürdigen Endeffekten. Diese Eigenschaften sind 1) *Die zauberhaft schnelle Wirkung auf die Sp. pallida.* 2) *Die epithelisierende Wirkung.* 3) *Die Wirkung auf die andern Spirochaeten.* 4) *Die allgemeine roborierende Wirkung.* So haben wir denn die Pflicht, es bei jedem Syphilitiker anzuwenden, bei dem keine Kontraindikation besteht, — *neben* Hg, — *statt* Hg und Jod, wo diese keinen Erfolg haben oder nicht vertragen werden, — statt ihrer auch, wo drohender Gefahr schleunigst vorgebeugt werden soll, Gefahr für den Patienten, — Gefahr für seine Umgebung.

Als willkommene Beigabe haben gerade wir Halsärzte dann noch die ausserordentliche Wirkung des Mittels auf die *nicht spezifischen lokalen Spirochaetosen* der Mund- und Rachenhöhle dankbar zu begrüssen.

Wir wollen nicht den vergeblichen Versuch machen, die Schleier der Zukunft zu lüften. Ob das Salvarsan auch Syphilis*heilend* wirken kann — obwohl jetzt schon mancherlei dafür spricht — das werden erst die wissen, die nach uns kommen. Dass es Organ- und Lebenrettend wirkt, das wissen *wir*! Dank und Bewunderung daher dem Genie und der Arbeit des Mannes, dem das gelungen.

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I

Syphilis

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SECTION XV

RHINOLOGY AND LARYNGOLOGY

DISCUSSION No. 3

DIE INDIKATIONEN UND DER RELATIVE WERT DER TONSILLOTOMIE UND DER TONSILLECTOMIE

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Mit Rücksicht auf den ausnahmslos kompetenten Leserkreis, dem das von mir übernommene Referat zugehen wird, habe ich geglaubt, alle unnötigen Auseinandersetzungen prinzipiell vermeiden zu müssen. Ich habe daher die Thesenform gewählt, welche sich mir bei einer früheren Gelegenheit,[1] als Basis einer sachverständigen Diskussion, wohl bewährt hat.

Die Thesen bringen über die verschiedenen kontroversiellen Fragen meine persönliche Meinung, indem ich jedoch die Existenzberechtigung entgegengesetzter Auffassungen durchaus anerkenne.

Das Verzeichnis der von mir benutzten Literatur erhebt keinen Anspruch auf Vollständigkeit, die ja auf diesem Gebiete schwerlich zu erreichen gewesen wäre. Ich bin mir wohl bewusst, dass manche äusserst wichtigen Arbeiten in diesem Verzeichnis und in meinen Verweisen fehlen. Mehrfach wird man ausführliche Literaturangaben in den von mir zitierten Arbeiten finden können.

Thesen

I. Unsre Kenntnis der physiologischen Bedeutung der Tonsillen ist eine ungenügende.

Weder die phagozythische, noch die Antitoxinbildende (24), noch die eliminierende (3, 113), noch die hämopoetische Funktion der Tonsille, noch ihre innere Sekretion ist einwandfrei festgestellt (46a, 65).

II. Halsbeschwerden, bleibende Stimmstörungen, Schädigung der geschlechtlichen Funktionen infolge von Tonsillenabtragung sind zwar proklamiert, nicht aber wirklich sichergestellt worden. Ausfallserscheinungen, resp. ein pathologischer Status, wie sie nach Entfernung der Schilddrüse, der Ovarien beobachtet werden, sind als Folgen der Tonsillenausschaltung nicht bekannt (11).

III. Ebensowenig aber ist es bewiesen, dass man — namentlich in

[1] *Die Bedeutung der Röntgenstrahlen für die Rhino-Laryngologie.* Referat für den II. Internat. Rhino-Lar.-Kongress, Wien, 1908.

der Jugend — die Tonsillen ohne irgend welchen Schaden entbehren könnte (69, 95).

IV. Die physiologische Bedeutung der Tonsillen dürfte in den Kinderjahren wesentlich höher anzuschlagen sein als im erwachsenen Alter (102).

V. Die Meinung mehrerer Kollegen (14, 29, 31, 49, 70a), dass die Tonsillen überhaupt nur als unnütze, resp. schädliche, die Gesundheit bedrohende Organe zu betrachten seien, ist energisch zurückzuweisen (46a, 69).

VI. *Hypertrophie der Tonsillen* kann Störungen der Atmung, der Deglutition, der Stimme hervorrufen. Sie kann die Gehörorgane gefährden und verschiedenartige Reflexerscheinungen auslösen.

VII. Es ist nicht jede hypertrophische Gaumenmandel als eine obstruierende Mandel zu betrachten. Manche Erwachsene besitzen vergrösserte Mandeln, welche zu Beschwerden niemals Veranlassung gegeben haben (63, 100).

VIII. Bei Hypertrophie des lymphatischen Rachenringes ist in der grossen Mehrzahl der Fälle die Vergrösserung der Rachenmandel klinisch weit wichtiger als diejenige der Gaumentonsillen.

IX. *Chronisch entzündete Tonsillen* geben oft zu einem unangenehmen Fötor ex ore Veranlassung. Sie können hinderliche lokale Beschwerden verursachen und mit einer ganzen Reihe nervöser Reflexerscheinungen (37, 73, 106) in kausaler Beziehung stehen. Sie sind die Brutstätten zahlreicher Mikroorganismen, welche gelegentlich zu chronischer Halsdrüsenentzündung und zu verschiedenartigen Allgemeininfektionen führen können.

Sie sind nicht selten die Ursache wiederholter akuter Entzündungen der Mandeln sowie des peritonsillären Gewebes.

X. Oft ist, bei tiefgehendem Zerfall und Sekretanhäufung bis an die Basis der Mandel, die Oberfläche derselben relativ wenig verändert (8, 103, 113).

XI. Bei chronischer Tonsillitis ist in der Regel *die obere, im Gaumen eingebettete Mandelpartie* am schwersten erkrankt.

Peritonsilläre Entzündungen gehen fast immer von dieser Stelle aus (58, 66).

XII. Die Bedeutung der Mandeln als Eingangspforte lokaler und allgemeiner Infektionen steht mit dem Umfange derselben in gar keinem Verhältnis. Häufig gehen rezidivierende Entzündungen von kleinen versteckten Mandeln aus.

XIII. *Akute Entzündungen* der Tonsillen sowie des peritonsillären Gewebes sind in der Regel, zwar unangenehme, dennoch ungefährliche Erkrankungen.

Sie können indessen mit heftigen, ja tötlichen Komplikationen einhergehen.

Auch können sie — namentlich wenn sie wiederholt auftreten — die Allgemeingesundheit und Leistungsfähigkeit aufs ernste schaden.

XIV. Sie können zu den verschiedensten benachbarten und entfernten *Allgemeinerkrankungen* Anlass geben.

Akuter und chronischer Gelenkrheumatismus (7, 11, 16, 40, 53, 81, 86, 92, 101), Muskelrheumatismus (11), Hautausschläge (79), Herz- (13, 35, 78, 79, 80a) und Nierenerkrankungen (20, 58a), eitrige Infektionen (6, 11, 22, 23, 25, 27a, 76, 84), Appendicitis (1, 2, 34, 61, 62, 72, 108, 109), Orchitis (28, 82), Peritonitis (76a), Poliomyelitis (27) und viele andern Affektionen können mit der Angina tonsillaris in kausalem Zusammenhang stehen.

XV. Am sichersten ist diese Beziehung für den *akuten Gelenkrheumatismus* festgestellt worden.

XVI. Wenn auch die primäre *Tuberkulose* der Tonsille eine Seltenheit darstellt, so ist es sehr wahrscheinlich, dass eine erkrankte Tonsille für die tuberkulöse Infektion, zunächst diejenige der Halslymphdrüsen, die Eingangspforte sein kann (5, 6, 15, 36, 57, 59, 60, 88, 105).

XVII. Irrigation der Mandellakunen scheint in den Händen mehrerer Kollegen eine erfolgreiche *konservative Behandlungsmethode* der chronischen Tonsillitis zu sein (91, 94, 97, 111).

Druck- und Quetschmethoden zur Herausbeförderung von Tonsillenpfröpfen sind nur wenig empfehlungswert.

Schlitzung der Mandeln, ohne Exzision von Mandelfragmenten hat nur sehr beschränkten Wert.

XVIII. *Die Tonsillotomie* — i.e. die Verkleinerung der Tonsille — kann mit dem Tonsillotom, dem Messer, der kalten oder der glühenden Schlinge oder mit der schneidenden Zange gemacht werden.

Die Wahl des Instrumentes wird von den Verhältnissen jedes gegebenen Falles, sowie von der Vorliebe des Operateurs bestimmt.

Die Meinung, dass die Tonsillotomie mittelst Tonsillotoms grössere Blutungsgefahr ergebe als diejenige mit sonstigen Instrumenten (11, 18, 27a), ist unberechtigt (21, 28).

Bei der galvanokaustischen Schlinge ist die unmittelbare Blutungs gefahr verringert, die Gefahr der Nachblutung dagegen erhöht (11, 12, 64).

Einfache Galvanokaustik steht, als Mittel zur Verkleinerung der Tonsille, den blutigen Methoden nach. Sie kann von Abschliessung von Krypten und dadurch erhöhter Gefahr vor sekundären Infektionen gefolgt sein.

XIX. *Die Tonsillotomie* bei Kindern bedarf weder allgemeiner noch lokaler Anästhesie. Bei Erwachsenen genügt einfache Cocainpinselung.

Die Operation kann in ambulanter Behandlung ausgeführt werden.

XX. Die Lebensgefahr ist bei der *Tonsillotomie* eine ganz minimale.

XXI. **Indikationen der Tonsillotomie.**

1. *Hypertrophie der Mandeln*, welche überhaupt eine Behandlung verlangt.

2. *Rezidivierende, akute Tonsillitis und Peritonsillitis* bei Kindern, wenn die lokalen Verhältnisse die Operation überhaupt gestatten.

3. *Tonsillitis chronica*, welche zu akuten Exazerbationen nicht geführt hat, wenn die lokalen Verhältnisse die Operation gestatten.

In diesen Fällen kann zuerst auch rein konservativ vorgegangen werden (46, 48, 91, 94, 97, 111).

XXII. *Die Tonsillectomie* ist *technisch* eine weit schwierigere Operation als die Tonsillotomie (55, 100).

XXIII. *Die Tonsillectomie* ist eine *eingreifendere Operation* als die Tonsillotomie.

Sie geht mit grösserem Blutverlust einher (11, 17, 19, 28, 43, 69, 100, 110; umgekehrt: 15, 30, 35a, 43a, 55, 75, 98 u. A.) und wird von wesentlich bedeutenderer entzündlicher Reaktion gefolgt (35a, 70, 80).

XXIV. *Die Tonsillectomie* ist quoad vitam wesentlich *gefährlicher* als die Tonsillotomie (69).

Eine Anzahl ernster Blutungen sind in der Literatur verzeichnet, und die wirkliche Anzahl solcher Fälle ist zweifelsohne noch bedeutend grösser (12, 50, 58a).

XXV. Indessen ist in der grossen Mehrzahl der Fälle von unmittelbar oder in den ersten Tagen nach der Tonsillectomie auftretender *Blutung* dieselbe durch einfache Kompression, resp. Tamponnade, zu stillen.

Arterielle Blutung ist relativ selten. Meistens lassen sich dabei die blutenden Gefässe mit der Pinzette fassen.

XXVI. Es kann aus jeder Stelle des Operationsgebietes bluten. Am wichtigsten in dieser Beziehung ist *die untere, der Zungenbasis angrenzende Mandelpartie.*

XXVII. Bei Tonsillenoperationen *in Narkose* sind die Gefahren der Narkose weit grösser als diejenigen der Operation an und für sich.

Die meisten Todesfälle, sowohl nach der Tonsillotomie wie nach der Tonsillectomie, sind gänzlich oder teilweise der Narkose verschuldet (56).

XXVIII. Die Tonsillectomie soll vorzugsweise *in lokaler Anästhesie* gemacht werden (9).

Novocain-Adrenalin-Infiltration ergibt in den meisten Fällen eine ausreichende Anästhesie.

Bei Kindern und bei sehr nervösen Erwachsenen ist meistens *die Narkose* nicht zu umgehen.

Lachgasnarkose (39) und sonstige kurzdauernde Betäubungen sind für die Tonsillectomie nicht zu empfehlen.

XXIX. Mit Rücksicht auf die häufig nicht unbedeutenden, venösen Blutungen und die entzündliche Reaktion ist es ratsam, die Tonsillectomie nur *in klinischer Behandlung* vorzunehmen (26, 28, 54, 104).

XXX. Als anatomisches Endresultat der Tonsillectomie können die beiden Gaumenbögen und die Fossa tonsillaris erhalten bleiben. In der Mehrzahl der Fälle findet eine Verwachsung des vorderen und des hintern Bogens statt unter Obliteration der Fossa. Auch kommt es vor, dass der hintere Bogen sich der lateralen Rachenwand nähert, woraus in der Regel ein Tiefstand des betreffenden Bogens resultiert.

Auch ist die Bildung von Taschen durch Narbenstränge beobachtet, welche zu Retention von Speisen Veranlassung geben können (80).

XXXI. *Gaumenlähmung* wird zwar unmittelbar nach der Operation verhältnismässig häufig beobachtet; jedoch scheint dieselbe ausnahmslos in kurzer Zeit wieder zu verschwinden (10).

XXXII. *Die Resultate der Operation* bei chronischer Tonsillitis mit akuten Exazerbationen und Allgemeinerscheinungen sind umso sicherer, je gründlicher alles Mandelgewebe entfernt worden ist.

Absolute Gewähr gegen spätere Anginen und deren Folgezustände vermag auch die totale beiderseitige Tonsillectomie nicht zu ergeben (68).

XXXIII. Das Zurückbleiben kleiner Mandelpartien, namentlich im untern Teil der Mandel, wird in der Regel den Erfolg der Operation nicht beeinträchtigen.

XXXIV. Weil in der Regel *der obere Pol der Mandel* am intensivsten erkrankt ist, soll diese Partie immer gründlich abgetragen werden (66).

Dahingegen dürfte es sich unter Umständen empfehlen, an der Zungenbasis eine kleine Mandelpartie stehen zu lassen (37, 65, 71, 102).

XXXV. Für die *Freilegung des oberen Mandelpols* empfiehlt sich die Einschneidung des vorderen Gaumenbogens bis zum Fundus der Fossa supratonsillaris (31, 33, 87, 90).

XXXVI. *Bei Sängern* soll für die Tonsillectomie eine strengere Indikation gestellt werden wie sonst. Der Einfluss dieser Operation auf die Singstimme ist oft ein vorteilhafter, kann aber auch ein dauernd ungünstiger sein (28, 44, 58a, 69, 80, 81a, 84).

XXXVII. **Indikationen der Tonsillectomie.**

1. *Bei Erwachsenen: Wiederholte akute Tonsillitis oder Peritonsillitis.* Die Tonsillectomie ist umsomehr angezeigt, wenn diese Entzündungen zu Allgemeinerkrankungen — Rheumatismus, Appendicitis, Nephritis u.s.w. — Veranlassung geben.

Bei Kindern gilt diese Indikation nur, wenn die lokalen Verhältnisse die Tonsillotomie nicht gestatten (kleine, versenkte Mandeln).

2. *Bei Erwachsenen: Tonsillitis chronica* ohne akute Exazerbationen, wenn Operation überhaupt angezeigt und die Mandel nicht hypertrophisch ist.

Bei Kindern nur wenn die Tonsillotomie, namentlich auch diejenige mit der schneidenden Zange (Morcellement), nicht ausführbar ist.

3. *Tuberkulose* der Mandel, wenn der Prozess auf die Mandel beschränkt ist.

4. *Maligne Neubildung* der Mandel (27a, 43a).

Der linguale Mandelfortsatz sowie die Gaumenbögen sollen zusammen mit der Mandel abgetragen werden (43a).

Nachfolgende Kauterisation erhöht die Heilungsaussichten (74).

In vielen Fällen, namentlich bei Carcinoma, werden ausgedehntere Operationsverfahren notwendig sein.

5. *Gutartige Neubildung* der Mandel, wenn die einfache Abtragung der Geschwulst auf Schwierigkeiten stösst (27a).

6. Trotz energischer spezifischer Behandlung hartnäckig auf die Tonsille lokalisierte *Lues* (100).

7. Hartnäckig sich in die Länge ziehende *Angina Vincenti* (83).

In den Fällen 3 bis 7 wird die Indikation nur ausnahmsweise zur Geltung kommen.

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SECTION XV

RHINOLOGY AND LARYNGOLOGY

DISCUSSION No. 3

INDICATIONS FOR AND RELATIVE VALUES OF TONSILLOTOMY AND TONSILLECTOMY

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THE subject of our conference seems for a variety of reasons opportunely chosen. In the first place, our conception of the relation of the tonsils to the rest of the system has within recent years become expanded from one chiefly of a hypothetical function and of local pathology to one including also important problems of general infection. Coincident upon a recognition of its importance in this respect, there has necessarily followed a change in our former ideas with reference to its removal, and the frequent failure of tonsillotomy to accomplish the results desired has led naturally to the operation of tonsillectomy. The sudden illumination afforded by this procedure in many previously dark corners of ætiology has naturally attracted a rush of wanderers seeking the right path, and in the impetuosity of the search there seems to have occurred not a few instances of aberration beyond the rays of light into new regions of obscurity. Whether such aberrations are more frequent in America than elsewhere I cannot say, but that they exist on both sides of the Atlantic is only too evident from the warnings of many observers, as well as from the personal experience of most of us. Makuen, J. H. Pearson, W. S. Syme, C. W. Richardson, J. N. Mackenzie, and Milligan, among others, have shown the functional ill-effects, dangers, and anatomical distortion from improperly performed operations of tonsillectomy. When, however, we consider that this operation occupies at times a highly important position, and also that it may be done without essential danger or anatomical injury, it certainly behoves us to determine when and how it may be best performed.

A comparison of the relative advantages of tonsillotomy and tonsillectomy involves logically first the determination of the question whether the complete excision of the tonsil is likely to prove in any way a loss to the system. I shall not attempt here to discuss this subject, but I will simply enumerate some of the reasons justifying the assumption that the system may dispense with this organ without essential detriment.

First, if we believe that the tonsils furnish something of value to the body, we should logically admit also the existence of a similar function

in the other histologically identical aggregations of lymphoid tissue in Waldeyer's ring. Consequently, on theoretical grounds at least, the removal of the tonsils during their period of activity should, if necessary, be followed by a compensatory increase of function, whether or not demonstrable clinically, in the neighbouring lymphoid structures.

Furthermore, in adult life retrograde metamorphosis of the tonsil frequently proceeds to such an extent that but few traces of lymphoid tissue may be detected, the greater portion of the organ being represented by fibrous tissue.[7] A progressive atrophy of the lymphoid tissue is manifestly taking place, and a mechanical removal of the organ will only anticipate the process of nature.

Finally in the last few years the tonsils have been excised both in children and in adults by the majority of laryngologists without, so far as reported, the establishment of any ill-effect upon the system from the extirpation itself. The question of influence upon the voice is here expressly excluded and will be considered later. While we should deprecate the unnecessary excision of this as of any other organ, yet it seems to me that until definite evidence is forthcoming from experienced observers that they have observed harmful effects to follow their tonsil enucleations, we may in the future continue to perform the operation of tonsillectomy, if otherwise indicated, without fear of systemic injury.

If we admit the possibility that the complete removal of the tonsils does not in itself constitute an irreparable loss to the system, and if we acknowledge that a tonsillotomy often fails to accomplish the result desired, we may ask why tonsillectomy is not always the operation of choice. I shall endeavour to present the indications for and relative advantages of the two operations by a study of the question first from the surgical or technical point of view, and second from the standpoint of ultimate advantage accruing to the patient.

The surgical aspect of the questions involved comprehends the following chief points :

(*a*) Trauma.

(*b*) Septic complications.

(*c*) Hæmorrhage.

(*d*) Alteration of the anatomical relationship of the parts involved.

(*a*) With reference to the relative amount of trauma and subsequent inflammatory reaction resulting from the operations under consideration, so many methods are in use by different operators that attempts at comparison would involve us in hopeless confusion if we did not first reach some sort of agreement as to what constitutes an ideal operation. Without attempting to cover the many ingenious methods devised by our colleagues, I shall endeavour to define what appears to me the preferable methods of procedure.

With reference first to tonsillotomy, the ideal operation removes as much of the tonsil tissue as possible, without injury to the pillars, and leaves an area of lymphoid tissue to be covered in by granulations. This

procedure is accomplished most typically by the various amygdalotomes, of which the knife moving in one plane leaves a flat more or less circular wound, represented by lymphoid tissue attached to the capsule of the organ. Consequently the area of trauma and liability to subsequent deformity are reduced to a minimum. Unfortunately in the case of imbedded tonsils these instruments are of little use, and the operator is obliged to employ punches to pass between the pillars and reach the lymphoid tissue. Although such instruments should not increase the risk of deformity, yet since the area of a hemispherical concavity is greater than that of a circle, the region of trauma is unavoidably extended, with consequent increase of post-operative inflammation.

Tonsillectomy may be done in a variety of ways, distinguished by the manner in which the capsule is separated from its surroundings. We may divide the methods for operation into two classes, namely those in which the excision is performed chiefly by the use of relatively blunt instruments, and those in which the excision or the greater portion of it is accomplished by sharp knives or scissors. Experience has shown that the amount of inflammatory reaction subsequent to the operation depends to a great extent upon the character of the surface left. In general the reaction is proportionate to the extent of necrosis supervening in the wound, particularly in the muscular tissues. Since the necrosis is increased by overstretching and crushing of the parts, the manner of handling the instruments is of primary importance, and two operators with the same general technique may achieve entirely different results so far as the subsequent discomfort of the patient is concerned. Where the finger is used to aid dissection the type of digit possessed by one operator may be as different from that of another individual as a tenotomy knife is from a periosteal elevator. In one of our Boston clinics a woman surgeon uses the finger dissection, leaving as clean and comfortable a wound as could be expected by any method. For many surgeons the finger operation exposes the patients to unnecessary trauma. While it is true that the use of a sharp instrument is less likely to be followed by reaction than a dull one, yet on the other hand, the cutting of vessels by a sharp knife is more likely to be followed by troublesome hæmorrhage than if these are broken, twisted, or cut with a dull instrument. I have therefore taken as the ideal tonsillectomy those methods by which the tonsil is dissected from the pillars and from the surrounding tissues by such instruments as will produce a minimum of trauma down to the point where the tonsillar artery enters, this spot being then cut through by the aid of a dull instrument such as a snare. It is of course assumed that the vessels in the pillars, so frequently the source of troublesome hæmorrhage, are left intact.

The question of technique is so important in endeavouring to reach a conclusion regarding the comparative advantages of the two operations, that statistics derived from published records are of little value, unless each reporter describes in detail the method which he employs. For this reason I shall describe the method which, after trying most of those in

use, I have found to give the best results. After a hypodermic injection of morphine and atropine, proportionate to the patient's age, ether introduced by nitrous oxide is administered to a point immediately beyond the abolition of reflex action when the throat is touched. It is important not to exceed this degree of anæsthesia to any marked extent, as we wish the patient's control over coughing and swallowing to be within prompt reach when operative bleeding begins. When the proper stage of anæsthesia is reached, the gag is introduced on the right side by a good illumination from a headlight, the patient being in a sitting position, and the left cheek drawn outward to the left by the etherizer. The prepared snare is passed over the tonsil forceps and allowed to hang free, while the forceps seize the right tonsil and draw it slightly out of its bed. A small sharp tenotomy knife performs the dissection from above downward, the cutting edge being used at first, and often later, as occasion requires, the back of the point, in order to avoid cutting the pillars or the lymphoid tissue itself.

The dissection proceeds rapidly down to the point where the tonsillar artery enters, when the tonsil is lifted upwards, and a horizontal cut is made, delimiting the organ from the lingual lymphoid tissue. The snare, which in the meantime has been hanging free, has now only to be slowly closed to sever the region of the artery and the tonsil comes away in the forceps. The procedure is then repeated for the other side. The size and shape of the knife blade are of the first importance, only a small one which can be easily turned in the hand and made to cut as described front or back meeting the requirements. The hypodermic injection of morphine and atropine serves the purpose of keeping the field of operation dry, and diminishing subsequent pain. The headlight permits accurate manipulation of the knife, and renders one independent of the varying conditions of daylight and of the presence of spectators. My reason for giving ether in preference to local anæsthesia has been chiefly the comfort of the patient, and also the apparent diminished tendency to post-operative bleeding. In several thousand cases at the Massachusetts General Hospital, at the Boston Children's Hospital, and in private practice, I have seen only one case of broncho-pneumonia, during the past eighteen years, following tonsil operations under ether in the upright position. This case was a mild one in a child, and could be explained on other grounds than inhalation.

(*b*) An examination of the literature shows a greater frequency of septic complications following tonsillectomy than had been previously recorded for tonsillotomy. In 1912 the following complications were gathered by C. W. Richardson in a review of the literature :

Hyperpyrexia with fatal termination and infarct of the lung appear to be rare. Mild general sepsis is, on the other hand, frequent. Severe sepsis seems uncommon so far as the number of reported cases indicates. Emphysema of the neighbouring tissues was found in two cases. Infection of the lungs and serous membranes is not uncommon. Torticollis attributed to sepsis has been noted in three cases.

In the consideration of the relation of sepsis to tonsil operations, the important question arises first whether the increased frequency of late years is attributable to changed methods of operating or to the fact that we are now removing tonsils more often than before for conditions which in themselves favour the occurrence of systemic infection. For many operators it is true that a far greater amount of trauma and inflammatory reaction follows their present operations of tonsillectomy than occurred after their earlier tonsillotomies. It must also not be forgotten that we are to a much less extent than formerly concerned chiefly with the removal of simple hyperplasia. We are now asked to operate upon tonsils which are themselves foci of infection, and which in the earlier days would not have been treated surgically. So far as the literature is concerned it is manifestly extremely difficult to determine from a description of the method employed precisely how much trauma is occasioned by a given operator, and yet the importance of this point cannot be too highly estimated. In no other region of the body do we find such an association of conditions favouring sepsis, namely a surface bathed in a fluid open to the reception of every chance pathogenic micro-organism, and a surface moreover which cannot be put to rest after the operation. While it may be unjust to read into the published accounts of septic complications an unnecessary amount of operative trauma, yet my own observations regarding the inflammatory reactions occasioned by student operators at the Massachusetts General Hospital lead me to believe that an improvement may be possible in the methods of their elder brethren.

So far as the second point is concerned, the removal of tonsils which are in themselves essentially septic has in my experience been followed by greater inflammatory reaction than occurs in cases of simple hyperplasia. This reaction seems often unavoidable, and for these cases, therefore, I have thought it especially necessary to operate with care, to avoid stretching of the parts, and to leave the wounded surface as clean and smooth as possible.

It may here be in place to record that I have yet to see an instance of septic complications following tonsil operation involving the ears or other parts of the system, beyond one doubtful case of broncho-pneumonia and two of exacerbation of previously existing infectious endocarditis. How much this good fortune is attributable to chance and how much to the use of sharp instruments for dissection is impossible to say, but I cannot help believing that the latter has been a considerable factor in the result.

(*c*) With reference to post-operative hæmorrhage it appears that local causes are most frequently from the tonsillar artery or from vessels in the pillars if these are wounded. The statistics of operators differ widely in this regard, some reporting that they have never had a hæmorrhage, while with others hæmorrhage has been so frequent that sewing of the pillars together is practised as a precautionary measure. In my own experience of twelve years' tonsillotomy work with the guillotine,

punch, and snare, I can remember only two cases of post-operative bleeding that required treatment of any kind; while in the last six years, where tonsillectomy has been the rule, I have seen at least ten instances of troublesome hæmorrhage, in which it has seemed best to suture the pillars. In these cases the bleeding has come from the tonsillar artery so far as could be determined. I have not seen hæmorrhage from the vessels in the pillars so frequently mentioned. When the bleeding once begins a clot is apt to form in the recess left by removal of the tonsil which keeps the vessels from undergoing retraction. I have on three occasions found the bleeding to stop promptly after removal of the clot without additional treatment.

With our present methods of suturing the pillars, and with the pliable hæmostatic clips, recently introduced, I believe that ample provision is available for the slightly increased risk of hæmorrhage following tonsillectomy, so that so far as this possibility is concerned, one may feel entirely free to choose whichever operation is indicated by the other circumstances of the case in question.

(*d*) With reference to the conservation of the anatomical relationships of the parts involved, tonsillotomy by its nature should produce no gross deformities, and yet it is frequently the cause of a serious result, namely the narrowing or occlusion of the lacunar orifices which are left in the stump of the tonsil. In a certain percentage of cases, I have found that my patients upon whom tonsillotomy had been performed reported after a lapse of one to several years with symptoms denoting chronic absorption of decomposing material in the crypts. Examination showed that the orifice of one or more crypts had been occluded, evidently by cicatricial contraction after the operation, with the resulting accumulation of detritus in the cavity.

The operation of tonsillectomy has been reproached for the extent of deformity which frequently supervenes. The removal of the tonsil and its capsule involves a larger area of wounding than does tonsillotomy, and in numerous instances the adhesions between the tonsil and its surroundings are so firm as to render a separation extremely difficult. Consequently the pillars may be cut or torn away, or they may become fused, or a stellate cicatrix may occur with resulting puckering and retraction of the parts. Such alterations of structure may have harmful consequences for voice production. Even if no such ill-effects are noted, still the anatomical distortion produces a feeling of dissatisfaction at least to the examiner. Such reproaches have certainly been justified in the past by the results which have been frequently seen. It is not, however, necessary that any deformity should occur. Among our best operators I do not find it, and I think you will agree with me that what some laryngologists can do we should all be able to do, or else we should withhold our hand from the operation of tonsillectomy.

In passing now to the consideration of the ultimate advantage accruing to the patient from the adoption of one or the other of the two procedures,

a clarification of our ideas will be favoured by bearing always in mind the precise object which we seek to attain.

In the first place, we shall undoubtedly agree that no operation shall be undertaken unless we can reasonably expect an improvement in a present undesirable condition or a restriction of harmful future occurrences.

I use here intentionally the words 'undesirable' and 'harmful' rather than 'abnormal', because I believe that much unnecessary surgery of the tonsil owes its origin to an exaggerated conception of the importance of correcting pathological and yet not necessarily injurious alterations. To an especial degree the lymphoid tissue of the fauces undergoes changes in response to influences from within and without, and imposes upon us the task of distinguishing between such as do not conflict with the welfare of the patient, and those which are to a greater or less extent detrimental.

Let us consider what alterations of the tonsils require surgical intervention. With the omission of neoplasms, for our present purpose these alterations may be comprised under the headings of harmful enlargement, chronic inflammation, and tendency to bacterial infection. These conditions may exist singly or in association.

First, what constitutes harmful enlargement? Here we have been especially prone to error, and there are few of us, I imagine, who have not, at some time, deposited upon the operating table tonsils of impressive size, which *in situ* have offended our sense of proportion, and if asked how the patient will be helped by the operation, we should have found it difficult to define precisely the manner of benefit.

The disturbances arising from simple hyperplasia are chiefly mechanical in nature, such as faulty articulation and tubal closure. Nevertheless patients are continually seen with marked tonsillar enlargement who show none of these symptoms or any others referable to the tonsils, and who are furthermore in vigorous bodily health. Such cases lead to the conclusion that tonsillar enlargement in itself is not necessarily injurious to the individual. If, however, the hypertrophy is associated with mechanical obstruction, we have first to inquire whether it is likely to be persistent or whether it will diminish within so short a space of time that we can wisely wait for this subsidence to occur.

A study of the natural process of development and retrograde metamorphosis of the faucial tonsils is here of the first importance. If we indicate upon a chart these changes, we shall find in the majority of individuals a line resembling the temperature curve of a fever, characterized by sudden onset, and gradual lysis. From birth until about the age of six, a rapid increase occurs in the number and size of the lymphoid units, which remain essentially unchanged until puberty, when a diminution supervenes, leading to a progressive shrinking of the organ, as old age is approached. During this whole period the lymphoid tissue experiences increase in size in response to various stimulations.

Such stimulations may be physiological, as in the enlargement of the tonsils accompanying the four periods of molar eruption,[8] namely at two, twelve, and seventeen years of age, or pathological and dependent upon the toxines of acute and chronic infections. In certain instances, for reasons that are not yet clear to us, the tonsils fail to undergo their normal process of involution and remain enlarged into middle and old age. In the examination of the tonsils we have therefore first to distinguish between those hypertrophies which are likely to be transitory, and those in which no material change can be expected. We may in the case of children reasonably look for a diminution in the size of the hypertrophied tonsil, the more so if the examination is conducted at a time when dentition is active. Furthermore, both in children and in adults, if the examination is made during or immediately subsequent to the occurrence of an acute infectious disease, much of the enlargement may be assigned to the temporary stimulation of the lymphoid tissue by the toxic influence of the affection. We must therefore discount, as occasion requires, the actual enlargement present, and ask ourselves how much of it is due to a transitory increase in the lymphoid units.

In such conditions of uncomplicated simple hyperplasia, the problem is purely a mechanical one, and involves only the removal of a sufficient amount of surplus tissue to free the fauces from the existing obstruction. A tonsillotomy is therefore theoretically adequate and is in practice usually sufficient. In early life, however, the influences which stimulate lymphoid proliferation may be so active as to effect a considerable subsequent enlargement of the organ, requiring eventually a second operation.

While in simple hyperplasia I do not believe that it is essential to excise all of the organ, nevertheless, if, however, it is the experience of a given operator that his method of enucleation produces a negligible increase of trauma, pain, and risk over a partial removal, it seems to me that he may be justified in employing tonsillectomy even in such cases. In my own experience, it is these very cases of simple obstructive enlargement which show the least difference between the two operations as regards trauma and risk, if the tonsillectomy is done by sharp dissection, with subsequent snaring of the large vessels. I am consequently led more and more to adopt tonsillectomy in all cases where I believe an operation to be indicated, but I should be loath to recommend this practice to all operators, and for those whose manipulation is clumsy or whose technique faulty, I should feel that a tonsillotomy should be here the method of choice, so far as it was in any way permitted by the circumstances of the case.

We come now to a consideration of the second cause for removal, namely chronic inflammation with retention of lacunar detritus.

At the meeting of the British Medical Association in 1903, the discussion upon this subject in our section showed that we had in the past sinned from errors of operative omission even more than in conditions

of simple hyperplasia from errors of commission. The difficulty of recognizing chronic sepsis is frequently considerable, as where concealed collections of detritus exist in tonsils of normal outward appearance. This class of cases calls always for treatment or for surgical intervention.

In these conditions a partial removal is most frequently inadequate. To be sure, a marked improvement may be expected, inasmuch as under favourable circumstances the long, more or less tortuous crypts, become shallower, and with consequently a smaller epithelial surface, so that they may be theoretically considered to exhibit less surface for infection and greater ease of cleansing during swallowing. Nevertheless a tonsillotomy is here especially apt to be followed by the cicatricial narrowing of the lacunar orifices already referred to, leading at times even to a greater accumulation of detritus than originally existed. While a tonsillotomy is therefore usually inadvisable in these cases of lacunar retention, it must not, on the other hand, be assumed that a tonsillectomy is our only alternative. Many tonsils occur of essentially normal size with one or two occluded crypts, which can be readily opened by scissors or a right-angled knife, so as to establish free drainage. If such a tonsil is in other respects harmless, and not abnormally subject to bacterial infection, a tonsillectomy would appear unnecessary. On the other hand, if the crypts are tortuous and contain much débris, especially if the tonsil is at the same time enlarged, incisions into the crypts are generally a waste of time, and it is better to proceed at once to tonsillectomy.

Our third cause for tonsil operations is found in a tendency of the organ to bacterial infections. Such infections may be acute, as in the pyogenic inflammations, or chronic, as in tuberculosis of the organ.

In the case of acute infections we have first to distinguish between occasional ones arising in virtue of a specially virulent type of micro-organism and those infections which show a predisposition to recurrence, dependent upon local or systemic weakness on the part of the host.

Of these, the first class is represented most typically by infections due to virulent strains of streptococci and the bacillus of diphtheria.

These may involve both the tonsils, whether or not previously diseased, and the follicles of the adjacent mucous membrane.

In a recent extensive epidemic in Boston of septic sore throat from milk infection, with numerous fatalities, I had opportunity of observing primary involvement of the pharyngeal follicles in patients from whom the tonsils had been previously enucleated, followed by severe general sepsis, and in one case by death. This epidemic involved individuals irrespective of age and bodily vigour.[9]

Such cases do not, therefore, enter into our discussion of operative procedure.

In the second class of acute infections, namely those showing tendency to recurrence, we distinguish :

(1) A primary vulnerability or diseased condition of the tonsils most frequently represented by chronic lacunar tonsillitis, favouring either

local acute inflammation as acute proliferative tonsillitis, intrafollicular and circumtonsillar abscess, or permitting the entrance of the infecting agent into the general system, and producing a variety of disturbances, such as endocarditis, nephritis, and arthritis. These types of tonsils call always for complete excision, when possible. We have here to consider a highly important point which merits full discussion, namely, in conditions of recurrent acute inflammations of other regions, such as the endocardium, the kidneys, or the joints, characterized by symptoms pointing to the tonsils as the probable infection atrium, how shall we determine when the safe and most favourable moment has arrived to perform the tonsillectomy? With reference to acute articular rheumatism and to the more or less chronic forms of infectious arthritis, I have in many cases removed the tonsils within a few days after subsidence of the acute joint manifestations without in any instance observing either local or remote ill-effects. The recommendation was even made at the Sixteenth International Medical Congress to remove the tonsils for acute articular rheumatism during the attack.[10] While the expediency of this advice may on theoretical grounds be questioned, we may at least assume that no long delay is necessary.

In the few cases of recurrent acute nephritis with supposedly tonsillar origin which I have seen, the tonsils were removed several weeks after the urine had become essentially normal without exciting renal irritation.

In recurrent infectious endocarditis, where the tonsils have been excised soon after the subsidence of acute cardiac symptoms, I have twice seen a lighting up of the process causing considerable anxiety.

I believe in this latter class of cases great care should be exercised not to operate until a competent internist has pronounced both the cardiac and the general condition sufficiently satisfactory.

The question of operating if elevation of the temperature exists cannot be answered by establishing a fixed rule. In many instances of recurrent general infection the temperature is maintained as the result of absorption of toxine from the tonsil such as occurs in some forms of chronic tonsillitis, with retention of lacunar detritus, and in tuberculosis of the organ. Such conditions require removal, but if the temperature cannot be thus explained, it is preferable not to operate until a reasonable time after it has completely subsided.

(2) A primary vulnerability on the part of the system to the various acute catarrhal infections. Here a tonsillectomy is usually indicated, if the tonsil shows pre-existing chronic inflammation. We are, however, frequently asked to operate upon tonsils for the purpose of preventing sore throats in the individual, and if we fail to remember that the tonsils form but a part of a penetrable lymphoid ring we may be involved in the subsequent disappointment when, in spite of a careful tonsillectomy, the sore throats nevertheless continue to appear. Especially in children with enlarged pharyngeal follicles and in adults with lateral pharyngeal

hypertrophy, severe types of sore throat occur, even after the most careful excision of the tonsils.

In both these classes a tonsillotomy may be helpful, but it is in general inadvisable. We have probably all seen patients in whom a tonsillotomy has been done, who after the operation have been more subject to tonsillitis and even circumtonsillar abscess than before, in whom an investigation has shown contraction of the lacunæ with retention of detritus. In persons of rheumatic tendencies, particularly, such subsequent tonsillitis is prone to occur even when hardly more than a trace of tonsil tissue is left.

It is, therefore, evident that acute infections of the fauces depend upon the various factors of the individual's resistance to infection, the presence of penetrable pharyngeal lymph-follicles, and virulence of the micro-organism, as well as upon the capacity of the tonsils themselves for admitting infection.

Of the chronic infections of the tonsils, primary tuberculosis is chiefly conspicuous as requiring surgical treatment. This condition is usually accompanied by a similar invasion of the cervical lymph-nodes, and not infrequently involvement of the adenoid. Here a thorough excision of the tonsils and adenoid should be performed previous to any operation upon the lymph-nodes of the neck, in order to close as far as possible this portal of entry.

Tonsillectomy has been advised in primary syphilis of the organ, and in persistent Vincent's angina, but of its advantages in these conditions I have no personal experience.[11]

The question frequently arises in the case of children with adenoid hypertrophy requiring removal whether the tonsils shall also be excised, even if not demonstrably causing disturbance. We may be influenced to take this course for several reasons. For instance, if the tonsils are left and become later the source of trouble, we may incur criticism from the parents for not having removed them at the time of the adenoid operation. This point is worthy of full discussion, but I shall content myself with a statement of what has seemed to me the wisest course to pursue under such circumstances. I believe that it is usually best to leave the tonsils where not demonstrably injurious, where simple, obstructive hyperplasia of the adenoid exists.

In infants and young children the lymphoid tissue of the throat has not yet reached its maximum stage of development, and a later operation upon the adenoid may be necessary, at which time the tonsils will probably have declared their character and can be removed if found to be injurious. It is important to explain these facts fully to the parents, who should be instructed to bring the child regularly for examination, at least once a year, for as long a period as may be necessary.

However, in children above three or four years of age, where an adenoid operation is contemplated chiefly for the sake of recurrent inflammation involving the middle ears, I believe that it may be wise to remove also

the tonsils, even if they are not definitely abnormal. Here we may assume the existence of systemic predisposition to pyogenic infection, or the presence of ready penetrability on the part of the lymphoid tissue of the fauces. Hence the removal of the tonsils with the adenoid may aid materially in accomplishing the object desired, namely the prevention of future attacks of acute otitis media. The infecting organisms may, of course, continue to find entrance through the follicles of the pharynx, but to a less extent, so far as my experience enables me to judge.

In considering the relative influence upon the voice of a partial and of a complete removal of the tonsils, we have in the first place to consider the part played by the soft palate and by the normal tonsils in phonation. As Makuen has shown, the soft palate has two important functions in voice production, one being a valvular function influencing vocal resonance, the other a power of tilting the thyroid cartilage and stretching the vocal cords, thus modifying the quality and pitch of the voice. The normal tonsil keeps the pillars apart and may theoretically be assumed to improve the vocal resonance. Under conditions of tonsillar hyperplasia the valvular action of the soft palate is impeded, while the quality and pitch are impaired.

'The muscles surrounding the hypertrophied tonsil are crowded out of their normal position and therefore they lack precision of action, and the resonance of the voice is impaired in proportion to the size of the tonsillar mass, and its interference with the valvular action of the palate.

The muscles forming the pillars of the palate instead of being quite straight in their vertical planes of action are bowed, the one anteriorly and the other posteriorly, to a degree corresponding to the size of the tonsil, and even moderately large tonsils sometimes so displace these muscles and limit their action as to keep the soft palate from rising, thus entirely preventing the performance of its valvular function during phonation, and as a result there remains an open channel from the oral cavity into the nostrils, giving undue nasality to the voice. Not only so, but the pressure upon these depressor muscles of the palate and their resultant sluggish action serve to put all the muscles of the palate in a more or less paretic condition, and we have the so-called relaxed palate, the result of which is always more or less evident in the strongly muffled character of the voice, and in its limited range of pitch, for owing to the laryngeal functions of the palato-pharyngii muscles, the quality and pitch of the voice cannot be controlled with any degree of accuracy when the soft palate is relaxed.' (Makuen.)

Chronic inflammatory conditions of the tonsil may have an unfavourable influence upon the voice both on account of the adhesions which usually coexist in the vicinity and also in virtue of the tendency to catarrhal pharyngitis and laryngitis.

While we should not expect to find a voice of extremely fine quality in an individual who has either obstructive hyperplasia or chronic inflammation of the tonsils, we must, for practical purposes, distinguish between the ordinary individual whose voice is used solely for speaking

and the singer. Since in the former class of cases comparatively extensive departures from the normal may exist in the tonsils without sufficient influence upon the voice to annoy its possessor, it is not often for this reason alone that we are called upon to operate. Our indications for removal here should follow the lines already discussed. I have myself not observed any ill-effect upon the speaking voice from a carefully performed operation either of tonsillotomy or tonsillectomy. On the contrary, where obstructive hyperplasia or chronic inflammation has existed an improvement has followed, either in the previously existing faulty resonance or tendency to catarrhal disturbances. In the case of tonsillectomy considerable post-operative deformity may exist without appreciable influence upon the speaking voice. Particularly fusion of the pillars seems to have little effect, provided there is no shortening or puckering from cicatricial retraction. Palatal changes, however, characterized by loss of muscular tissue or by restriction of mobility, are likely to have unfavourable results. It is not alone the graver sorts of deformity that should receive our condemnation, but as Makuen well says, ' In this age of radical operations upon the tonsils by finger and other blunt dissection there is grave danger of using greater force than these delicate structures will bear and of thus stretching them to the breaking point.'

In the case of singers a distinction is to be made between beginners whose voices are in process of training and those whose voices have already attained a definite degree of development. In the first instance harmful alterations of the tonsils are likely to hinder the vocal apparatus from attaining its possible limits of perfection. The principles of procedure previously discussed may here be followed in general by skilful operators with, however, the recommendation in the case of especially promising voices to exercise all possible patience in carrying out other available measures before proceeding to operation. This advice is particularly urgent for soprano voices where a slight change in the movement of the palatal muscles may unexpectedly lower the whole range without a commensurate improvement in quality. I have, to be sure, seen a student, with a fairly good mezzo voice, who after a tonsillotomy done on account of chronic tonsillitis, develop suddenly a contralto of fine quality, but such good fortune cannot be predicted. Furthermore, I should prefer a tonsillotomy, if the conditions permitted, to a tonsillectomy with its greater risk of palatal alteration.

In the case of experienced singers, the above remarks apply with even greater force. In the first place, where the voice is truly a great one, pathological changes in the tonsils, if they exist, are probably not harmful or the voice would not have become great. Only in the rare event of tonsillar changes threatening the general health so seriously as to jeopardize in this way the voice, should any operation be performed, an association of circumstances certainly rare. Extremely common, on the other hand, is the problem of determining whether the tonsils should be removed in the case of singers who have undergone several

years' training and whose voices leave something to be desired. Here we ask: Is the unsatisfactory quality due to the tonsils or does it exist independently of them? The general attitude of teachers on the question of tonsil operations has probably been justified by their experiences in the past when tonsillotomy was the usual procedure, and it has not been shown that enucleation has had a more beneficial influence upon the voice. Many of these singers are predestined to attain only mediocrity, and are constantly seeking our help to remedy vocal imperfections which are actually inherent in a faulty development or training of the vocal apparatus. The risk of causing disappointment, or even of making matters worse, should consequently render the laryngologist extremely conservative in resorting to operative measures in such cases.

While recommending this attitude of caution, I do not wish to be understood as advocating always a policy of non-interference. I should not hesitate to do a tonsillectomy in singers whose persistent laryngitis is attributable to chronic inflammation of the tonsils, but I should insist that the patient understand and assume the possible risk involved.

Conclusions

1. It has not been demonstrated that complete removal of the tonsils is followed by harmful effect upon the general system.

2. Tonsillotomy involves usually less trauma than does tonsillectomy, but in the latter the method of removal is of primary importance, a sharp dissection down to the tonsillar artery, with snaring of the vessels, giving the least amount of inflammatory reaction.

3. Of the two operations tonsillectomy shows a larger percentage of septic complications, due both to the greater trauma usually occasioned and also the relatively larger number of septic conditions where of late years an operation is undertaken.

4. The relative frequency of post-operative hæmorrhage is not definitely established, but, in view of the available methods of treatment, it is no longer a serious complication if dependent upon local causes.

5. While gross deformities of the parts involved are not likely to follow tonsillotomy, yet cicatricial occlusion of the lacunar orifices is frequent, and may lead to an intensification of the original chronic inflammation. Tonsillectomy in unskilled hands may be followed by marked and injurious distortion, but with good technique should have no other alteration than an approximation and occasionally a partial fusion of the pillars.

6. The indications for operation should be determined by the pathological changes of the tonsils which are actually a detriment to the individual.

7. Simple hyperplasia, if obstructive, or favouring catarrhal conditions, and if persistent, may be sufficiently treated by a tonsillotomy, especially in children.

8. The systemic ill-effects of chronic tonsillitis may be increased by a tonsillotomy. Complete removal is here preferable to a partial one, although mild cases of chronic inflammation may be sufficiently relieved by appropriate treatment without excision.

9. Infections of the fauces due to virulent micro-organisms may not be prevented by removal of the tonsils.

10. Recurrent local or general infections having their origin in the tonsils require tonsillectomy as soon as a favourable moment for operating arrives. Tonsillotomy may be expected here to prove inadequate.

11. Recurrent acute catarrhal infections of the throat require complete removal of the tonsils if these show chronic inflammation, although immunity against subsequent attacks is not necessarily secured.

12. Local tuberculosis of the tonsil requires complete removal of the organ.

13. In young children with adenoid enlargement requiring removal, the tonsils should not be excised unless demonstrably causing injury or favouring attacks of acute middle-ear inflammation.

14. If an impairment of the speaking voice is dependent upon tonsillar disturbances, these may be corrected according to the principles already given, and if a tonsillectomy is indicated, it may with proper technique be performed without anxiety.

15. In singers, slight alteration in the tension of the palatal muscles may influence the voice either favourably or unfavourably. In the case of beginners with harmful alterations of the tonsils, a partial or complete removal may usually be done, if the local or general welfare of the patient demands it. With increasing length of singing experience a correspondingly conservative attitude should be maintained, particularly in respect of truly fine voices.

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SECTION XV

RHINOLOGY AND LARYNGOLOGY

DISCUSSION No. 4

(Jointly with Section XVI, Otology, Discussion No. 5)

SPECIAL TREATMENT OF THE THROAT, NOSE, AND EAR, DURING THE ACTIVE STAGES OF CERTAIN INFECTIOUS FEVERS

Report by E. W. GOODALL, M.D.

I propose to deal with the subject which has been set for discussion under the following heads :

I. A description of the morbid conditions of the fauces, nose, and ear in the diseases mentioned.

II. The results, temporary and permanent, local and general, of these morbid conditions.

III. The class and proportion of cases in which these conditions occur.

IV. The treatment, local and general.

1. To prevent the results mentioned under II.
2. To render the patient free from infection.

I. A Description of the Morbid Conditions of the Fauces, Nose, and Ear

It is unnecessary for me to dwell at length upon this description, because the morbid conditions are familiar to nearly every medical man. In most of the diseases in question, though by no means in all of them, the primary lesion is in the fauces and nasal passages, and the neighbouring cavities are secondarily affected by direct extension of inflammation. I shall take the various diseases in the order of the frequency of the faucial lesions.

1. *Scarlet fever.* In this disease there may be any degree of severity, from a simple tonsillitis or catarrh of the fauces to the most severe and extensive inflammation of the soft parts, so extreme as to impede both deglutition and respiration. In those cases in which the inflammation is moderate, a quantity of thick, ropy, tenacious muco-pus is exuded. In more severe cases there is to be observed not only this muco-pus, but also a thick exudation, at times even membranous. When the œdema is extreme, extensive sloughing may follow, resulting in wholesale destruc-

tion of the soft parts. As the exudate clears off, ulceration is revealed, especially along the edges of the uvula and of the soft palate and its pillars. The ulcerative process may spread to the pharynx and larynx. A characteristic and not uncommon lesion of scarlet fever is perforation of one or both of the anterior pillars. This perforation is the result of a localized gangrene, caused, most probably, by the pressure of the inflamed and swollen tonsil upon the œdematous pillar. The perforation is oval and from $\frac{1}{2}$ to $\frac{3}{4}$ of an inch in length. It has been suggested that it may be due to the rupture of a tonsillar or peritonsillar abscess through the anterior pillar. But such abscesses are very uncommon in scarlet fever. The faucial lesion often takes a considerable time to heal, not infrequently several weeks.

In cases where the fauces are much inflamed there is almost invariably present an acute inflammation of the nasal passages. The œdema of the mucous membrane is frequently sufficient to cause blocking of the posterior nares. The discharge, at first thin and watery, soon becomes purulent, and excoriates the nostrils. In some cases the nasal mucous membrane is ulcerated, but the ulceration is rarely deep.

2. *Small-pox.* In this affection the faucial lesion is occasioned by the presence of the eruption. Each pock gives rise to a circumscribed erosion or ulcer. If the eruption is numerous, the lesions become confluent, and the palate, tonsils, and pharynx are involved in an extensive, ragged ulceration. While the eruption is coming out the mucous membrane may be intensely swollen.

The nasal passages may be affected in a similar manner.

3. *Erysipelas.* The mucous membrane of the palate and pharynx is much swollen, and presents a shiny, dull red appearance. There is usually but little secretion. Occasionally blebs form, which rupture and give rise to superficial erosions. The inflammation may spread to the nasal fossæ or to the larynx.

4. *Influenza.* Acute catarrh of the fauces, pharynx, and nasal passages is fairly common. At first the mucous membrane is red and dry, but soon a considerable quantity of watery discharge is exuded, which later becomes muco-purulent. Tonsillitis, with thick exudation simulating diphtheritic membrane, is occasionally present.

5. *Typhoid or enteric fever.* Catarrh of the fauces is by no means infrequent during the early stages of this disease, and there may be very definite tonsillitis, with pultaceous exudation. I have on several occasions known typhoid fever to be misdiagnosed as diphtheria because of the presence of this lesion.

Later in the course of the disease (third week and after), ulcers may form on the tonsils or pharynx ; but they are rarely of any depth. Epistaxis is a not infrequent early symptom of typhoid. Otherwise symptoms referable to a lesion of the nasal fossæ are very uncommon.

6. *Measles.* Pronounced faucial and nasal lesions are not common in this disease. During the initial and eruptive stages the naso-pharynx

is involved in the general catarrh of the respiratory tract, and coryza is usually marked. But it is rare to find definite exudation, and still more rare to meet with ulceration.

But I have on several occasions observed, in patients convalescent from measles, acute and severe inflammation of the fauces and pharynx, which has resulted in deep and extensive ulceration. In a few instances the larynx has also been involved.

7. *Whooping-cough.* Moderate nasal catarrh is not very uncommon during the early stage.

8. *Mumps.* Slight tonsillitis is occasionally present.

9. *Chicken-pox.* In a very few cases the eruption may come out on the palate and pharynx. But it is usually very scanty on those parts and gives rise to no trouble. I once observed a case in which several pocks on the palate became gangrenous, and there was considerable destruction of the velum.

10. *Cerebro-spinal meningitis.* In most cases there is moderate coryza. Ker,[8] of Edinburgh, who has had considerable experience of the disease, writes of it as follows: 'Some congestion of the fauces is often observed, but actual sore throat is hardly ever complained of. That, however, a comparatively acute inflammatory process often occurs in the naso-pharynx, there can be little doubt.'

11. *Rubella* (*German measles*). There is seldom more than moderate catarrh of the nose and pharynx, and even that is not present in every case.

12. *Anterior poliomyelitis.* In this disease nasal catarrh occasionally is present, but sore throat is rare. There are, however, reasons for believing that the infective agent is contained in the secretions of the fauces and nose.[4]

The preceding description has dealt only with lesions of the throat and nose; those of the ear have been left out of account. This is because in most of the diseases the aural lesion is secondary to that of the naso-pharynx, by direct spread of the inflammation to the middle ear through the Eustachian tube. In two of them, however, namely measles and influenza, it is probable that the middle ear is affected at the same time as the pharynx or nose. But this early involvement of the ear is rarely severe, and a secondary inflammation may take place later.

The aural lesions, therefore, belong naturally to the second division of our subject, and will be described under it.

It must be borne in mind that the nasal and faucial mucous membranes are frequently the seat of pathogenic micro-organisms (known or unknown, the cause of the particular disease or of some of its complications), without the presence of any notable lesion. This is especially the case in scarlet fever, measles, influenza, and cerebro-spinal meningitis. It will be necessary to take account of this fact in considering the question of treatment.

II. Results of these Morbid Conditions

1. *Local, of the fauces.* In those diseases in which the faucial and nasal inflammation is at all severe, the temporary results are pain and discomfort, dysphagia and dyspnœa, in proportion to the severity of the lesion. One of the permanent results is loss of tissue and cicatrization, but I have never known these, however extensive, to give rise to permanent disability. But the local inflammation, which is so common in several of the diseases under consideration, not infrequently leaves behind it chronic enlargement of the tonsils and adenoid growths in the naso-pharynx, with all the serious results to development and to respiration which these conditions bring in their train. Scarlet fever, measles, and to a less extent, whooping-cough, are the diseases which are most responsible for setting up adenoids and chronic tonsillitis.

2. Of parts immediately adjoining the fauces the *ear* is the most important and the most frequently affected. Except in measles and influenza, and perhaps also in some cases of whooping-cough, the inflammation of the ear is secondary to that of the fauces, by way of the Eustachian tube, through which infection of the ear by pus-forming organisms may easily take place, especially in scarlet fever. But in most cases the inflammation in the first instance gives rise to a clear, watery, non-purulent discharge, which may cease without becoming purulent, but which often becomes purulent sooner or later. Now and again I have met with instances in which the patient has complained of earache and has exhibited a rise of temperature during the course of one of the acute infectious diseases, and the symptoms have subsided without a discharge and without a perforation of the tympanic membrane. But such cases are by no means common compared with the number in which perforation takes place.

Upon the consequences of otitis media it is quite unnecessary for me to dilate. I will only observe that while a mastoid abscess, suppuration of the antrum or mastoid cells, and inflammation and necrosis of a portion of the mastoid process are not very uncommon within a few weeks of an attack of scarlet fever or measles, the more serious lesions (intracranial suppuration, sinus thrombosis, and pyæmia) are rarely met with during this period, so that they are seldom seen in fever hospitals. I have, however, known a cerebral abscess and pyæmia to arise within four to six weeks of the otitis, and periostitis and ostitis of the mastoid and petrous portions of the temporal bone even earlier.

The text-books of general medicine would lead us to believe that the onset of otitis media is nearly always attended with pain. But this is not my experience in the acute infectious diseases. It is easy to understand that the additional pain due to otitis would not be elicited by the medical attendant or nurse in the case of a young child who was already the subject of an acute and painful inflammation of the throat. But, as a matter of fact, even in older children and in the convalescent stage of scarlet fever, when the throat symptoms have subsided, and in measles,

in which the throat is not painful, earache is neither a frequent nor a pronounced early symptom of otitis. The most frequent sign is a discharge from the ear. In some cases it may have been preceded by a rise of temperature. I have made particular inquiry on this point in the wards of the Eastern Hospital during the past autumn and winter, and I am sure that in the vast majority of cases pain is not an early symptom, at any rate in scarlet fever and measles. In those cases in which pain is present or suspected an otoscopic examination may reveal otitis ; but I must confess that I have not found it easy to assure myself that there was otitis in the cases of young children suffering or believed to be suffering from earache, in whom there was no perforation of the membrane.

When pain is present it is not always located to the middle ear ; it may be referred to the parts around the ear, or be connected with enlargement of the glands just below the ear.

Deafness is usually present. Its degree depends upon the severity of the otitis. I have frequently had under observation children who were almost stone-deaf for four or five weeks during the course of a severe purulent otitis. In such cases perfect hearing is seldom recovered ; but it usually becomes fair, and may be even good. In these cases a large perforation of the membrane remains. It is exceedingly rare for a patient to leave the hospital very deaf, let alone stone-deaf. The persistence of complete deafness is a strong reason for thinking that the internal ears have been involved. But I have never known a patient to become stone-deaf in whom there has not been severe otitis media. Welch and Schamberg,[13] however, have recorded a case of scarlet fever in which absolute deafness resulted without otitis media. They concluded ' that the internal ear was doubtless diseased, perhaps as the result of a localized meningitis '. Of course, if complete deafness supervenes in a child who has not yet learned to talk, deaf-mutism may result. But I have met with only one such case during an intimate association with fever hospitals extending over twenty-three years.

Facial paralysis is occasionally noticed, and that in cases which are by no means severe. It is seen, however, most often in young children, and disappears as the otitis clears up. I have only once known the paralysis to become permanent.

But in certain of the acute infectious diseases permanent deafness may result without a previous otitis media. In mumps it is usually due to otitis interna, but may in some cases be caused by a cerebral lesion. The deafness which is to be met with so often during the acute stage of typhoid fever rarely depends upon a lesion of the ear. It passes off as the patient convalesces, and is probably psychical.

In cerebro-spinal meningitis deafness may occur at any time, and may be absolute and permanent. Sixteen of 152 of Ker's cases became permanently deaf.[9] It appears to be a question whether the deafness is due to disease of the internal ear or to inflammation of the auditory nerve within the cranial cavity.

Other organs adjacent to the fauces which may suffer secondarily to the lesions therein are the *lachrymal duct and sac*, chiefly in scarlet fever, and the *frontal* and *ethmoidal sinuses* and the *antrum of Highmore*, especially in influenza. In nearly all the acute diseases the *mouth* may be inflamed, and stomatitis, simple or ulcerative, occur. This complication is met with most often in measles, scarlet fever, typhoid fever, and small-pox. The ulcerative process may invade the lips, and I have twice known a considerable degree of stenosis of the mouth to result. In severe cases of stomatitis the maxillary bones may become necrosed and the teeth lost.

It is possible, and indeed probable, that stomatitis is not always secondary to faucial inflammation, but is a secondary infection derived from without. The ulcerative process may start in the front part of the mouth and spread to the fauces and larynx. Again, in certain diseases, especially measles, stomatitis is a primary lesion, and of more importance than the faucial or nasal inflammation.

Severe ulceration of the fauces may lead to septic broncho-pneumonia by inhalation (scarlet fever and small-pox).

3. In respect of the *general results of the local lesions* it is hardly necessary to do more than mention the fact that ulceration of the fauces may be the source of pyæmia and septicæmia, especially in scarlet fever, and that otitis, whatever its cause, may give rise to pyæmia.

III. The Class and Proportion of Cases in which these Conditions Occur

In Section I it has been stated that faucial and nasal inflammation is not common, and even when present is very slight, in the following diseases: mumps, whooping-cough, chicken-pox, rubella, and anterior poliomyelitis. Inflammatory lesions of the adjacent organs and structures are also uncommon in these diseases; in fact, in rubella and chicken-pox they are very rare. Of all the other diseases which are under consideration it may be said generally that the secondary implication of adjacent organs depends upon (*a*) the severity of the faucial lesion, and (*b*) the age of the patient. The more extensive, deep, and persistent the lesion, and the younger the patient, the higher the probability of the invasion of the ear and other cavities, structures, and organs adjacent to the fauces. In respect, however, of that portion of this statement which relates to the faucial lesion, measles and influenza form an exception. In neither of these diseases, and especially the former of the two, is the faucial lesion severe, and yet otitis and other sequelæ are common.

In an inquiry which I made some years ago into the age incidence of certain of the complications of scarlet fever I found that of 1,000 consecutive cases of that disease 12·2 per cent. developed otorrhœa, and that the incidence varied with the ages of the patients as follows: under 5, 20·4 per cent.; 5 to 9, 8·4 per cent.; 10 to 14, 4·3 per cent.; 15 and

upwards, 1·3 per cent.[5] From what has just been stated it is also clear that the incidence of otitis and other complications of the various acute infectious diseases will vary with the character of the epidemic, according as the cases are severe or as the proportion of young children is high. The infectious diseases which are most responsible for otitis are scarlet fever, measles, cerebro-spinal meningitis, influenza, small-pox, and typhoid fever. As regards scarlet fever the statistics published by the Metropolitan Asylums Board[11] show that since the year 1899 the incidence of otitis has varied from 11·4 to 15·6 per cent. For many years past the severity of scarlet fever, as indicated by its fatality, has been steadily diminishing, and on the whole the complication rate has also been decreasing. The fatality of upwards of 800 cases of scarlet fever treated in the Eastern Hospital during the year 1912 was only 0·79 per cent.,* and the otitis incidence was 5·4 per cent.

The incidence of otitis in measles is not so readily obtained. Not till December 1910 were cases of that disease admitted to the Asylums Board hospitals ; and even now only cases from the poorest classes of the community are admitted. The fatality of the cases admitted to the Eastern Hospital during 1911 was 20·4 per cent., and the otitis incidence 13·1 ; for 1912 the percentages were 12·4 and 9·3 respectively. Probably at the present time scarlet fever and measles are about equal in their power to produce otitis ; and considering that recently measles has been a much more severe and fatal disease than scarlet fever, it must be concluded that, were the severity of the latter disease to revert to what it used to be some thirty or forty years ago, the incidence of otitis media would likewise increase.

In respect of the other infectious diseases there is very little statistical evidence available of the incidence in them of otitis and similar complications. In the Asylums Board hospitals the incidence of otitis in typhoid fever since 1902 has varied from 2·3 to 6·6 per cent. The incidence of the same complication upon 1,045 cases of whooping-cough in 1911 was 5 per cent. There seems to be no doubt that otitis is common in cerebro-spinal meningitis ; but that disease has not been frequent of late in the British Islands. It is also not uncommon in influenza, and, amongst children, in small-pox. Under the head of otitis I have included all cases in which there has been otorrhœa, purulent or not.

From inquiries I have made during the past few months I have reason for believing that the incidence of otitis following scarlet fever and measles in the hospitals of the Metropolitan Asylums Board is somewhat over-estimated. In not a few of the cases that have been under treatment at the Eastern Hospital I have ascertained that the inflammation of the ear had been in existence for some time (months or years), before the patient was attacked by the disease for which he was sent to the hospital, and that the chronic or latent aural affection has been awakened into fresh activity by the recent malady.

* Corrected for deaths due to previously existing diseases.

IV. The Treatment

1. To prevent or check the results mentioned under heading II

(i) *Local*

(*a*) *Fauces and nose.* In those diseases in which the faucial and nasal lesions are the source of the otitis and other complications (and of these diseases scarlet fever is the most important and typical), the object of treatment is to abate or limit the local inflammation.

The methods most commonly adopted for the fauces are gargling, spraying, syringing, and swabbing, with solutions which are intended either for checking inflammation (astringents and antiseptics), removing the products of inflammation (solvents), or destroying the pathogenic bacteria (antiseptics and germicides). Gargling and spraying are rarely of use except in mild cases and adults. Syringing and flushing are most useful methods, and are in fact indispensable when there is general inflammation with much exudate. Of these two measures flushing performed by means of a douching apparatus is the best ; and the most efficacious solutions are those containing an alkali. When there is ulceration, the ulcerated surfaces should be swabbed with an antiseptic solution, which, however, should not be of an irritant character. Many of these could be mentioned. I prefer either perchloride of mercury (1 in 2,000), or medicinal izal, pure or diluted with water according to circumstances. Unless the inflammation is severe and the fauces are very painful, swabbing is in my opinion preferable to syringing and douching. Of recent years the opinion has been gaining ground amongst medical men attached to fever hospitals that syringing and flushing or douching occasionally set up otitis by forcing septic material up the Eustachian tube. I am, however, inclined to doubt whether material is actually forced into the tube. Whatever method of treatment is adopted, whether syringing, flushing, or swabbing, the act of swallowing is excited. If in the first two of these methods there is too much liquid used at a time, some of it will find its way, without the use of force, into the Eustachian tube just as the latter is opened during the act of swallowing, and may carry with it some of the products of the faucial inflammation ; and so the tube and then the middle ear become infected. I do not remember to have met with a case in which there was reason for thinking that liquid had been forced into or had even flowed up the tube as far as the middle ear. In swabbing properly performed there is no excess of liquid present, and therefore there is very little chance of any of it getting into the tube. For the efficient performance of swabbing, the patient's mouth may, if necessary, be kept open by a gag.

For the treatment of the nasal passages douching with boracic or an alkaline solution is in most cases all that is required.

In my opinion it is not necessary to employ local treatment in cases in which the fauces and nose are unaffected.

(*b*) *Ear.* As I have already stated, in the large majority of cases the first sign that the ear has become affected is the occurrence of a discharge. So that unless a daily otoscopic examination of the ears of every patient is made, it is very seldom that the opportunity of viewing the membrane in the preperforative stage presents itself. A daily examination is, however, quite impracticable in a fever hospital in which there may be 200 to 400 cases under treatment at one and the same time, most of them being children under seven years of age. Incision of the membrane at an early stage is, therefore, rarely practised in these institutions. But if otitis media is diagnosed or even suspected before perforation takes place, an effort should be made to prevent sepsis from occurring after incision or perforation by gently syringing the ear with an antiseptic solution and lightly plugging the meatus with antiseptic gauze, which should be secured in its place with a pad of sterilized wool and a bandage. The discharge, in a case in which the ear has never before been affected, is at first nearly always clear, non-purulent, and inoffensive to the smell. In many cases the discharge ceases under treatment without becoming offensive or markedly purulent. But not a few, in spite of the same treatment, become septic. This secondary infection may come through the Eustachian tube or the external meatus. The latter route is very probable in cases in which the faucial lesions are not severe or persistent. The chief care, therefore, is threefold: to provide for free drainage for the discharge, to cut short the inflammation, and to prevent secondary septic infection. To secure the first of these objects it may be necessary in a few cases to enlarge the opening in the membrane which has been caused by the disease. Usually, however, the perforation is of some size and is situated in the lower part of the membrane. The second and third objects are attained by gently syringing out the ear with antiseptic and astringent lotions. In fever hospital practice those most in favour are boracic acid and weak carbolic acid (1 in 60 to 80). Syringing should be performed every three or four hours, and the dressing mentioned above should be used. After a week or two peroxide of hydrogen may be employed in addition to these solutions. A few drops of the peroxide should be instilled into the ear and retained there for a few minutes, at the end of which the ear is to be syringed out with the boracic. If the discharge becomes chronic, and especially if, as is then often the case, it is also septic, stronger antiseptics and astringents should be used. It is essential after syringing and before applying the dressing that the meatus and external ear should be thoroughly dried with pledgets of sterilized wool mounted on suitable probes.

That these measures are efficacious in the majority of cases is shown by the following figures. Of 303 cases of scarlet fever which were discharged directly from the Eastern Hospital during the period January 1, 1912, to the middle of February 1913, in only one was otorrhœa still present; of 421 cases of measles in 8 there was persistent otorrhœa, and in 2 of these certainly the otitis was of long standing; of 90 cases of

whooping-cough in only 1 was there otorrhœa when the patient left the hospital. Again, during the ten years 1900 to 1909 there were 153,607 cases of scarlet fever treated in the hospitals of the Metropolitan Asylums Board.[11] In 20,349 of these there was otitis media (13·1 per cent.); but only in 920 cases was there a mastoid abscess or disease of the temporal bone (0·6 per cent.). That is to say, only 4·5 per cent. of the otitis cases resulted during the time they were detained in hospital in the spread of inflammation beyond the middle ear. I may add that the practice in most of the fever hospitals is to keep the aural cases under treatment till the discharge has ceased; but if this event has not happened before the end of twelve weeks, then the patient is allowed to leave the hospital. I need hardly say that in such cases advice is given to the patient or his friends to apply for further treatment to a doctor or hospital.

I have no doubt that some of the cases which seem to be cured at the time they leave the fever hospital relapse at a later date; but I doubt whether many of them do. At any rate I seldom hear of them. On this point I will quote C. B. Ker, of the Edinburgh City Hospital for Infectious Diseases. In that city Ker has better opportunities of obtaining information concerning the patients who have been under his care in the City Hospital than have the medical superintendents of the fever hospitals in the metropolis, where there are so many special and general hospitals to which patients can resort, and where, too, in consequence of the large size and extent of the city, it is very difficult to follow up patients after they have left the fever hospital. 'In Edinburgh, if there is any subsequent trouble, the Ear Department of the Royal Infirmary is practically the only place for the patient, and yet I am informed that the bad results dreaded by the advocates of operative interference are not seen among the persons admitted to that institution. I cannot believe that my patients die miserably at home, as the great majority of them are attended to by competent practitioners, who would certainly send them to the Infirmary should occasion require. Moreover, while the City Hospital, like other similar institutions, is accused of many things, I have not heard complaints of serious sequelæ in connexion with otorrhœa after dismissal from hospital, and I should hardly fail to do so should they occur with anything like the frequency which is alleged.'[10]

Within recent years it has been advocated in certain quarters that if an otorrhœa persists for more than six weeks, the antrum and, if necessary, the mastoid cells should be opened up, or even the complete operation performed, even if there be no evidence that these structures are involved. But I am of the opinion that such a proceeding is not justifiable except in the most chronic cases, and that a prolonged course of treatment on the lines already indicated should be carried out before resort is had to operation. And I believe that in this opinion I am supported by all my colleagues in the Asylums Board service. I have already stated Ker's views on the question. He says further that of 1,800 cases of scarlet

fever treated in the Edinburgh City Hospital during the year 1908 in only one was an operation considered necessary. His experience is in accordance with mine at the Eastern Hospital.

(ii) *General*

Under this head I refer to measures, other than local applications, by which it is hoped that the *local* conditions will benefit directly. These are treatment by the subcutaneous injection of a specific *serum*, and treatment by inoculation with specific *vaccines*.

The diseases in which these methods are most likely to be of benefit are scarlet fever, small-pox, and erysipelas. In erysipelas I have known good results from the use of an anti-erysipelatous (anti-streptococcic) serum. I have not had the opportunity of inoculating a vaccine in the faucial form of this disease ; but as, according to Horder,[6] vaccine treatment is of value in the cutaneous form, it may be expected to be also of service in the faucial variety.

Of the serum treatment of scarlet fever it is difficult to speak with certainty. Occasionally one meets with a case in which a polyvalent anti-streptococcic serum, in doses of 20 to 50 c.c., has appeared to work wonders ; and yet perhaps in the next case and with the same serum no benefit whatever results. Of recent years severe cases of scarlet fever have been so few in number that there has not been the material upon which to base conclusive evidence as to the value of any treatment so far as the acute faucial inflammation is concerned. Both the serum and the vaccine treatment of scarlet fever depend upon the belief, for which there are strong grounds, that the septic troubles are due to the secondary invasion by organisms of various kinds, but especially streptococci. The vaccine treatment of scarlatina anginosa, with the view of cutting short the disease and preventing the occurrence of such complications as otitis and rhinitis, has been practised now in a fair number of cases by several observers. But opinions vary as to its value, probably because the number of cases treated by each separate observer has been few. Boughton and Weaver[2] treated 88 cases and came to the conclusion that they received no benefit. Foord Caiger,[1] of the South Western Hospital, Stockwell, was disappointed with the results in the few cases in which he tried the method ; and I did not observe any beneficial effect in a small number of cases. On the other hand, Jochmann and Michaelis,[7] after an experience of the treatment in 52 severe cases, with 14 deaths, speak favourably of it. They state that no complications occurred in those patients who survived. Their opinion is supported by that of Norman H. Walker,[12] of the Willesden Isolation Hospital. According to him 5,000,000 to 20,000,000 cocci should be inoculated as an initial dose, to be followed by twice that amount in three or four days. An autogenous vaccine appears to be the best ; but as this may take some time to prepare, a stock vaccine, made from a large number of different

strains of streptococci, may be used at first. Eyre,[3] speaking generally of vaccine therapy, expresses his preference for ' a virulent autogenous vaccine not more than two generations removed from the human body, cultivated under optimum conditions, suspended in 0·1 saline, . . . standardized against normal blood, sterilized by exposure in a water bath of a temperature corresponding to the thermal death-point of the organism in question for one hour, dilution to the requisite extent with 0·25 per cent. tricresol solution, and employed in small doses during its efficient period of from three to six months '. Until we are able to use the vaccine treatment in the acute infectious diseases in a large number of cases with vaccines prepared in the same way and given on an agreed system of dosage, I fear that it will be a long time before we are able to arrive at a definite opinion as to their value.

With these reservations I may say of the vaccine treatment of otorrhœa and rhinorrhœa persisting after an attack of one of the acute infectious diseases, that there is a stronger hope of its efficacy. The most frequent organisms present in these discharges are strepto- and staphylo-cocci, but a variety of others may also be present, and therefore it is best to employ an autogenous vaccine. Hitherto I have not resorted to vaccine until the discharge has failed to yield to local treatment, which should be continued with the use of the vaccine. But, so far at any rate as a persistent nasal discharge is concerned, I am of the opinion that it is unwise to persevere too long with syringing, as in many cases it seems to keep up the discharge.

Adenoids will also aid in the continuance of a rhinorrhœa or an otorrhœa ; and if the latter conditions do not improve under any of the methods already mentioned, the adenoids should be removed. But I do not recommend their removal too soon after an attack of an acute infectious disease, still less during the attack itself. I have seen disastrous results follow the removal of enlarged tonsils and adenoids during the early stage of convalescence from scarlet fever. The operation should not be done till at least eight weeks after the acute attack ; and, if it can be arranged, the patient should be sent to convalesce in the country for the two or three weeks before it is proposed to operate.

2. To render the patient free from infection

The virus of the disease is believed to be present in the fauces, nasal passages, and perhaps the middle ear, in all of the infectious diseases under discussion. But in only a few of them are there reasons for concluding that it remains active in these parts after the acute stage of the disease has passed, namely, in scarlet fever, influenza, erysipelas, and cerebro-spinal meningitis. Of these four diseases scarlet fever is the one for which the strongest evidence can be brought forward for the persistence of infective material in the naso-pharynx. Hence, any of the measures which have already been recommended for the purpose

of curing chronic inflammation of the parts mentioned will at the same time assist towards rendering the patient free from infection. There are, however, a certain number of cases of scarlet fever in which a recovered patient who presents no morbid state of the naso-pharynx or ear still remains infectious. We know that cases of the same nature are to be found in diphtheria, in which disease the specific virus can be demonstrated. Is there any means of ridding such patients of these active micro-organisms? Unfortunately, my experience in diphtheria goes to show that there is no certain method of securing this end. Neither douching, nor swabbing, even with strong solutions of germicides, nor serums, nor vaccines, can be relied upon to free the mucous membranes from the specific micro-organisms. Fortunately, the number of cases in which the infection persists is hardly appreciable, except in the case of scarlet fever; and in that disease it is seldom above 4 per cent. of the recovered cases. And the means which are most likely to secure the disappearance of the infecting agent in scarlet fever are, in addition to those which will bring about the cure of any morbid condition of the naso-pharynx, those which are adapted to promote an improvement in the general health and surroundings of the patient.

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SECTION XV

RHINOLOGY AND LARYNGOLOGY

DISCUSSION No. 5

TRATTAMENTO E PROGNOSI DEI TUMORI MALIGNI DELLE FOSSE NASALI E DELL' EPIFARINGE

Relazione del Prof. GHERARDO FERRERI, Regia Università di Roma

Considerazioni generali

Fin dal 1893, in un mio lavoro riguardante ' I tumori maligni della volta naso-faringea ', dichiaravo indispensabile distinguere in una classificazione a sè le malattie dell' epifaringe, seguendo in ciò l' avviso di Lennox Browne che si coordina con la legge della divisione del lavoro e con ragione di sviluppo e morfologia.

Lennox Browne cominciò col separare il naso dalla faringe mediante un piano immaginario tangente il bordo posteriore del vomere. Bosworth divise la stessa faringe in altre due parti, cioè la volta naso-faringea o epifaringe e la regione oro-faringea o ipo-faringe ; divisione importantissima dal punto di vista fisio-clinico, essendo la prima porzione sovratutto incaricata del respiro e la seconda della deglutizione. A detta di Trautmann il cavo naso-faringeo o epifaringe comincerebbe dalle coane e si estenderebbe per l' altezza di due centimetri fino ad una linea tirata trasversalmente innanzi al tubercolo faringeo.

Nei trattati classici di chirurgia quando si viene a parlare dei tumori maligni delle prime vie respiratorie, in genere questa distinzione tra epi- ed ipo-faringe è trascurata. Alf. Poland, Langenbeck, Kocher, Labbé, Castex, Broca, Krönlein ecc., parlano dei tumori maligni della retrobocca. Krönlein in una sua memoria (1897) (61 operazioni personali di cancro della faringe) dichiara che il cancro risiede molto raramente nella parte nasale della faringe, non avendone egli riscontrati che due casi. Roudié in un suo recente lavoro dice che gli epiteliomi della volta faringea debbono essere raccolti assieme ai tumori maligni delle fosse nasali, per cui non ne tiene conto nella sua tesi.

I tumori maligni della volta faringea sono molto più rari di quelli del naso e delle cavità accessorie ; però i tumori primitivi dell' epifaringe sono un pò più frequenti di quelli dell' ipofaringe dove Winiwarter notò un caso sopra 548 e v. Langenbeck uno sopra 230. Orth non potè riunire che 18 casi dell' ipofaringe ; solo Krönlein riunendo gli uni e gli altri

raccolse 28 casi di cancro primitivo. König ha detto che i carcinomi limitati alla faringe sono di una rarità estrema.

L' esperienza di ogni giorno smentisce l' opinione assai diffusa fra i chirurgi generici che essi s'intendono altrettanto bene di rinologia come gli specialisti. Senza dubbio la chirurgia del naso non è che un adattamento dei principii della chirurgia generale, come vuole Heath, ma bisogna altresì riconoscere che l' abitudine e l' esperienza sono necessarie in una specialità che richiede strumenti particolari e una tecnica manuale propria.

Essendo nei tumori maligni la prima indicazione l' ablazione totale, non dovremo ritrarci dinanzi ai metodi operativi che necessitano le più grandi demolizioni. Ma di fronte alla gravità eccezionale di esse, che non sempre raggiungono la guarigione, sorse la necessità, sentita anche in tutte le altre branche della medicina, di vagliare tutti quei mezzi conservativi messi in opera contro le neoplasie maligne.

Crediamo anzi che sia prezzo dell' opera cominciare a fare una disamina di tutti i mezzi terapeutici che, astraendo dalla chirurgia, furono e sono usati in terapia. Si deve anzi notare che proprio ora, dopo un periodo chirurgico attivissimo, si sente il bisogno di scaricarsi degli insuccessi operativi per tentar altre vie, rievocando la medicina umorale, che pareva tramontata per sempre.

Accingendoci a questo lavoro di sintesi c'è in verità da domandarsi se si sia fatto qualche passo decisivo nella cura dei tumori maligni. Per conto nostro il problema è ancora più grave per la difficile accessibilità delle regioni di cui ci occupiamo e per i loro rapporti con organi così indispensabili alla vita vegetativa e di relazione.

È spiacevole, prima di accingerci a trattare della cura dei tumori maligni delle prime vie aeree, il dover confessare che tutti i tentativi scientifici che si sono succeduti per delucidare la loro genesi e natura, nonchè la cura in rapporto a queste conoscenze, avranno fatto progredire in alcuni punti speciali il dottrinale oncologico, ma nel complesso siamo in un periodo di confusionismo in cui tutte le teorie, anche le più unilaterali, fondate sopra alcune osservazioni cliniche di laboratorio ineccepibili, mostrano di avere qualche attendibilità.

Nel periodo Virchoviano, ammessa la dottrina embrionale, si poteva parlare, secondo canoni consacrati dalla sicura esperienza clinica, di terapia chirurgica dell' epitelioma, del sarcoma, dell' endotelioma ecc. Oggi, in cui si mette persino in dubbio da alcuni che il cancro e il sarcoma rappresentino delle specie morbose assolutamente fisse, e si crede che sieno suscettibili di trasformazione o meglio derivazione l' uno dell' altro (non sono più rese rigide le frontiere fra questi varii tumori), sembra che dobbiamo attendere nuovi lumi dalla dottrina umorale, che va riacquistando terreno.

Data questa nebulosa concezione patogenetica dell' attuale periodo è giustificato che, svolgendo il tema dei tumori maligni, non mi azzardi di dividerli scolasticamente in capitoli ben distinti. Se, ad esempio, volessimo insistere nella classificazione antica e nello stesso tempo aderire

alla dottrina di coloro che ammettono una etiologia parassitaria, dovremmo potere identificare per ogni singola varietà di tumore maligno una determinata e stabile specie di microrganismo : il che non è finora dimostrato.

Siamo dunque costretti a parlare di questo capitolo di clinica in un momento di transizione, nel quale tutto quello che era creduto solidamente fondato viene ripreso in esame e rifuso nel crogiuolo di una analisi critica minuziosa, di cui non si possono prevedere i risultati ultimi.

Profilassi

Poichè nemmeno approssimativamente la causa del cancro è conosciuta, si comprende che deve essere assai relativa la profilassi o per meglio dire la prevenzione di questa malattia nelle prime vie respiratorie. Non cesserà tuttavia di avere una qualche efficacia la lotta contro tutte quelle cause generiche occasionali che s' imposero per lo più all' attenzione dei medici nello studio dell' anamnesi. Non vi ha dubbio, ad esempio, che fra le cause che sembrano favorire la produzione dei cancri sono le irritazioni croniche di fronte alle quali sono assai più discussi, secondo alcuni, come fattori patogeni i traumi, l' eredità, il contagio.

Specialmente nelle prime vie respiratorie l' importanza patogena delle irritazioni croniche è evidente, sia che dipendano da fattori d' indole chimica e meccanica, sia che si pongano in rapporto con le stesse affezioni morbose di cui il naso e la faringe sono sede prediletta, come l' ozena, la sifilide ed i catarri cronici. Se un tempo aveva un valore indiretto l' uso di fiutare il tabacco, ora lo ha tra le cause predisponenti l' abuso della sigaretta che induce molti all' abitudine di fare uscire il fumo dal naso.

Seguendo queste idee generali la cura preventiva del cancro dovrà consistere anzitutto nell' evitare le cause capaci di produrre questo stato d' irritazione cronica del naso e della volta, richiamando i pazienti a non trascurare in una certa età anche quei disturbi soggettivi che per la loro tenuità non preoccupano. Si divulghi sempre di più l' idea che negli stati di irritazione cronica del naso e della volta la degenerazione cancerigna può essere già avvenuta quando meno l' interessato poteva sospettarla.

Quante volte abitudini di pigrizia e di indifferenza avranno fatto rinviare di giorno in giorno la visita dello specialista, perdendo miseramente il tempo utile per la cura radicale di una lesione ancora localizzata. Senza dubbio la precauzione è una cosa aleatoria contro una malattia della quale non si conosce l' essenza e la scienza medica non è riuscita ancora ad andare al di là di un programma molto generico.

Tutti i più eminenti cultori di oncologia segnalano la sempre maggiore perdita cagionata dai tumori maligni, tanto che Dolbat pensa essere il cancro ' le plus grand tueur d'hommes si l'on excepte la tuberculose ', e Czerny prova con la statistica alla mano che negli ultimi dieci anni il cancro è salito al secondo posto tra le cause di mortalità tra gli adulti dei paesi civilizzati.

Quando sarà organizzata pure in Italia la statistica in modo razionale ed uniforme come in Inghilterra per lo studio del cancro, non solamente risulterà confermato il fatto che la cifra *assoluta* delle morti per tumori maligni in genere è raddoppiata in circa 20 anni nel nostro paese, ma che in questo numero conglobale vanno crescendo in proporzione cospicua quelle per neoplasmi delle prime vie respiratorie.

Vi sono molti che negano importanza al fatto constatato dalle statistiche dell' aumento dei tumori maligni nell' ultimo ventennio, sostenendo che ciò va ascritto alle migliori condizioni igieniche che permettono ad un numero maggiore di individui di raggiungere età avanzate, allora quando cioè è più frequente l' insorgenza dei tumori. Si può ribattere questa asserzione sostenendo invece che, come è avvenuto per altre forme morbose, i progrediti mezzi diagnostici hanno incolonnato fra i tumori malattie prima definite altrimenti e che la propaganda igienica sanitaria rende accorti molti i quali prima non si curavano del loro male.

Terapia medica generale

Si deve tenere in grande considerazione il fatto che agli specialisti sovente capitano gli scarti dei chirurgi generici, vale a dire tutti quei tumori maligni delle prime vie respiratorie dichiarati inoperabili o perchè troppo tardi diagnosticati o perchè recidivi di vaste demolizioni.

Sarebbe contro l'onestà professionale che non venissimo in soccorso anche di questi pazienti inoperabili, abbandonandoli al loro destino, che prima di essere fatale percorre un lungo periodo di atroci sofferenze. In attesa di un' era più fortunata, che non è vano sognare, per la cura del cancro, noi dobbiamo con tutti i mezzi di cui la terapia moderna dispone soccorrere questi infelici con la convinzione che si può in qualche modo non solo alleviare le loro sofferenze, ma prolungarne l' esistenza, sovratutto combattendo le complicazioni flogistiche che sono la principale causa di emorragie ed alterazioni funzionali che prima ancora dell' intossicazione da neoplasma conducono a morte.

È qui che l' abilità degli specialisti può rivelarsi con la scelta giudiziosa dei mezzi curativi che abbiano due fondamentali direttive, l' una rivolta ad agire sul neoplasma stesso per arrestarne la fatale evoluzione, l' altra semplicemente palliativa contro i disturbi funzionali ed infettivi da esso provocati.

Passando ora nel campo delle cure mediche dei tumori maligni del naso e della volta faringea, la questione, sebbene di attualità viva tanto per i clinici quanto per i sofferenti, resta ancora chiusa in un cerchio di mistero dal quale pare non riesca a togliersi. La dolorosa constatazione è che i tentativi di sottoporre i neoplasmi maligni alle cure mediche, dopo aver suscitato facili entusiasmi e magari apparenti successi, sono uno dopo l' altro sistematicamente caduti.

Occorre però, in questo periodo di attesa, discernimento nel vagliare tutte le tendenze terapeutiche, basate talune in concezioni metafisiche

quali non fanno che ritardare la soluzione del problema. Purtroppo finora tutto ciò che fu esperimentato nel nostro campo fuori del coltello « dominare questo malanno ha fallito al crogiuolo della clinica e non beneficamente influenzato le statistiche. Solo può affermarsi che la tecnica migliore ed il frutto dell' altrui esperienza ci fa sperare che anche nelle affezioni cancerose delle prime vie aeree si potranno presto annoverare quei successi che si vengono vantando nelle altre regioni del corpo.

Allo stato attuale per la cura dei tumori maligni abbiamo due categorie di rimedi: la chirurgica, che è tuttavia la migliore allorquando è precoce, e la medica incruenta. Questa può avere due direttive: (*a*) agendo sui germi cancerigni, ed uccidendoli od attenuandoli, sieno essi cellule atipiche, o microrganismi; (*b*) favorendo la reazione dell' organismo colpito.

L'azione diretta si è dimostrata finora la più efficace, sia che essa si sia esplicata con mezzi fisici sulla superficie o nel parenchima neoplastico, sia che attraverso l' organismo venisse influenzata la struttura del tumore, per la maggiore fragilità degli elementi neoplastici.

Gli studi moderni sono rivolti a ricercare sostanze che si fissino elettivamente sugli elementi cancerosi in modo da ridurne o sopprimerne la vitalità.

Poichè i tumori nella struttura e nella biologia sono fra di loro dissimili, occorrerebbe adattare a ciascuno di essi un agente adequato, ma non possedendo noi rimedi individualizzati dobbiamo procedere con un certo empirismo basato sulla reazione pressochè identica ai mezzi di cui disponiamo. È certo che i sarcomi sono i più sensibili alle cure incruente, mentre i carcinomi resistono di più e le guarigioni finora furono eccezionalmente rare.

Si sa che i tumori maligni hanno tendenza ad immunizzarsi contro i rimedi curativi, i quali finiscono per perdere ogni prestigio e le cellule neoplastiche si moltiplicano, quasi la loro attività venisse esaltata; da qui è sorta la necessità dei trattamenti combinati che si rafforzino nella loro azione sia definitiva sia temporanea.

Terapia fisica

Elettrocaustica. Merita una seria attenzione da parte dei chirurgi ciò che Price-Brown asserisce, in base ad una numerosa statistica da lui fatta, sui risultati dei metodi operativi in genere adottati per l'asportazione dei sarcomi nasali. Egli viene a concludere che questi tumori hanno per lo più origine dalle parti molli e non dalla sostanza ossea che esse ricoprono e che mentre in principio sono a forma peduncolata rapidamente divengono sessili. Le masse sarcomatose tendono ad allargarsi ed a comprimere le parti vicine con le quali prendono aderenza, senza però che queste conservino la vitalità ed il potere di riproduzione.

Le recidive sempre hanno luogo nella regione del peduncolo ed è in vista di questa contingenza che il chirurgo, secondo Price-Brown, deve

sempre aver per punto di mira la sede d'impianto del tumore. Allorchè il tumore è molto bene limitato e se ne può ben circoscrivere il punto d'origine, egli preferisce un intervento endonasale a qualsiasi operazione extra-nasale ; ma siccome nè il coltello, nè il laccio, nè le forbici, nè *la curette*, nè tutti questi metodi combinati possono con successo asportare un largo sarcoma che ha sempre un' estesa base d' impianto, consiglia quale metodo efficace di cura, e come strumento di escissione il coltello galvanico ripetutamente e regolarmente usato fino alla completa distruzione del neoplasma.

In tutti i casi in cui i sarcomi nasali non hanno invaso le pareti ossee della faccia Price-Brown è fautore convinto del cauterio elettrico, in base ad una statistica di 51 casi tenuti in osservazione fino all' esito finale, in cui i pazienti furono operati ora per la via interna ora per la via esterna : la guarigione completa in quelli operati per la via endonasale fu raggiunta nel 31 per cento, in quelli operati dalla via extranasale 24 per cento.

Elettrolisi. In tutte le operazioni della faringe le difficoltà dell' asepsi sono quasi insuperabili, benchè siensi esagerate a tal punto le precauzioni preliminari da sostenere persino utilissima la tracheotomia preventiva in tutte le ablazioni di tumori faringei. Se l' estirpazione di essi dalla via naturale della bocca non offre sufficienti garanzie antisettiche, le incisioni esterne che permettono di giungere meglio sui tumori maligni della faringe hanno il grave inconveniente che per mancanza di vitalità le ossa temporaneamente resecate quasi mai si saldano, i pericoli di una morte immediata sono maggiori e ancor più comuni le emorragie secondarie.

Non si deve fare un conto serio degli splendidi risultati di cui parlano gli operatori, perchè li vediamo troppo abituati a pubblicare le proprie osservazioni personali a pochissima distanza dal momento dell' intervento. Qualunque sia stata la demolizione compiuta, la recidiva a breve scadenza sembra essere la regola nei tumori maligni della faringe. È raro che la vita si prolunghi oltre un anno. Vale dunque procedere ad ardimentose operazioni per ottenere così scoraggianti risultati, lasciando intanto gli infermi, per quel pò di tempo che sopravvivono, orribilmente deturpati ?

Se non esistono operazioni radicali cruente sarà molto più saggio il contentarsi dell' elettrolisi ; metodo di cura che non domanda spietate demolizioni e può distruggere la più gran parte del tumore, specialmente quando gli aghi cadono sopra i più grossi tronchi vasali nutritizii.

Nella nostra clinica, avendo adoperato l' elettrolisi per la distruzione di grandi tumori della volta naso-faringea, anche quando i disturbi a carico del sesto nervo craniense, che giace aderente al corpo dell' osso, indicavano uno stato di cose da rendere impossibile qualsiasi tentativo chirurgico, avemmo da notare numerosi vantaggi.

Anzitutto un pericolo molto minore di emorragie immediate e secondarie, caduta di grandi masse del tumore necrosato, essendosi trombizzati

i principali vasi sanguigni, oppure subinvoluzione con rimpiccolimento del neoplasma, in modo da cessare i fenomeni di soffocazione, la chiusura delle narici e delle trombe di Eustachio, le cefalgie, la lacrimazione ecc. : inoltre nessun bisogno di anestesia, mentre sappiamo che i neoplasmi faringei provocano spesso la morte nell' atto della cloroformizzazione (Caldwell), ostruendo improvvisamente l'apertura della glottide ; niente bisogno della tracheotomia preventiva, onde rendere più facile il compito di qualsiasi trattamento di asportazione cruenta ; allontanata la minaccia della morte immediata, delle bronco-polmoniti cancrenose, dello schock, della mancata adesione delle ossa temporaneamente resecate ; infine nessun incitamento ad un tumultuario sviluppo della neoplasia maligna, come avviene in genere quando si osa affrontarla col coltello chirurgico.

Holger Mygind di Copenhagen è uno dei pochi che, oltre la nostra Clinica, si è servito dell' azione elettro-chimica per la distruzione dei tumori maligni faringei. Egli adoperò la corrente prodotta con la batteria galvanica, raccomandata da Apostoli per le operazioni ginecologiche. La forza della corrente da lui usata variava dagli otto ai dodici milleampères, oltre la quale l'applicazione arreca dolore. La seduta dura venti minuti. L' elettricità si applica per quattro o cinque giorni di seguito e poi, prima di riprendere le sedute, si lascia un intervallo di tre o quattro giorni di riposo.

L'elettrode positivo nelle prime sedute va applicato esternamente sul collo o il lato destro della testa, non comprendendo il padiglione, in forma di una larga piastra fenestrata di zinco, coperta di garza bagnata. In seguito ambedue gli elettrodi vanno applicati nel tumore stesso in forma di aghi d'oro. Mygind sostiene che il trattamento elettrolitico trasforma gradualmente questi tumori faringei in una massa grigia necrotica alla loro periferia, senza cattivo odore e qualsiasi altro sintomo di putrefazione e senza elevazione di temperatura e difficoltà di deglutizione.

Folgorazione.—In alcuni neoplasmi maligni delle narici e del cavo naso-faringeo al posto della elettrocaustica, quando essi siano facilmente aggredibili, potremo usare la folgorazione, seguendo la tecnica indicata dal Keating-Hart, avendo però prima eseguita la più larga eseresi chirurgica dei tessuti invasi dalla neo-formazione.

Secondo Ghilarducci una folgorazione molto intensa e prolungata ritarda se non impedisce la recidiva locale. Il valore terapeutico della folgorazione sarebbe sovratutto evidente nei carcinomi. All' eliminazione dell' escara succederebbe un processo di granulazione molto rapida ed una sollecita cicatrizzazione delle parti folgorate. Gli infermi migliorano notevolmente nelle condizioni generali e terminano del tutto i dolori atroci che li tormentavano prima dell' atto operativo. Le esperienze di Ghilarducci hanno dimostrato che la folgorazione dei nervi periferici non solo determina la loro distruzione ed una consecutiva paralisi, come già dimostrarono le esperienze di Bergonier, ma agiscono a distanza sui centri nervosi spinali, producendo alterazioni strutturali nei nervi periferici

che si dipartono dalla regione folgorata, assai più estese di quelle riscontrate dopo semplice resezione o strappamento degli stessi nervi.

Alla folgorazione segue una linforrea costituita da sierosità rosea con molti polinucleati neutrofili. Essa si prolunga talvolta per settimane ed è fortemente tossica, per cui il metodo in questione spesso riesce infruttuoso ed è causa di complicazioni negli interventi eseguiti entro le cavità accessorie del naso, da cui non sempre è facile allontanare con un drenaggio cotesta profusa linforrea.

Diatermia.—La diatermia od elettro-coagulazione, cioè l' applicazione locale della corrente ad alta frequenza, fu pure tentata per la cura dei tumori maligni. Essa ha il vantaggio, secondo alcuni, anche sul coltello chirurgico, di non aprire vasi e quindi di non dar luogo a disseminazioni.

Forse dai chirurgi arriverebbe ad avere la preferenza, se non fosse per sè stessa pericolosa a motivo della sua azione a distanza, per cui si va incontro ad estese e gravi distruzioni, seguite da imponenti emorragie secondarie al distacco delle parti necrotizzate. Ecco perchè si tende ora a sostituire alla diatermia l' elettro-disseccamento, ma è questo un tentativo troppo recente perchè si possa darne un giudizio.

La *termoterapia* che, applicata sotto tutte le forme nei tumori accessibili, pare abbia dato buoni risultati, non è per ragioni anatomiche di possibile esperimentazione in quelli del naso e del cavo.

Radio-terapia.—Nei tumori maligni nasali che abbiano già invaso la pelle e divenuti quindi inoperabili si è pure ricorso all' azione dei raggi X, la quale determinerebbe negli elementi cellulari irradiati speciali trasformazioni chimiche il cui ultimo termine è la degenerazione e la morte. Con la radio-azione non si ha però una distruzione immediata, come dopo una scottatura da fuoco e da caustici, ma le cellule colpite tanto sane che malate, dopo un periodo di latenza, durante il quale non si hanno fenomeni morfologici evidenti, subiscono un processo involutivo, muoiono, sono sostituite e immobilizzate da connettivo giovane, in cui sopravvivono rari elementi cancerosi allo stato di vita attenuata, quando pure ne sopravvivono.

I raggi X, se si sono dimostrati attivi contro le malattie di cui noi ora ci occupiamo, è innegabile che in molti casi hanno piuttosto avuto azione stimolante. Pare che questi insuccessi debbano ascriversi ad errori di tecnica. Sembra che con la filtrazione dei raggi, con la insensibilizzazione della cute e con l' uso dei fuochi incrociati, si sia giunti ad evitare la maggior parte degli inconvenienti.

Come regola generale conviene dire che è preferibile l' azione prolungata a quella rapida, specialmente per le neoplasie voluminose, allo scopo di evitare l' intossicazione per subito riassorbimento dei tessuti distrutti.

I raggi X rendono buoni servizi, dopo l'intervento chirurgico, per sopprimere i germi cellulari eventualmente rimasti ed impedirne così la migrazione e consecutive metastasi.

Radiumterapia. L'azione del radium nelle cellule cancerose è dimostrata istologicamente ; essa è complessa. Una parte delle cellule muoiono e sono eliminate in massa sotto forma di escara, o lentamente sotto forma di suppurazione prolungata ; un' altra parte risentono quella che si dice azione trasformatrice, per cui si riavvicinano al tipo normale perdendo i loro caratteri di malignità.

Sui tumori maligni si agisce col radium in tre modi : con applicazioni esterne di apparecchi a radiazioni composte (raggi α, β, γ), con applicazioni esterne mediante apparecchi a radiazioni molto penetranti (raggi γ), con applicazioni intraneoplastiche mercè apparecchi a radiazioni ultrapotenti.

L'uso di uno piuttosto che dell' altro di questi metodi è consigliato dalla minore o maggiore malignità del neoplasma. Così nei tumori maligni a più lento sviluppo, costituiti da tessuto non troppo ricco di cellule e quanto più essi si allontanano dal tipo embrionale, bastano le applicazioni superficiali con gli apparecchi del 1° e 2° tipo. Nei tumori poveri di stroma e ricchi di cellule embrionali si consiglia l' immissione nel parenchima di tubi di radium o radiazioni molto penetranti (raggi γ). I risultati, man mano che si perfeziona la tecnica, sono sempre più incoraggianti e specie nei sarcomi si ottennero effetti insperati.

Il radium è anche ottimo coadiuvante della chirurgia a cicatrizzazione avvenuta e si usa allo scopo di colpire quegli elementi neoplastici che sfuggiti al coltello e latenti per tempo vario sono causa di recidiva. Si applica il radium sulla cicatrice e sulle stazioni linfatiche viciniori. Alcuni consigliano addirittura l' iniezione sottocutanea, nel campo prima occupato dal tumore, di minime quantità di sali insolubili di radium (solfato di radium).

Oltre il radium si sono adoperati altri corpi radioattivi e cioè il mesothorium, il thorium X, le emanazioni, l' actinium e la plecbenda uranifera.

Il succedaneo preconizzato del radium sarebbe il mesothorium, di migliore accessibilità specie per il prezzo, metà del radium. Esso fu usato con la stessa tecnica, specialmente nelle neoplasie superficiali ed in qualche caso con miglior successo, pare per la minore penetrabilità dei suoi raggi.

Le emanazioni dei corpi radioattivi furono utilizzate contro i cancri per iniezioni endovenose di gas sciolti in soluzione fisiologica ; sopratutto furono usate le emanazioni di radium e di thorium.

Furono inoltre adoperate delle pomate radifere e le iniezioni endotumorali di sali insolubili.

I risultati in genere furono buoni, ma si ebbero a deplorare alterazioni gastro-intestinali e renali mortali per cui specie il thorium X è guardato con un certo terrore e deve essere usato con molta prudenza.

Nei tumori maligni naso-faringei, in particolare quelli inoperabili, vennero pure sperimentate sostanze radio-attive per iniezioni locali, secondo la tecnica consigliata da Delbet, Petit, Czerny e Caan. A questo

scopo si adoperarono il solfato e bromuro di radio ed anche l' actinio sospesi in soluzione fisiologica (0,5 di sale in 10 c.cm.), iniettando dopo lungo scuotimento del recipiente.

Le iniezioni intraneoplastiche devono muovere a principio dalla dose di 0,01 gr. e poi gradatamente raggiungere gr. 0,1–0,2, venendo eseguite a giorni alterni mediante una siringa di Record munita di ago-canula. Queste iniezioni provocano notevole reazione associata a brivido, perciò Petit preferisce un siero radio-attivo preparato nel modo che segue ; si sottopone un cavallo a due riprese, distanti di parecchi mesi, all' iniezione di un milligr. di solfato di radio insolubile immesso per la giugulare. Il sangue prelevato anche dopo molto tempo da questa iniezione presenterà il siero radio-attivo.

Terapia medicamentosa

La cura del cancro ha risentito, nei suoi tentativi di rimedi generali, l' influsso delle idee fondamentali in medicina, in una data epoca. Ignorandosi la vera natura di questi tumori, si è proceduto per analogia e sovente con idee preconcette e assolutamente ipotetiche. Così, quando si scoprì nel mercurio l'agente specifico della lues, si ricercò pure un medicamento per le affezioni cancerigne, ritenute diatesiche. E allora i chimiastri hanno concepito la speranza di neutralizzare, con prodotti farmacologici, questo presunto virus generatore del cancro : noi ricorderemo ad esempio il tentativo di un antico chimico di Montpellier. Egli pretendeva che il virus canceroso consistesse in un fluido ammoniacale carico di ossido di azoto, donde ne concluse che l'acqua ossigenata e la potassa dovevano essere usate come mezzi neutralizzanti della malattia. Altri, movendo dal concetto di un profondo perturbamento della crasi umorale dell' organismo colpito dal cancro, propose come unica prescrizione e unico alimento del malato l'acqua distillata.

Potremmo enumerare molte ricette fondate su tali principii patogenetici, ora in completo disuso ; ma è bene non dimenticare che il problema della cura del cancro affaticò la mente bizzarra e generale del Paracelso, il quale preconizzò fra i *rimedi specifici* delle affezioni cancerose diversi prodotti chimici, fra i quali l' acetato di rame, il muriato di barite, il carbonato e tartrato di ferro ecc. Uno è oggi di nuovo risalito in onore, e cioè l'arsenico.

Arsenico. Fra le sostanze chimiche più usate nella cura dei tumori maligni fu l' arsenico, applicato localmente allo scopo di distruggere le neoplasie ; sovratutto l' anidride arseniosa ebbe gran voga e diede risultati non disprezzabili, sia adoperata in polvere, sia in pomate ed in soluzioni alcooliche (polvere di frate Cosimo, di Manech, Dupuytren, Rousselet ecc., pomata di Hebra, empiastro di Unna ecc., soluzioni di Czerny, Trunecek).

Disgraziatamente nei tumori di cui noi ci occupiamo non è possibile applicare questo trattamento terapeutico per ragioni ovvie.

All' arsenico però è riconosciuta una azione oncotropica attendibilissima, per cui la sua somministrazione per uso interno ed ipodermico

ha fautori convinti, mentre altri credono che la sua efficacia sia dovuta al potere ricostituente generale. Sono raccomandate nei tumori maligni varie formule contenenti arsenico (acqua di Levico e Roncegno, liquore del Fowler, granuli di acido arsenioso ed arseniato di soda, cacodilati, atoxil, arsenico colloidale ecc.).

Da alcuni venne espresso il dubbio che anche nei tumori maligni l' etiologia si possa riportare ad una spirillosi di natura ignota, per il fatto che negli individui affetti fu spesso riscontrata positiva la reazione di Wassermann. In base a tale ipotesi ed alla cognizione dell' utilità terapeutica arrecata dal Salvarsan nelle malattie da spirilli in generale, fu sperimentata l' azione dell' arseno-benzolo nei tumori maligni naso-faringei.

Mentre la cura negli epiteliomi non diede risultati soddisfacenti, meno lievi e temporanee migliorie circa il dolore ed il volume, nei sarcomi talvolta portò alla necrosi e alla eliminazione del tumore. Ma chi può toglierci il dubbio che non siasi trattato di un effetto svoltosi non sopra un tumore maligno vero e proprio, ma invece su una forma di sifilide ignorata? Non va nemmeno trascurato che qualche nodulo neoplastico può scomparire in seguito a necrosi da iniezione locale, quindi per le gravi lesioni che il Salvarsan determina in sito a carico di tessuti avariati. Czerny si ripromette migliori risultati dal neo-salvarsan che presenterebbe il vantaggio della solubilità e quindi più pronta penetrazione nei tessuti.

Dopo ciò dobbiamo riconoscere che neppure con l'arsenico si è giunti ad alcun esito positivo.

Chemoterapia

Poichè le cellule del cancro sono inverse di quelle normali e per la struttura e per il metodo di funzionamento, si ritiene che la loro composizione chimica sia pure diversa e che quindi esse devono reagire in un modo speciale a certi reagenti chimici. Si tratta dunque di trovare delle sostanze, le quali abbiano delle affinità chimiche per le cellule del cancro e non già per le cellule normali. Grazie a tale affinità esse si combineranno colle cellule del cancro e, così facendo, ne altereranno la costituzione in modo da renderne impossibile lo sviluppo ulteriore e quindi ne provocheranno lo sfacelo, la scomparsa.

Si sa che carattere delle organizzazioni neoplastiche, di fronte al rapido accrescimento, è il disgregamento cellulare per autolisi. La chemoterapia cerca di accrescere il potere autolitico.

La ricerca di queste sostanze ha dato dei risultati positivi. Si sa, ad esempio, che l' arsenico portato in contatto ad un certo grado di concentrazione col tessuto canceroso lo distrugge rapidamente, mentre se viene portato in contatto del tessuto sano nello stesso grado di concentrazione non svolge nessuna azione distruttiva.

In base a questa constatazione sono stati preparati molti rimedi che sono infatti capaci di distruggere i nodi del cancro. Sfortunatamente è difficile praticarne l'applicazione in modo che si riesca ad uccidere

tutte le cellule del cancro: ora basta lasciarne illesa una sola, perchè da essa si riformi poi il tumore.

Inoltre questo genere di cura è impraticabile nei cancri interni, poichè le sostanze in questione per la maggiore parte se vengono introdotte nel sangue in dose sufficiente per andare a distruggere il tumore interno, svolgono anche delle azioni tossiche sull' intiero organismo. In altre parole, quando queste sostanze vengono usate internamente, la loro dose terapeutica, cioè quella che distrugge le cellule del tumore, è troppo vicina alla loro dose tossica, vale a dire a quella che distrugge anche le cellule normali. Insomma una differenza apprezzabile esiste fra la reazione delle due specie di cellule, differenza che è a svantaggio delle cellule del cancro, ma è così piccola che cessa di essere apprezzabile praticamente.

Fra le sostanze che hanno una tale azione sulle cellule dei tumori stanno in prima linea specialmente il selenio, il tellurio, il rame. Ora un gruppo di studiosi, a capo dei quali sta Maurizio Ascoli, hanno constatato che le azioni alternanti di questi metalli nelle cellule degli organi e quindi anche sulle cellule dei tumori si accentuano se i metalli vengono usati allo stato colloidale, cioè allo stato di suddivisione molecolare estrema.

La facoltà organotropica specifica di una sostanza è il primo e più prezioso carattere che ne stabilisce il valore in chemoterapia; per essa noi siamo sicuri che la sostanza adoperata si dirige là dove miriamo, risparmiando al massimo il resto delle cellule dell' organismo. Questa direzione tuttavia non è sufficiente da sola, per cui vediamo che lo jodio e l' eosina, nonostante il loro spiccato oncotropismo, non hanno valore terapeutico sui tumori. Occorre che oltre il requisito della direzione la sostanza abbia anche quello dell' attività modificatrice che il caso richiede. Ad esempio i composti colloidali hanno congiunte nella stessa molecola ambedue le suddette proprietà. In chemoterapia insomma si ha bisogno di due elementi distinti e cioè di un involucro fissatore o veicolo e di un corpo attivo. Il veicolo è destinato ad avere il massimo di affinità per l' elemento che si vuole influenzare, in modo da dar tempo al corpo attivo di agire. Di corpi attivi la chimica è oltremodo doviziosa, ma il difficile è trovarne il veicolo o avvolgente.

Nei tentativi del Wassermann, finchè i composti di selenio e tellurio venivano adoperati da soli, non si riusciva che ad avvelenare i topi senza nessun benefico effetto del tumore. La scoperta apparve solo dopo che quei corpi ebbero trovato l' involucro fissatore nell' eosina.

Wassermann, spappolando pezzi di epitelioma fresco in soluzioni di telluriti e seleniti alcalini, notò fenomeni reattivi evidenti e cioè la pigmentazione degli elementi vitali in nero o viola scuro coi telluriti e in rosso coi seleniti; a ciò seguì in breve la distruzione delle cellule neoplastiche.

Queste esperienze di Wassermann e suoi collaboratori indussero a perfezionare i preparati chimici e la tecnica relativa, al punto di per-

mettere l'attacco dei tumori maligni e le loro propaggini con una carica di tellurito e di selenito introdotta nella rete circolatoria, per spingerlo ad agire automaticamente dove esistono chemocettori influenzabili, cioè localizzazioni neoplastiche.

Wassermann ed i suoi seguaci constatarono che l'impiego dei telluriti e seleniti, mentre determinano un processo regressivo iniettati nella massa dei tumori, non lo provocano per via endovenosa, probabilmente per la scarsa vascolarizzazione dei neoplasmi, e quindi si dettero a pazienti ricerche per trovare sostanze che funzionassero da veicolo. A tale uopo riscontrarono utile il miscuglio con le fluoressine. Fu prescelto il composto eosinselenico nel quale il metalloide mercè l' eosina si mantiene fisso al suo radicale acido, finchè percorre tessuti fisiologici, ma, appena invade aree neoplastiche, si dissocia e va ad infiltrare il tessuto.

Wassermann con le sue esperienze di laboratorio sui sarcomi e carcinomi dei topi ha concluso per la guarigione di questi tumori, mercè speciali preparati selenio-eosinici. Questo nuovo indirizzo nella terapia dei neoplasmi maligni degli animali ha aperto l'adito alla speranza di poter agire sui neoplasmi dell' uomo.

Si è anche usato di curare il cancro colle iniezioni di metalli ridotti allo stato colloidale, partendo dal principio che, bastando in tal caso l' introduzione di dosi piccole, sarebbe stato più facile sorvegliare l' andamento della cura ed evitare le azioni tossiche imbarazzanti. Ma l' esperienza fatta finora con questo metodo è troppo scarsa perchè sia possibile trarne delle conclusioni attendibili. In realtà il Gaube du Gers ha comunicato d'avere ottenuto buoni risultati dall' uso del rame colloidale, mentre i tentativi fatti con l' iridio, il rutenio, il rubidio, il rodio, l' osmio, lo stagno, il piombo, l' oro, non risposero allo scopo ; di questi si dimostrarono più attivi, oltre il rame, il platino, lo zinco, il cobalto e l' argento. Fu notato che le iniezioni endevenose di tali sostanze attive determinarono iperemie intense, e talora emorragie interstiziali dei tumori, cui segue rammollimento e mobilizzazione del tessuto. È conclusione ovvia allo stato delle cose che i metalli pesanti hanno corrisposto male allo scopo ; solo si può dire che qualche efficacia hanno dimostrato quando furono usati assieme ad altri rimedi.

Una speranza nuova si ebbe dall' introduzione nella terapia del cancro, della colina, che sarebbe un derivato della lecitina sotto l'azione o dei raggi X o dei corpi radioattivi. La colina è la base attiva della neurina di Parlavecchio o della cancroina di Adamkiewicz. Fu adoperata prima la colina basica, ma forse a causa della sua labilità dette gravi intossicazioni da neurina ; si provò in seguito la borocolina (combinazione con l' acido borico), la quale agirebbe sui tessuti ricchi di lecitina come lo sono i cancerosi.

Nella cura del cancro si tentò eziandio l'uso di altre sostanze organiche più o meno definite, fra cui la tripsina, l'amilasi alternata o no ad antiamilasi, l' aldeide formica, l'acido acetico, l' acetone, lo jequirity, la resorcina, la cancroina, le nucleine ecc.

Immunoterapia

Bisogna riconoscere che il problema terapeutico del cancro ha compiuto qualche passo buono quando si è basato sulle migliori conoscenze attuali dei poteri difensivi che l' organismo oppone alle invasioni cancerose, sia rinforzando la reazione umorale dell' economia, sia favorendo i processi reattivi del connettivo perineoplastico.

È giuocoforza però dichiarare che fino adesso tutti questi tentativi terapeutici teorici non si sono dimostrati per nulla superiori ai vecchi rimedi empirici generali o locali. Dimodochè, essendo ben lontani dall' avere scoperto uno specifico del cancro, resta assioma terapeutico che la cura chirurgica precoce tiene il primo posto.

In generale fu osservato che fra i tumori maligni delle prime vie respiratorie si mostrano abbastanza sensibili alle cure incruente quelli derivati dal connettivo, all' opposto di quelli di origine epiteliale. In tutte queste neoplasie maligne è la zona periferica d' infiltrazione quella che presenta maggior resistenza a tutte le cure. Persino il tessuto cicatriziale che si forma al posto dei tumori asportati o distrutti diviene quasi sempre il punto di partenza di recidive. Secondo alcuni persistono ivi germi residuali del tumore. Theilhaber invece crede che la recidiva locale avvenga per alterazione nutritizia delle cellule comprese nella cicatrice, in un organismo già predisposto alle neoplasie ; donde l' importanza di intervenire sulle cicatrici residue di tumori curati. Questo scopo si raggiungerebbe col mantenere vascolarizzato il tessuto di cicatrice mercè mezzi iperemizzanti che provocherebbero il rammollimento ed il riassorbimento dei tessuti cicatriziali.

Sieroterapia. Per ciò che riguarda la cura diretta, alcuni studiosi sono partiti dal concetto seguente : le cellule del cancro, siano esse prodotte o no dalla presenza d' un parassita, costituiscono degli elementi profondamente diversi per struttura e per attività biochimica dalle cellule normali ; quindi l' organismo deve comportarsi rispetto a loro come si comporta in generale ogni volta che entrano in esso degli elementi vivi estranei, cioè reagirà col produrre sostanze incaricate di attaccarli e di distruggerli.

In certo numero di casi questo sforzo dell' organismo risulta efficace e quindi il tumore maligno non si sviluppa o viene presto distrutto ; in altri casi lo sforzo dell' organismo risulta inefficace ed allora il tumore maligno può svolgere tutta la sua azione malefica. Lo scopo della cura consiste dunque nell' aiutare l' organismo nello sforzo che esso fa di produrre le sostanze protettive.

Ora uno dei metodi più noti per ottenere questo scopo consiste nello introdurre nell' organismo stesso i succhi di altri organismi che siano affetti dalla malattia in questione e quindi stiano già compiendo quello sforzo spontaneamente. È possibile procurarsi questi succhi in quantità sufficiente, ricorrendo agli organi di animali, nei quali lo sviluppo del cancro sia stato indotto in via sperimentale.

Questa è l' origine della cosiddetta immunoterapia o sieroterapia del cancro, alla quale naturalmente si unirono i tentativi di coloro, e non sono pochi, che credono d' aver scoperto i germi vivi del cancro e quindi ricorrono, per preparazione del siero, a quel materiale organico che in base alle speciali indagini loro conterrebbe in maggior numero i germi vivi specifici od i loro prodotti.

I tentativi fatti in questa direzione sono stati numerosi ; ma, sebbene la sieroterapia del cancro possieda già una base logica e sperimentale abbastanza ampia, i risultati pratici da essa ottenuti sono stati assa scarsi. Se io ne ho parlato l' ho fatto e per amore di completezza e perchè il metodo di una cura del cancro, di cui si parla maggiormente oggi, quello di Odin, sarebbe precisamente un metodo sieroterapeutico.

Ricordiamo eziandio il siero di Jansen, di efficacia sperimentale non dubbia, ed il siero di cani vaccinati (Vidal), questo però fu trovato molto tossico per l' uomo. Si usò anche il siero di animali vaccinati in ipermononucleosi (Vaughan) ed infine il siero normale che avrebbe un potere oncolitico manifesto.

Va ricordata infine la isosieroterapia, cioè l' inoculazione di essudati di cancerosi (Machay ed Hodenpyl). Furono prescelti quelli pleurici e il liquido ascitico ; ma Ewing opina che i successi presunti siano i medesimi a cui si giungerebbe con l' iniezione di siero di sangue normale.

Vaccinazione. Si preconizzarono come vaccini terapeutici gli estratti di tessuti cancerosi ; essi determinerebbero un' immunità attiva dell' organismo canceroso.

Valore di vaccino hanno le tossine del Coley che avrebbero forte azione ipertermizzante, la cancroidina od antimeristema di O. Schmidt, estratta da un micetozoo che sarebbe il microrganismo del cancro e dalle spore di *mucor racemosus* che sarebbe il suo ospite, il siero di Doyen, il siero di Sanfelice,tolto dal saccharomyces neoformans, e la tubercolina.

Opoterapia

Nei tumori maligni del naso e dell' epifaringe con metastasi ghiandolari fu consigliata la cura tiroidea, allo scopo di provocare per modificazioni organiche metaboliche la degenerazione degli elementi neoplastici e la sostituzione connettivale di essi. Questo trattamento si basa sull' interpretazione avanzata da Woods che la genesi dei tumori dipenderebbe da perturbate correlazioni organiche, da anomalie nelle funzioni endocrine, con difetti compensati dalla somministrazione di principii istogeni, donde il ripristino nell' equilibrio tra fattori biochimici e proliferazione di tessuti.

Fra i molteplici costituenti organici saggiati furono preferiti quelli di tiroide e timo che la maggioranza degli autori indica come più atti a dare benefici risultati nelle diverse forme neoplastiche. A tale scopo si è adoperato l' estratto alcoolico etereo di tiroide di vitella e pecora (hyrochrom) in tabloidi, due o tre pro die.

Con questa cura molte volte si ottenne la riduzione, il rammollimento, la fluttuazione delle metastasi glandolari ; la pelle sovrastante si arrossava e l' incisione dava luogo alla fuoriuscita di pus ; la ferita in breve si chiudeva con cicatrice perfetta. Altre volte l' infiltrazione neoplastica glandolare riducevasi di volume e scompariva senza lasciar più traccia di riproduzione del tumore (Jones, Woods).

Nell' endocrino-terapia si possono fare rientrare i fermenti epatici e l'adrenalina. Fu il Mahu che nel 1903 propose le iniezioni di adrenalina nella sostanza stessa del tumore. Col medesimo metodo vennero anche curati epiteliomi ulcerati, riscontrando che si arrestavano le persistenti emorragie, si attenuava il dolore e migliorava eziandio lo stato generale dei pazienti.

Nessuna soddisfacente spiegazione si è però saputa dare riguardo all' azione dell' adrenalina sui tessuti neoformati, sia dell' epitelioma, sia del sarcoma delle prime vie respiratorie. Fisiologicamente si sa che l' adrenalina determina una costrizione tanto delle grandi arterie quanto delle piccole, perchè aumenta la tonicità di tutti i tessuti contrattili e quindi anche del protoplasma endoteliale dei capillari sanguigni e linfatici, donde restringimento del loro lume e diminuzione di scambio fra il sangue ed i liquidi interstiziali. Bisogna però tenere conto che le iniezioni ripetute di adrenalina hanno provocato arterio-sclerosi, glicosuria ed alterazioni del fegato e dei reni.

Autolizzati. Per la cura dei tumori maligni si agita da qualche anno fra gli oncologi la questione degli autolizzati organici ottenuti o con tessuti fetali o con tessuti normali (fegato, milza, midollo delle ossa ecc.), oppure con tessuti neoplastici. Il laboratorio ha dimostrato che gli autolizzati aumentano i poteri inibitori dell' organismo per i tumori maligni, moderano lo sviluppo di essi e giungono perfino a farli scomparire. Su questi risultati sperimentali confortati dai lavori di Fichera, Brancati, Blumenthal, Carlo Lewin, C. Graff, Cimoroni, ecc. ecc., non vi è più discussione.

Trasportati nel campo clinico gli autolizzati hanno anche corrisposto in molti casi e noi stessi potemmo osservare in due infermi di epitelioma delle fosse nasali una netta azione di remora con l'uso dell' autolizzato Fichera. I fautori della cura delle neoplasie maligne con gli autolizzati non dubitano che, migliorata la tecnica, si dovranno certamente ottenere risultati più decisivi anche nell' uomo.

Terapia combinata

Da quanto abbiamo detto risulta che i metodi per la cura incruenta del cancro abbondano, il che significa che il problema è ancora insoluto. I migliori di essi al crogiuolo della clinica possono considerarsi una speranza per l' avvenire più che una realtà del presente.

Pertanto nessuna cura medica ha sull' uomo valore di specifico ; possono ottenersi risultati parziali ed anche arresti momentanei, ma i successi definitivi sono discutibili e si vengono assottigliando mano mano che ci allontaniamo dall' esperimento.

Tuttavia da alcuni trattamenti (radium, raggi X, elettricità sotto varie forme, preparati arsenicali, chemoterapia alla Wassermann, autolizzati ecc.) si ha un' azione inibitrice e talora regressiva che in molti casi ha permesso l' intervento chirurgico laddove in primo tempo esso non fu ritenuto possibile.

Migliori successi la terapia medica ha ottenuto con la cura combinata o mista di due o più fra i metodi incruenti, nell' intento di accrescerne il potere oncolitico. Esempio caratteristico ce lo dà l'azione combinata del radium e dei raggi X. Le radiazioni sortono maggiore effetto, se contemporaneamente si iniettano nello spessore del tumore sali organici di argento od il carbonato di bismuto ; questi sali produrrebbero raggi secondarii localmente più attivi. Müller associò i raggi X e Stein il radium con la diatermia conservativa oppure la folgorazione.

Oggi si parla molto di un metodo che sembra la rievocazione della terapia empirica ed alchimistica, ma che tuttavia pare abbia dato buoni risultati ; vogliamo dire della cura alla Zeller, che consiste nella somministrazione del silicio per via interna in una con l' applicazione locale di una pasta arsenicale che distrugge tutti i tessuti che incontra. Il silicio a questo scopo fu già consigliato dal medico inglese Batty, con l' idea che vetrificasse i tessuti neoformati. Anche in cancri accessibili del naso pare che Zeller abbia ottenuto vittoria.

Laddove le cure incruente pare diano vera soddisfazione si è nel trattamento post-operatorio ; in questo campo tengono il primo posto le irradiazioni coi raggi X e col radium, eseguite tanto sulla cicatrice quanto sulle vie linfatiche, per prevenire e distruggere le recidive. Anche la folgorazione si è dimostrata buona associata alla chirurgia.

Come trattamento contro la recidiva post-operatoria si è ricorso con vantaggio alla termoterapia ed all' uso di medicinali iperemizzanti locali, come la tintura di jodio, la glicerina al xero e vioformio, la fibrolisina ecc.

In conclusione il coltello è ancora l' arma meno infida contro i tumori maligni. Tutte le cure escogitate potranno coadjuvarlo, non sostituirlo, e dovranno essere adoperate da sole soltanto allora quando il chirurgo è persuaso che l'opera sua debba riuscire assolutamente inutile o dannosa.

Cura chirurgica

Abbiamo esposto tutti i metodi di cura medica degni di menzione nella lotta contro i tumori maligni, riservandoci di parlare in fine della cura chirurgica, quella cioè a cui tuttavia si debbono i maggiori successi, sempre quando essa sia precoce e generosa.

Posta la conformazione anatomica della regione, la tecnica chirurgica delle più complicate e demolitrici. Vi sono interventi mercè i quali fosse nasali, seni e cavo faringeo vengono messi allo scoperto e ce ne sono di quelli diretti ad uno solo dei segmenti del naso. L' indagine clinica più progredita ci permette oggi la diagnosi di un tumore al suo inizio anche se esso è indovato in uno dei seni della faccia.

Alle più precise diagnosi si deve anche la possibilità di interventi endonasali che sono praticati con successo specie contro i sarcomi, i quali non raramente si originano nelle fosse nasali sotto forma peduncolata.

Nell' esposizione della chirurgia dei tumori tratteremo quindi prima degli interventi nel naso e nell' epifaringe come quegli che danno modo ad agire anche sui seni e poi parleremo degli interventi sui seni separatamente. Va da sè che anche quando l' azione chirurgica è diretta contro un solo seno, se il reperto operatorio indichi maggiore diffusione del processo, conviene allargare l' intervento. Per questa ragione mi sono diffuso nella chirurgia del seno sfenoidale, perchè fino ad un certo punto essa è comune a quella dell' ipofisi, le cui neoplasie maligne talora finiscono per invadere le cavità dello sfenoide.

Prima di accingermi alla descrizione particolareggiata mi è grato constatare che, al giorno di oggi, la rinologia, per merito dei suoi cultori, si è tolta dal vassallaggio della chirurgia generale ed essa non rifugge ormai più dai più gravi interventi, che or non sono molti anni erano di esclusivo dominio dei chirurgi generici.

Anestesia.—Una delle cose non trascurabili nel trattamento chirurgico dei tumori maligni nasali e dell' epifaringe è la scelta dell' anestesia, argomento che è sempre di attualità nonostante le ampie discussioni in proposito.

I pericoli che accompagnano la somministrazione del cloroformio nei prolungati interventi gravi sulla testa e sul collo spinsero molti chirurgi generici e specialisti, sovratutto in Inghilterra e negli Stati Uniti, a sostituire l'anestesia cloroformica con quella mista d' ossido d' azoto ed etere. È noto che nonostante tutte le precauzioni dell' arte le morti per cloroformio si sono avute fin dal principio dell' uso dell' anestesia, anche nei più semplici interventi, quale l' asportazione delle tonsille e delle vegetazioni adenoidi, e come essa sia sovratutto pericolosa nella giovane età denutrita ed indebolita dalla stessa discrasia prodotta dalla natura maligna dei tumori che richiedono l' atto operatorio.

Al cloroformio sovratutto si è fatto carico poi che per ottenere una profonda anestesia occorre somministrarne una quantità rilevante, per cui è facile andare incontro ad un intossicamento dell' organismo che già durante l' operazione si manifesterà con una notevole depressione circolatoria, spiccato pallore, respirazione stertorosa : fatti che richiedono da parte del chirurgo e degli assistenti una continua vigilanza del soggetto.

Si è adunque consigliato di sostituire al cloroformio la combinazione del protossido d' azoto coll' etere per determinare col primo una rapida incoscienza, col secondo una notevole economia di anestetico. Alcuni svantaggi che sono stati riscontrati nella somministrazione del protossido di azoto, vale a dire la cianosi e l' aumentata secrezione di muco, potranno essere evitati con più perfetta tecnica permettendo sovratutto la contemporanea inspirazione di aria.

Nelle operazioni nasali e faringee, nelle quali senza dubbio è d' impaccio la presenza del cloroformizzatore, si è parlato di far seguire alle

inalazioni di protossido d' azoto l' anestesia per il retto con l'etere. Questo metodo fu preconizzato fin dal 1847 da Buxton per le operazioni sulla testa e sul collo. A questo metodo si è imputato di determinare talora meteorismo, diarrea, melena.

Non disponendo del protossido di azoto prima di procedere alla eterizzazione alcuni hanno proposto e sperimentato il bromuro di etile il quale determina minore cianosi e secrezione di muco. Del bromuro di etile si deve esigere un' accurata preparazione per evitare l' azione deprimente sul cuore dei bambini e degli adulti con alterazioni cardiache. Ne hanno fatto uso nella specialità per i primi E. Meyer, Grabower e Rosenberg. Ne furono anche partigiani Schmidt, Gleitzmann. La maschera deve escludere completamente l' aria. L' amministrazione del bromuro d' etile deve essere continua, sotto il controllo del polso fino a che non si senta abolito il tono muscolare. L' anestesia si ottiene nello spazio tra i 15 ed i 30 secondi, ma non oltre i 40. Appena ottenuto il rilasciamento muscolare s' inizierà l' uso dell' etere.

La narcosi per la via rettale, secondo il processo classico di Pirogoff (vapori di etere), non entrò mai nella pratica comune, perchè riconosciuta pericolosa, oltrechè incerta, nella sua efficacia.

Si notarono casi di gravi lesioni intestinali, vale a dire congestioni e ulcerazioni che dettero luogo non solo a diarree, ma anche a emorragie e peritoniti. Legnes, Marel e Verliac vollero porre riparo a questi inconvenienti : (*a*) coll' impiego di bromuro d' etile che non solo abbrevia la durata dell' amministrazione dell' etere, ma impedisce l' elevazione esagerata della pressione nel grosso intestino ; (*b*) sostituendo ai vapori di etere a 50° dell' aria saturata per gorgoglio d' etere, alla temperatura ordinaria ; (*c*) colla spinta dell' etere mediante una corrente d' ossigeno invece dell' aria. A tale scopo questi autori hanno ideato un apparecchio speciale e riuscirono ad evitare gl'inconvenienti deplorati nella narcosi per via rettale.

Nelle ablazioni di enormi tumori del naso-faringe Wyth consiglia di fare l' anestesia quasi esclusivamente con la morfina, non dando che pochissima quantità di cloroformio ed etere per inalazione. Durante l' operazione si dovranno poi fare sempre iniezioni intravenose di soluzione salina per prevenire il collasso e lo schock cagionati dalla emorragia.

Nelle operazioni nel naso e nell' epifaringe si pensò di ridurre i dannosi effetti dell' etere e del cloroformio, che per la lunga durata di siffatti interventi esigono una gran quantità di anestetico, colle misture proposte da Schleich nel 1895. Esse sono composte di etere, cloroformio e benzina rettificata in tre progressivamente graduali proporzioni, in modo che il N. 1 contiene meno cloroformio delle altre due.

La teoria su cui si basa Schleich è che un buon anestetico deve avere una temperatura di ebollizione quasi eguale a quella del corpo umano. La sostanza ideale sarebbe il bromuro di etile, ma la presenza del bromo lo rende troppo pericoloso. Dopo varie esperienze questo autore concluse che la sua mistura N. 1, essendo la più evanescente, sarà utile nelle

operazioni più leggere e quelle N. 2 e 3 producendo un' anestesia più profonda sono migliori negli interventi più gravi.

Si è riconosciuto unanimemente che l' anestesia con tale metodo è assai rapida, non è accompagnata da eccitamento, nè da cianosi, non determina sgradevole accumulo di muco nella trachea, non provoca il vomito ed il risveglio è rapido. Questo metodo ha richiamato l'attenzione specialmente dei rinolaringologi, poichè per i nostri malati è utile un immediato risveglio che riveli eventualmente un' emorragia post-operatoria.

Nella chirurgia della testa e del collo si è in questi ultimi tempi studiato il modo di raggiungere un' anestesia profonda e prolungata del paziente e nello stesso tempo di eliminare dal campo operativo gli ostacoli gravi all' operatore costituiti dalla presenza dell' apparecchio destinato all' anestesia, dall' azione sgradevole dei vapori anestetici e dal cloroformizzatore.

Perciò nell' anestesia generale per gl' interventi nella bocca, naso e faringe si è andato vieppiù diffondendo il metodo preconizzato da Crile nel 1905. Quindici minuti innanzi alla narcosi il paziente riceve un' iniezione ipodermica di solfato di morfina e atropina. La narcosi è quindi cominciata nel modo solito o all' etere o al cloroformio. Quando l' anestesia è completa s' introduce un apribocca, pennellando le narici e la faringe con una soluzione di cocaina e adrenalina. Dovendosi aggredire il cavo-faringeo con uno dei metodi per la via orale procederemo allora all' anestesia coll' intubazione nasale della faringe. S' introducono 2 tubi di Nelaton nelle narici fino a tanto che i loro occhielli siano giunti proprio al disopra dell' apertura della laringe ; allora un tampone di garza è stipato nell' ipofaringe per evitare che scenda sangue nelle vie respiratorie. Un tubo di vetro a Y riunisce i cateteri a un tubo che va esso stesso ad innestarsi a un imbuto di cui la parte svasata è chiusa da una flanella. In questo si riversa l' anestetico impiegato, o cloroformio o mescolanza anestetica.

Per gli interventi che debbono iniziarsi dalla via nasale l' anestesia generale si potrà ottenere seguendo l'intubazione boccale della laringe, cioè spingendo i tubi di Nelaton fino all' ostio laringeo e tamponando strettamente l' ipofaringe e le coane con garza. In questi interventi il più semplice metodo è lo stesso strumentario di O'Dwyer. Sulla canula di O'Dwyer si adatta direttamente il tubo di cautchouc che va all' imbuto anestetico.

Superiore per perfezione di strumentario è quello proposto e da tutti ormai accettato da Kuhne di Cassel.

Meltzer con un dispositivo speciale previa intubazione o tracheotomia pratica la narcosi eterea. I vantaggi dell' apparecchio di Meltzer sono che mediante la manovra di speciali rubinetti è possibile far respirare solo aria o miscela di aria ed etere senza pressione o sotto pressione graduabile. Lo strumentario è stato semplificato e reso più pratico da D'Avack ed esperimentato con successo dal Prof. Alessandri nel suo quartiere chirurgico.

Nella chirurgia delle prime vie respiratorie l' anestesia ideale sarebbe quella mediante la cloro-flebo-narcosi, se non avesse l' inconveniente di aumentare l' emorragia in operazioni nelle quali la perdita di sangue è per sè stessa imponente e di gran pena per l' operatore.

L' anestesia è rapidissima (3 o 4 minuti) e avviene senza periodo di eccitamento, ma con un leggero delirio. Dapprima i pazienti accusano un sapore dolciastro in bocca e quindi un senso di ebbrezza seguita da incoscienza, risoluzione muscolare e scomparsa dei riflessi. La respirazione si mantiene sempre profonda e calma, il polso di ritmo normale. Finita la cloro-flebo-narcosi gli operati si risvegliano rapidamente dopo cinque o sette minuti, senza nausee, nè vomito, soltanto con la presenza quasi immediata nelle urine di una scarsa quantità di albumina, emoglobina ed emazie; in genere questi disturbi scompaiono entro 24 ore.

I chirurgi generici prescelgono la grande safena a 4 o 5 centim. dal suo sbocco nella vena femorale, ma noi abbiamo preferito nei nostri casi la vena mediana basilica. Si usa una soluzione fisiologica satura di cloroformio (0,60 per cento) a 37°, procurando che nei primi minuti la velocità del liquido iniettato eguagli 100 c.c.; in seguito diminuiremo la velocità del liquido in modo che non sorpassi i 50 cent. cubici per minuto. In tal guisa si può giungere a far penetrare nelle vene fino a 2500 cent. cubici di soluzione equivalente a 15 grammi di cloroformio.

La cloro-flebo-narcosi è contro-indicata nei vecchi, negli arterio-sclerotici, nei nefritici, nelle gestanti e negli epilettici per evitare i pericoli di una elevazione della pressione sanguigna. In tutti gl' interventi sul naso, faringe e cavità accessorie, specialmente dopo la tracheotomia preventiva, la cloro-flebo-narcosi può rendere buoni servizi.

Nelle anestesie profonde per operazioni nel naso e cavo è generalmente consigliata la posizione alla Rose o di Trendelenburg, ma ora vi sono chirurgi di larga esperienza che non credono indispensabile ciò, sostenendo che al pericolo che il sangue possa nella narcosi discendere nei bronchi, sempre controllabile ed evitabile, sta di fronte la maggiore emorragia che diminuisce la resistenza dell' organismo contro lo schock operativo. In posizione di Rose o di Trendelenburg è molto più facile che il sangue per le trombe penetri negli orecchi, dando luogo coi materiali settici cui si mescola a complicazioni flogistiche che assumono maggior gravità in individui che, dopo operazioni chirurgiche sul naso e sulla volta, richiedono tamponamenti locali di più o meno lunga durata.

Concludendo diremo che fra gli anestetici al cloroformio sono da preferirsi le miscele di Schleich e quelle di protossido di azoto ed etere; fra i metodi di narcosi i migliori sono quelli di Meltzer e di Kuhne come quelli che lasciano libero il campo operatorio.

Quando si agisce su tumori epiteliali non emorragici ci potremo servire con vantaggio della cloro-flebo-narcosi.

In qualche caso sarà contro-indicata qualunque anestesia generale e dovremo accontentarci di quella locale che a maggior ragione dovrà

essere adoperata negli interventi endo-nasali. Il problema dell' anestesia locale sarebbe presto risolto, se la cocaina e suoi sali non avessero così alto potere tossico e non ci fossero tanto numerosi casi di idiosincrasia.

Gli anestetici locali si adoperano o per iniezioni sottomucosa ed ipodermica a soluzioni terapeutiche o per applicazioni locali in soluzioni concentrate. Invece della cocaina si usano con successo la novocaina, la olocaina, la stovaina e l' alipina, sole od unite all' adrenalina, la quale, oltre all' azione ischemizzante, di incomparabile valore nei nostri interventi, pare che esalti il potere analgesico delle sostanze menzionate.

Indicazioni generiche per il metodo di intervento

I tumori maligni delle fosse nasali possono originarsi in un punto qualunque della pituitaria, ma più sovente si osservano nella zona media o superiore delle fosse nasali, attorno al cornetto medio, al corpo dello sfenoide, ed al livello dell' orifizio anteriore delle cavità accessorie, nel labirinto etmoidale. Riassumendo si possono dividere in neoplasmi del piano inferiore, medio e superiore. Per sciegliere quel modo d' intervento applicabile a qualunque caso, bisogna ben delimitare il punto d' impianto del tumore.

1° I tumori della zona inferiore si possono estrarre dalle vie naturali, grazie all' istrumentazione ricca e variata che si possiede, qualunque sia il volume e l' estensione del tumore. La resezione del mascellare superiore e l' operazione di Rouge saranno riservate per i tumori che hanno sorpassato i limiti delle fosse nasali.

2° Tumori della zona media. Se il tumore di questa regione è piccolo, facile a delimitarsi nei suoi contorni, si può ancora intervenire per le vie naturali. Bisognerà sempre cauterizzare il punto d' impianto con il galvano-cauterio e sorvegliare le recidive durante mesi ed anni. Se il tumore è più voluminoso e più esteso, bisognerà, per fissare la natura dell' intervento, tener conto delle deformazioni esterne. Non sempre è facile rendersi conto se il tumore è accessibile dalle vie naturali, ma si potrà provarne l' estirpazione e si arresteranno le manovre intranasali per ricorrere alla via esterna qualora il volume e la diffusione del tumore lo esigano.

3° Tumori della zona superiore. Quando i neoplasmi nascono nella porzione dell' etmoide che corrisponde all' os planum, all' unguis ed al terzo superiore della lamina perpendicolare di quest' osso, bisogna ricorrere alla estirpazione per via esterna, che solo permetterà di togliere i prolungamenti orbitari o sinusali che esistono quasi sempre.

4° Tumori dei seni in cui bisogna intervenire generosamente dall' esterno.

5° Tumori del cavo naso-faringeo per i quali unica via logica è la esterna.

6° Tumori combinati che reclamano gli interventi più demolitori, sempre dall' esterno.

Medicina operatoria dei tumori delle narici e dell' epifaringe

Il più radicale ed esteso intervento chirurgico per aggredire ed asportare i tumori maligni delle fosse nasali e del cavo naso-faringeo, comprese le cavità accessorie, consiste nella temporanea o permanente resezione di uno o di ambedue i mascellari superiori, eseguendo prima la tracheotomia oppure l' intubazione di Kuhne per evitare i pericoli della pneumonia ab ingestis e coll' allacciatura della carotide comune ed esterna, quando si tema una straordinaria perdita di sangue. La resezione del mascellare superiore fu per prima eseguita nel 1821 da Gensoul e dopo di lui portarono modificazioni al metodo Velpeau, v. Langenbeck, Weber, Kocher ed altri. Secondo Krönlein la mortalità salirebbe al 25,5 per cento e secondo König al 30 per cento nella totale demolizione del mascellare superiore.

Von Langenbeck invece d'asportare completamente il mascellare propone una resezione osteoplastica parziale, facendo un' incisione attraverso le parti molli della guancia, aprendosi una breccia temporanea nel mascellare, sollevando la porzione palatina ed alveolare. Questo processo subì varie modificazioni, le quali possono differenziarsi in trans-mascellare e palatina.

La via palatina fu praticata la prima volta da Nelaton, il quale, dopo di aver distaccato il palato molle ed inciso il periostio di quello duro nella linea mediana, resecava i due terzi posteriori del palato duro per penetrare nelle parti profonde del naso e del naso-faringe.

Gussenbauer invece, dopo aver inciso il periostio lungo la linea mediana, seziona l' osso lungo il bordo alveolare, distaccando la porzione ossea del palato in modo da aprire il pavimento del naso e del seno mascellare. Asportato il tumore, sutura il lembo osteoperiosteo.

Bergmann giudica i precedenti metodi insufficienti.

Chalot consiglia d' incidere il culdisacco del labbro superiore al disopra dei primi due denti molari e di sollevare le parti molli, rasente la spina nasale anteriore inferiore, in modo da scoprire il pavimento delle due cavità nasali. Introduce quindi due cateteri di Nelaton che fa uscire dalla bocca. Alla loro estremità attacca due seghe di Gigli colle quali delimita la resezione del pavimento del naso, poscia con lo scalpello distacca l' inserzione inferiore del setto, in modo che il palato duro possa essere abbassato sulla lingua. Rimosso il tumore il lembo è rimesso in sito e fissato con sutura metallica.

Partsch nel 1898 portò una notevole modificazione al processo di Chalot. Egli fa una incisione trasversa della mucosa nel fornice del labbro superiore ; sollevate le parti molli, penetra nel naso da una delle aperture piriformi, quindi con largo scalpello attraversa il setto e la parete facciale dei seni mascellari e, dirigendosi in senso orizzontale sul pavimento del naso, abbassa l' intero palato sulla lingua. Dopo l' asportazione del tumore sutura il lembo.

Kocher invece divide il labbro superiore con un' incisione perpendi-

colare fino all' apertura nasale e quindi incide rasente il fornice gengivale e con robusto scalpello separa il processo alveolare ed il palato duro dalla parete facciale e mediale dell' antro. Allora con la sega a catena e lo scalpello divide in due il palato duro, ottenendo così un' ampia breccia che domina le cavità nasali ed il cavo faringeo. Dopo tolto il tumore riunisce i due lembi laterali con punti metallici. Dal punto di vista cosmetico tanto i processi di Chalot e Partsch e sopratutto quello di Kocher sono preferibili, non nascondendo però che sono tutti gravi traumi chirurgici.

I metodi nasali per mettere allo scoperto i tumori maligni comprendono non solo la demolizione delle ossa nasali, ma delle pareti; secondo Kummel vanno distinti in metodi osteoplastici con rovesciamento di tutto il naso, ed incompleti che consistono in resezioni temporanee o permanenti della regione nasale superiore.

Linhart incide le parti molli sulla linea mediana, indi con lo scalpello e con la sega di Gigli distacca l' osso nasale e la branca montante del mascellare dal processo frontale, indi con una leva frattura in fuori quest' osso compreso l' unguis. Chassaignac invece circonda con un' incisione la radice del naso dall' uno all' altro angolo interno dell' occhio per scendere al labbro superiore lungo l' ala del naso; indi seziona con lo scalpello o con la sega lo scheletro osseo compreso il setto e rovescia il naso dal lato opposto.

Von Langenbeck pratica un' incisione cutanea, risparmiando il periostio, che dal mezzo della glabella lungo l' osso nasale scende in basso, seguendo l' apertura piriforme, fino all' ala nasale. Indi con la sega, dall' apertura separa le ossa nasali e queste vengono distaccate alla base pure con la sega. Infine, per frattura, allontana le ossa nasali e l' apofisi nasale del mascellare.

Killian pratica un' incisione dalla glabella a mezzo il dorso del naso, scolla il periostio in corrispondenza del seno frontale, disseca l' osso nasale corrispondente e lo rovescia in fuori, lasciandolo aderente al periostio ed alla pelle.

Gussenbauer fa un' incisione cutanea a forma di larga V, aperta in alto, che partendo dalla metà d' un sopraciglio opposto passa con forte curva o convessità inferiore sulla radice del naso; indi pratica con lo scalpello un lembo osseo a forma di cuneo limitato dalle lamine perpendicolari dell' etmoide alla loro congiunzione col vomere e lo rovescia in alto.

Winkler aggiunge due incisioni perpendicolari in alto a quella cutanea del Gussenbauer, in modo da rendere possibile la resezione temporanea della parete anteriore dei due seni frontali.

Kuster risparmia la parte cartilaginea del naso.

Moure incide le parti molli dalla metà della regione intersopracigliare all' ala del naso, mettendo allo scoperto lo scheletro, fino all' unguis, dell' osso nasale, della spina nasale e della spina frontale; ottiene così una larga breccia per l' etmoide. Brockaert completa il metodo di Moure, aprendo il seno mascellare per giungere a quello sfenoidale.

Denker solleva il naso *in toto*, incidendo sul fornice gengivale e distaccando l' osso fino al bordo orbitale.

La via palatina è stata difesa da Manne, Nelaton, Kocher: essa consiste sia in una semplice incisione del velo, sia in una sua combinazione con una resezione mediana del palato osseo. L'inconveniente di questo procedimento è quello di dare uno spazio insufficiente; inoltre se il tumore è grosso bisogna attraversarlo per aggredirne il peduncolo. Il suo vantaggio consiste nella evitata formazione di cicatrice e nel fatto sopratutto che la breccia può essere lasciata aperta per molto tempo in modo da poter sorvegliare le recidive; queste sono molto frequenti ed è assolutamente necessario il preoccuparsene, qualunque sia la via prescelta.

Metodo Faure.—Nei tumori maligni del cavo che si propagano in basso, l' A. consiglia la resezione temporanea o definitiva della branca montante del mascellare inferiore, la cui asportazione dà un largo campo per intervenire nella parete laterale della faringe e nelle tonsille.

Nei cancri dell' epifaringe l' intervento operativo sarà giustificato quando il neoplasma è al suo esordio e si trova ancora nettamente isolato. Sarà controindicata l' operazione allorchè fin dal principio si manifestino gangli aderenti e soprattuto una catena carotidea diffusa.

Medicina operatoria dei tumori maligni del seno mascellare

Nella chirurgia dei tumori maligni del seno mascellare deve essere unica direttiva quella dell' asportazione in toto dell' osso. Le operazioni conservative sono insufficienti ed espongono sempre alle recidive.

I vari metodi di resezione del mascellare superiore, per tumori maligni dell' antro d' Higmoro, non differiscono in genere che per il modo di incisione cutanea.

1° Dieffenbach fa un' incisione verticale mediana che dalla radice del naso arriva a sezionare il labbro superiore, il taglio deve giungere all' osso; dall' estremità superiore si parte un' altra incisione fino all' angolo esterno dell' orbita.

2° Velpeau fa un' incisione normale che dalla commessura labiale, circondando la guancia con convessità posteriore, va all' angolo esterno dell' occhio; a questa incisione Malgaigne aggiunge lo spaccamento del labbro superiore. Questo metodo ha il grave inconveniente di ledere il dotto di Stenone e molti vasi importanti, senza contare che deturpa l' estetica.

3° Weber fa un' incisione fino all' osso che, partendo dalla metà del labbro superiore il quale è sezionato perpendicolarmente, segue il solco naso-labiale e giunto all' angolo interno dell' occhio segue la curva anatomica del bordo orbitario inferiore fino all' angolo esterno.

4° Huguier fa un' incisione simile a quella di Velpeau, ma più esterna, per cui dal lobulo dell' orecchio arriva al labbro superiore.

5° Gensoul fa una prima incisione verticale che dall' angolo interno dell' orbita scende lungo il solco naso-genieno e va a spaccare il labbro

superiore in corrispondenza del canino; quindi ne pratica una seconda che dall' angolo esterno dell' orbita scende in basso. Le due incisioni sono riunite da una terza che partendo dall' ala del naso si dirige indietro.

6° Lisfranc fa due incisioni, di cui una parte dalla commessura labiale, con direzione meno curva di quella di Velpeau e quindi meno pericolosa per il dotto di Stenone, e giunge in alto fino ad un dito dall' apofisi orbitaria esterna del frontale; la seconda seziona il labbro superiore in corrispondenza del canino e rimonta in alto lungo il solco naso-genieno fino all' angolo interno dell' orbita.

7° Michaux, onde non ledere il sacco lacrimale come avviene col metodo di Dieffenbach, fa l' incisione naso-labiale uguale a quella di detto autore, indi sbriglia l' angolo palpebrale esterno con lunga incisione verso il basso e giunge sull' osso, incidendo il sacco congiuntivale della mucosa.

8° Liston fa un' incisione sinuosa che, partendo dall' angolo esterno dell' orbita, circonda a curva il margine inferiore di esso; risale fino all' angolo interno, ridiscende lungo il solco naso-genieno e circonda l' ala del naso, portandosi nella linea mediana, per sezionare perpendicolarmente il labbro superiore.

9° Heyfelder ha un metodo identico al precedente; ne differisce nell' incisione orizzontale che egli pratica dall' interno del sacco congiuntivale; di più, giunto con quella perpendicolare all' ala del naso, scende direttamente in basso.

10° Langenbeck fa un lembo a convessità inferiore che, partendo dalla radice del naso, va scorrendo nel solco naso-genieno, a circondare la guancia, e poi rimonta fino ad un dito in fuori dell' angolo esterno dell' orbita.

11° Durante usa la incisione di Liston, però fa passare l' incisione orizzontale nel culdisacco congiuntivale per impedire l' edema della palpebra, inevitabile quando essa si recide alla sua base.

Qualunque sia il processo di incisione delle parti molli, la sezione ossea è uguale per tutti. Si deve operare con la narcosi generale ed in posizione di Rose, per impedire che il sangue penetri nelle vie aeree, a meno che non si sia proceduto ad una tracheotomia preliminare ed al tamponamento della faringe. Rende buon servizio in questo atto operativo la intubazione alla Kuhne per la narcosi.

Verneuil, sempre nell' intento di non permettere la caduta di sangue in trachea, preferisce di incidere la mucosa orale dopo aver scoperto l' osso da asportare. Durante non è di tale avviso e pratica il taglio dei tessuti molli in un tempo, cercando beninteso di procedere rapidamente nell' intervento, giacchè la rapidità è la più forte ragione di successo.

Fatto il taglio delle parti molli si denuda l' osso in corrispondenza delle sue tre facce anteriore, esterna e superiore; indi si passa al distacco del malare, che si può fare o con lo scalpello o con la sega del Gigli, la quale si farà entrare nella fessura sfenomascellare ed uscire al disotto dell' osso, poi si manovrerà tirando dalla profondità alla superficie. Il tempo successivo al distacco nasale si praticherà o con lo scalpello

o meglio con una pinza osteovora, separando l' apofisi montante dal mascellare. Il distacco palatino infine si compirà previa sezione del palato molle, rasente a quello duro ; la mucosa di questo, se è possibile, dato il processo morboso, deve essere conservata per poter servire da setto buccо-nasale ; la sezione ossea si fa con lo scalpello o con pinza osteovora. Il distacco pterigoideo infine si farà con una forbice ad osso curva sul piatto, introdotta nella cavità orale, in modo che il tagliente vada dietro e rasente alla tuberosità del mascellare. Compiuto così l' isolamento dell' osso con la pinza di Farabeuf e con movimenti di torsione, onde vincere le ultime connessioni, si estrae la massa ossea. L' emorragia a questo punto imponentissima si frena col tamponamento. Si suturano quindi le parti molli e la mucosa, se si potè risparmiare quella ricoprente il palato duro.

Le protesi praticate negli interventi chirurgici della faccia hanno una grande importanza non solo per riparare all' estetica, ma per impedire gravissimi disturbi vitali, come il passaggio di materiale dal naso nella bocca e viceversa, per rendere possibile la masticazione e la deglutizione, per migliorare il timbro della voce.

Berget è d' opinione che la protesi metallica può rendere buoni servizi, ma è da consigliare solo quando non è possibile l' autoplastica.

Medicina operatoria dei tumori maligni dei seni frontali

1° Metodo di Killian. Incisione cutanea che va dall' estremità esterna del sopracciglio alla parte mediana della radice del naso, per scendere fino ad incrociare l' apofisi montante del mascellare. Si asporta quindi tutta la parete esterna del seno e parte dell' apofisi montante dell' osso nasale. A questo modo si rende possibile eziandio l' esplorazione dell' etmoide.

2° Metodo di Laurens. Incisione cutanea lungo tutto il sopracciglio e demolizione ossea comprendente tutta la parete anteriore del seno, l' arcata sopraorbitaria e la parete inferiore ed orbitaria del seno stesso.

Nell' asportazione dei tumori del seno frontale sarà desiderabile che l' estetica del viso non rimanga deturpata, per cui i chirurgi si sono preoccupati che le incisioni non fossero troppo appariscenti. Se per la estensione del tumore dovrà scoprirsi largamente l' osso e non fosse sufficiente l' incisione sopraorbitaria, l' unica sussidiaria da consigliare è quella perpendicolare sulla fronte. Però nella maggioranza dei casi, anche per tumori molto estesi, gli autori ormai ritengono sufficiente l' incisione curvilinea sul sopraciglio, per cui si rendono inutili i tagli ad angolo retto di Verneuil, il taglio a croce di Dolbeau, quello ad L di Lucas Championnière e quello a C di Boryd, il quale ultimo però è l' unico che possa sopperire alla bisogna negli enormi osteomi del seno frontale.

Come ben sappiamo, i tumori primitivi dei seni frontali sono rarissimi ; per lo più non sono che la propagazione di quelli iniziatisi nell' etmoide. Nei processi operatorii che dovranno adunque compiersi sul seno frontale, non sarà molto raro che si abbia da intervenire anche sull' etmoide.

Dato però che il tumore sia primitivo del seno frontale, allora ricorreremo ai processi classici a larga apertura del seno frontale e secondo i metodi di Killian e Laurens.

Medicina operatoria dei tumori maligni dell' etmoide

I tumori maligni dell' etmoide, sia che abbiano origine dall' osso spongioso, sia che provengano dalla mucosa, hanno tendenza tanto ad occludere le fosse nasali, quanto a far prominenza nell' orbita. Lo spostamento del bulbo oculare sarà verso l' infuori ed alquanto in alto ed in basso, a seconda che il tumore prema verso la parte inferiore o verso la parte superiore della parete interna orbitale. Il deturpamento del viso e la diminuzione di funzione dell' occhio sono molto solleciti e sono fra le cause non trascurabili che consigliano d' operare al più presto possibile, prima che il chirurgo sia indotto dallo sviluppo del tumore verso la base del cranio a scoprire anche l' encefalo. Le due vie per estirpare i tumori maligni primitivi dell' etmoide sono: l' orbitale e la nasale.

Metodo orbitale

1° Processo di Maisonneuve: pratica un taglio a C che segue tutta la metà interna del contorno dell' orbita, cominciando subito dopo il sopraciglio.

2° Processo di Maisonneuve: pratica prima un taglio verticale sulla bozza nasale e poi un taglio parallelo al sopraciglio ed al disopra di questo, che comincia alla radice del naso e termina alla tempia.

3° Processo di Sprengel: dalla glabella fa scendere in basso ed infuori un taglio che incontri sotto il sopraciglio il primitivo taglio di Maisonneuve, costituendo così una specie di Y.

4° Processo di Andrews: fa un taglio curvilineo che, partito dal mezzo della radice del naso, si porta al centro del bordo orbitale inferiore.

Metodo nasale

1° Processo di Mott: fa un' incisione verticale che dalla bozza nasale, un centimetro e mezzo sul sopraciglio, scende in basso lungo il solco naso-genieno fino alla pinna: ad essa è aggiunto un taglio trasversale che dal mezzo della palpebra superiore del lato malato si porta fino all' angolo interno dell' occhio del lato opposto.

2° Processo di Lenoù: s' incide il naso sulla linea mediana ed asporta l' osso nasale del lato infermo.

Nei tumori maligni dell' etmoide Moure preferisce il proprio processo, descritto nella medicina operatoria sfenoidale come il più semplice, senza gravità ed in pari tempo più che sufficiente ad aggredire in toto il corpo dell' etmoide ed estirparlo anche completamente. È facile infatti, secondo lui, con una sgorbia da avanti indietro seguire la lamina cribrosa fino a livello del corpo dello sfenoide e nei casi di diffusione al seno sfenoidale è possibile penetrarvi.

Riguardo al trattamento dei tumori maligni dell' etmoide vi sono

coloro che propendono, quando è possibile, per l'asportazione completa attraverso le vie naturali senza operazioni esterne (Moure). Altri invece credono poter ancor seguire le vie naturali fino a quando si tratta di un tumore unicamente nasale, ma non più nel caso di propagazione ai seni (Garel). Altri infine rifiutano qualsiasi intervento endonasale, considerandolo come un colpo di frusta destinato a favorire la diffusione del tumore (Castex). Il sollevamento della faccia che passa in rinologia come metodo di Goris fu proposto per la prima volta nel 1898 da Bardenhauer di Colonia allo scopo di avere una via larga verso la parte profonda del naso senza fare alcuna incisione alla faccia. Questa operazione è utilissima, specialmente nei tumori etmoido-mascellari, e siccome ha il vantaggio di non lasciare traccia è preferibile nelle donne e nei giovani non a completo sviluppo.

Guisez sistematicamente ha adottato per l' apertura del labirinto etmoidale la via orbitaria. L' incisione cutanea parte dal terzo interno del sopraciglio e si ricurva a mezzo centimetro indentro dell' angolo interno dell' occhio. Il globo oculare ed il sacco lacrimale sono reclinati. Asporta l' os planum e l' unguis, reseca un centimetro della branca montante del mascellare superiore con porzione dell' osso nasale. Per questa via si può vuotare completamente l' etmoide e successivamente anche il seno sfenoidale. Secondo l'A. questa sarebbe la via più diretta e più corta di quelle proposte. Collet invece sostiene che il vuotamento dell' etmoide può farsi benissimo anche dalla via endo-nasale, purchè si adoperi per l' asportazione del cornetto medio ed il vuotamento delle cellule la pinza di Luc, cui Merck ha avuto l' idea di associare l' articolazione della pinza di Grünwald.

3° Metodo di Piquèt-Taubert.—La tecnica di questi autori è da consigliarsi in quei tumori che abbiano invaso diversi seni dello stesso lato. Si fa un' incisione nel sopraciglio del lato malato e giunti alla radice del naso si scende in basso lungo il solco naso-labiale. Si apre il seno frontale e si resecano le ossa nasali dal basso in alto. A questo modo è possibile l' intervenire nelle cavità nasali e contemporaneamente sui seni: frontale, mascellare, etmoidale, sfenoidale.

Medicina operatoria dei tumori maligni del seno sfenoidale

La topografia del seno sfenoidale avrebbe fatto pensare che non fossero aggredibili i tumori indovati in questa cavità profonda, ma chirurgi generici e specialisti hanno dimostrato che ciò è possibile. I metodi sono tre, a seconda che si prenda la via nasale, orbitale o faringea.

Via nasale

1° Processo Ippocratico.—Consiste nel taglio mediano della parte cartilaginea del naso, e nel rovesciare la pinna così divisa. Resecando il turbinato medio e respingendo al lato opposto il setto, si può giungere ad aprire la parete anteriore di uno dei seni sfenoidali.

2° Processo Padula.—Al taglio Ippocratico mediano di tutto il naso, protratto fino all' angolo naso-frontale, si fa seguire il distacco dell' osso nasale corrispondente presso la linea mediana e la frattura dell' apofisi montante del mascellare superiore. Per distaccare l'osso nasale dal frontale in corrispondenza della sutura è necessario con un piccolo scalpello aprire la sutura naso-frontale. Allora con una forte pinza, le cui branche sono coperte di caoutchouc, si frattura l' apofisi montante del mascellare superiore che è rovesciata con il lembo. Nel caso che l' apofisi opponga resistenza, dopo di aver rovesciato il lembo, se ne scalpellerà lo spigolo dal dentro in fuori e dal sotto in sopra, in modo da poterne fratturare una porzione sufficiente per avere un' ampia breccia che ponga in evidenza la fossa nasale.

3° Processo Langenbeck.—Si praticano due tagli verticali, uno nella linea mediana e l' altro lungo l' orlo interno dell' orbita ed il solco naso-genieno, i quali si arresteranno all' impianto della cartilagine della pinna sulle ossa nasali; quivi saranno congiunti da un taglio curvilineo che segna l' impianto della cartilagine. Occorrendo maggiore spazio, tutta la pinna può essere compressa fra i due tagli; poscia con lo scalpello si pratica l' osteotomia lineare lungo i due tagli verticali, s' incide la mucosa sottostante e si ribatte in alto il lembo quadrilatero, distanziando le ossa nel punto più alto possibile.

4° Processo Boeckel.—Si fa un taglio nel solco naso-genieno che dall' angolo interno dell' occhio finisce alla base della pinna. Dall' estremo superiore di detto taglio se ne traccia un altro orizzontale, che girando sul dorso del naso giunge al punto analogo del lato opposto. Seguendo queste due incisioni si scalpella l' impalcatura del naso e poi con robusto coltello si recide verticalmente il setto nasale, separandolo anche dal pavimento con le forbici di Liston. Allora con una pinza a lunghe branche protette di caoutchouc, l'una introdotta nella narice e l' altra nella guancia del lato opposto, si frattura verticalmente l' apofisi montante del mascellare superiore e si reclina lateralmente il naso. In tal guisa ambedue le cavità nasali sono ampiamente visibili ed, ove occorra, asporteremo i turbinati inferiori per scoprire il corpo dello sfenoide, la parte inferiore della parete basilare occipitale ed il fornice faringeo.

5° Processo Laurence.—Consiste in due tagli verticali lungo i solchi naso-genieni, congiunti da uno orizzontale tracciato tra la base del naso ed il labbro superiore. Si dividono le apofisi montanti con lo scalpello sul fondo dei tagli verticali; si recide il setto con un taglio orizzontale ed uno verticale e quindi si rovescia sulla fronte tutto il naso, facendone lussare o fratturare le ossa presso la sutura fronto-nasale.

6° Processo Ollier.—Tenendo conto che le arterie principali vengono dal basso (arteria del sotto setto, arteria dell' ala del naso ecc.), Ollier fa un' incisione a V rovesciato. Comincia un poco indietro del bordo posteriore dell' ala nasale a destra, va in alto ed in dentro fino all' angolo naso-frontale e poi traverso la linea mediana ridiscende con direzione contraria al punto omologo di sinistra. L' impalcatura ossea del naso

sarà scalpellata lungo l' incisione cutanea, oppure si toglierà colla sega del Gigli. Reclinato in basso il naso si asportano i turbinati, si demolisce il setto e così si arriva a scoprire la parete anteriore dello sfenoide.

7° Processo naso-mascellare di Langenbeck.—Si sezionano i tegumenti con due incisioni che si uniscono ad angolo acuto nel ponte zigomatico : la prima parte dall' ala del naso, la seconda dall' angolo interno dell' orbita. Le pareti molli vanno incise fino all' osso, ma non si scollano : con la sega o lo scalpello si scontinua l' osso secondo il taglio inferiore ; indi si separano successivamente il ponte zigomatico, l' apofisi montante del mascellare con il pavimento dell' orbita, e con una robusta pinza si rovescia tutto il lembo osteo-periosteo in alto.

8° Processo naso-orbito-mascellare di Boeckel.—Si fa un' incisione che segue la curva anatomica del solco naso-genieno e giunta all' ala del naso si dirige in fuori per due dita ed anche più ; in alto l' incisione segue il bordo orbitario inferiore fino al forame sotto orbitario ; ne risulta così una linea di taglio a forma di berretto frigio con base posteriore-esterna. Con la sega a catena o lo scalpello si separano successivamente l' apofisi montante del mascellare, il mascellare superiore rasente il pavimento delle fosse nasali, l' osso malare ed il pavimento dell' orbita. Il lembo così delimitato si rovescia in fuori e verso la linea mediana. Si demolisce in seguito la parete interna del seno mascellare ed i turbinati.

9° Processo di Roux.—Si pratica un' incisione che, partendo dall' angolo esterno dell' orbita, ne segue il contorno inferiore ; indi raggiunto l' angolo interno, si dirige in basso, circonda l' ala del naso fino alla linea mediana e passa il labbro superiore. Il mascellare è scontinuato lungo la volta palatina. Per sezionare il malare si fa una piccola incisione verticale in corrispondenza di quest' osso, in modo che sia possibile l' introduzione della sega a catena. Il lembo viene rovesciato come il precedente.

10° Processo di Jansen.—Consiste nell' apertura del seno mascellare dalla fossa canina, demolizione ossea dell' angolo formato dalla parete superiore con la interna e posteriore dell' antro, larga breccia nelle cellule etmoidali posteriori. A questo modo si giunge sui seni sfenoidali, la cui parete antero-inferiore viene rimossa col cucchiaio o con pinze speciali.

11° Il processo Finret differisce dal precedente, perchè raggiunge il seno sfenoidale passando dall' interno dell' antro e risparmiando l' etmoide.

Via orbitale

Bergh, nel caso che il tumore abbia invaso l'etmoide e lo sfenoide ed abbia già compromesso il bulbo dell' occhio, trova giustificato di operare simultaneamente sullo sfenoide e sull' etmoide. Perciò vuota lo sfenoide con uno dei processi innanzi indicati fra i metodi nasali, enuclea il bulbo, recide la lamina papiracea dell' etmoide e poi applica un drenaggio che dall' orbita raggiunge la faringe. Il metodo di Bergh, che data dal 1886, fu ripreso da Knapp nel 1893, lasciando in sito il globo oculare. In

seguito Goris, Chipault, Laurens e Guisez ne perfezionarono la tecnica. Si pratica un' incisione curvilinea che, partendo dalla metà del sopraciglio, circonda l' angolo interno a 4 o 5 mm. dalla commessura palpebrale e seguendo il contorno inferiore dell' orbita si arresta al suo quarto interno. Questa incisione interesserà le parti molli fino al periostio. Gli organi della parete esterna dell' orbita, i tendini dell' orbicolare, il muscolo di Horner e il sacco lacrimale devono essere delicatamente spostati all' esterno. A questo punto con un sottile staccaperiostio si scopre l' unguis e la lamina papiracea dell' etmoide e con la sgorbia la si apre prudentemente ; infine col cucchiaio si vuota l' etmoide in modo da giungere a scoprire il seno sfenoidale.

Negli interventi del seno sfenoidale, tanto che si tratti di suppurazione quanto di asportazione di tumori, l'apertura della parete anteriore del seno deve essere più ampia che sia possibile, anche per permettere la sorveglianza della cavità (Panmore, Berens).

La scelta della via d' accesso al seno sfenoidale per l' asportazione dei tumori maligni è molte volte subordinata alla direzione cui tendono le propaggini del tumore nell' invadere le regioni vicine. Alcune complicazioni endo- ed esocraniche, come la meningite, la trombosi del seno cavernoso e dell' oftalmica, la nevrite ottica ed il flemmone dell' orbita ecc., che dipendono dai rapporti anatomici del seno sfenoidale, consiglieranno alle volte di scegliere un metodo o l' altro operativo. Tutte le operazioni, che si propongono di aggredire il seno sfenoidale dalla faccia laterale, inferiore ed anteriore, hanno per lo più il difetto comune di fare una breccia abbastanza grande in basso, dove l'operatore non ne ha gran bisogno, e viceversa di dar poca luce in alto dove è la necessità maggiore d'agire.

Comparando la via d' accesso orbito-etmoidale con quella mascellare, deve notarsi che nella prima l' operatore non riscontra che una sola obliquità indentro ed indietro nel piano orizzontale, che viene soppressa resecando la branca montante del mascellare superiore, e l' osso proprio del naso ; nella seconda l' obliquità invece è doppia dal di fuori all' indentro e dal basso in alto. Resecando l' angolo antero-interno del seno mascellare non si sopprime che una di queste obliquità, cioè quella dal di fuori all' indentro, ma persiste l' obliquità dal basso in alto. Ciò costituisce della mascellare una via più complessa che quella etmoidale per raggiungere i tumori sfenoidali.

Via naso-mascellare

1° Processo di Jean-Louis Faure.—Primo tempo: incisione cutanea trasversale fino all' osso, di 2 o 3 cm., sul margine inferiore dell' occhio. Indi altra incisione che dalla estremità interna della prima sotto l' angolo dell' occhio scende verticalmente, seguendo il solco naso-genieno, fino alla inserzione della narice corrispondente che si trova così completamente distaccata. Questa incisione verticale può alle volte essere sufficiente, l' incisione sottorbitale essendo inutile o ridotta ad un cm. appena.

Secondo tempo: scollamento del lembo cutaneo. Terzo tempo: sezione ossea: s' ingrandisce d' un cm. circa di fuori ed in alto l' orificio anteriore della fossa nasale. Successivamente soppressione della parete interna del seno mascellare ed asportazione del cornetto inferiore e di buona parte del setto osteo-cartilagineo nasale. Quarto tempo: asportazione del tumore e sutura esterna con punti staccati.

2° Processo di Moure.—Si scolla il naso con un' incisione fatta dall' angolo interno dell' occhio fino alla radice di esso e poi si pone a nudo con lo stacca-periostio l' osso proprio nasale e la parete anteriore del mascellare superiore. Per scoprire convenientemente questa superficie ossea bisogna praticare perpendicolarmente alla prima un' incisione che segue il bordo orbitario. Si seziona in seguito la branca montante del mascellare superiore, rasente il pavimento del naso, con una pinza tenaglia, e poi si divide con una sgorbia il seno in due parti, incidendo il bordo orbitario. Ciò permetterà di tagliare tutta la parete esterna della fossa nasale, compreso il cornetto superiore. Si avrà così un' ampia cavità nella quale il seno è aperto, il che aumenta un poco di più le dimensioni della breccia verso la parete esterna. L' osso proprio del naso sarà resecato, dopo avere reclinato il canale naso-lacrimale membranoso, in guisa da evitarne la sezione. Quest' operazione non solo offre grande spazio nelle fosse nasali, ma permette di scoprire quasi per intero la parete naso-faringea, dimodochè sarà più facile asportare i tumori del seno sfenoidale. Lo spazio così ottenuto sarà ancor più grande in ragione del notevole sfiancamento che di solito subisce il cavo sfenoidale per il fatto stesso dell' esistenza del tumore che bisogna estrarre.

Alle volte nel vuotamento dei tumori maligni dei seni sfenoidali può sopravvenire tale abbondante emorragia da essere necessaria la legatura provvisoria della carotide primitiva (Robert C. Myles).

3° Processo di Luc et Fauret.—Resezione completa della parete anteriore del seno mascellare fino al setto naso-sinusale, che viene successivamente asportato con la pinza *emporte-pièces* di Lombard. Si continua la resezione della faccia anteriore del seno mascellare fino ad abraderne l' angolo antero-interno; si ha così una larga apertura imbutiforme d' accesso a tutta la faccia anteriore dello sfenoide. Allora si aggredisce con la sgorbia la parte inferiore interna della faccia anteriore del seno sfenoidale, immediatamente al disopra della coana, dove non si corre alcun rischio.

Via nasale

Metodo Vacher.—La via naturale del naso è stata anche tentata per penetrare nel seno sfenoidale in quei casi in cui per l' ampiezza delle narici (ozenatosi, eredo-sifilitici, processi distruttivi dell' osso) la faccia anteriore del seno apparisce nettamente nel campo dello speculum.

1° tempo: ottenuta l' anestesia si dilata al maximum l' apertura delle narici e si procede fino all' osso con un' incisione che segue il solco nasale, dall' alto in basso, dal fuori in dentro, fino al terzo della parete

inferiore. 2° tempo: si distaccano rasendo l' osso tutti i tessuti con uno stacca-periostio di dentro o di fuori. Si pone così a nudo il solco mascellare per una larghezza di otto a dieci mm. ed un' altezza di dodici a quindici mm. 3° tempo: si fa saltare questa parete ossea con lo scalpello o con la pinza tagliente, in modo da fare una larga apertura che permetta di porre allo scoperto la parete anteriore del seno sfenoidale.

Nella chirurgia dello sfenoide si possono utilizzare con vantaggio alcuni metodi operativi ideati per aggredire l' ipofisi.

1°. Processo transetmoidale del Giordano. Questo A. delimita un lembo osteoplastico con base ai lati delle pinne nasali ed abbassa tutto lo scheletro del naso, indi vuota l' etmoide in modo da scoprire lo sfenoide.

2°. Processo con vuotamento dell' orbita di Schlösser (molto aggressivo). Si inizia con una rinotomia laterale alla Chassaignac, indi si prolunga l' incisione superiore lungo il bordo orbitario inferiore fin oltre l'angolo esterno dell' occhio, si asporta la parete orbitaria interna; poi si isola l' osso malare che si porta infuori assieme alla base dell' orbita svuotata; quindi si asporta l' etmoide e si scopre lo sfenoide (questa operazione non fu praticata nell' uomo).

3°. Processo nasale modificato di Schlösser. È meno aggressivo del precedente. Rinotomia laterale; asportazione dei cornetti, del setto, della parete orbitaria interna e di quella del seno mascellare. Si vuota quindi l' etmoide e si scopre lo sfenoide.

4°. Processo nasale modificato di V. Eiselberg.—Si pratica la rinotomia laterale come nei processi precedenti. Si aggiunge però un' incisione nel mezzo della fronte per potere asportare la parete anteriore del seno frontale, si reseca il setto ed i turbinati e si vuota l' etmoide per giungere nello sfenoide. In qualche intervento più recente l' Eiselberg ha praticato un' incisione che, seguendo la curva anatomica, va dal mezzo di un sopraciglio al mezzo dell' altro in modo da poter formare un lembo osteoplastico, indi ha eseguito la rinotomia e tutti gli altri tempi dell' operazione prima descritta.

5°. Processo nasale modificato di Hochenegg.—Consiste nella rinotomia laterale secondo Chassaignac a cui si aggiungono due incisioni perpendicolari che vanno in alto nella fronte, partendo dall' interno delle sopraciglia, in modo da determinare un lembo quadrato che viene sollevato assieme alla parete ossea del seno frontale. A questo modo si abbrevia la distanza dello sfenoide. Nel resto l' operazione è uguale a quella consigliata da Eiselberg.

6°. Processo nasale modificato di Praul.—Si pratica prima una rinotomia verticale all' Ollier, ribattendo il naso sulla bocca, indi una incisione verticale alla Eiselberg per resecare la parete del seno frontale e si continua come nei processi precedenti.

7°. Processo nasale modificato di Cushing.—È identico a quello di Hochenegg e di Eiselberg, ne differisce per la forma del lembo. Questo A. pratica un' incisione uguale a quella di Giordano, per quanto riguarda il naso, indi pratica un taglio a forma di omega i cui piedi arrivano agli

angoli orbitari interni. A questo modo si delimita un lembo osteoplastico frontale che è ribattuto in basso col naso.

8°. Processo nasale modificato di Dialti.—Si fa un' incisione mediana nel dorso del naso, che alla glabella si biforca seguendo le due sopracciglia fino al terzo medio. Si pratica quindi la rinotomia mediana e si ribatte in alto un lembo osteoplastico comprendente la parete anteriore dei seni frontali. Si continua poi come nei processi descritti.

9°. Processo nasale modificato di Kocher.—Incisione che parte dalla glabella e, giunta al limite inferiore delle ossa nasali, si biforca, delimitando le parti molli del naso che vengono ribattute in basso, previa eccezione del setto. Divarica poi lo scheletro superiore del naso e reseca il setto in alto; quindi con un robusto speculum schiaccia contro la parete i cornetti medii e superiori e giunge nello sfenoide, risparmiando gran parte dell' etmoide ed i seni frontali.

10°. Processo nasale modificato di Karavel.—Incisione ad U circondante il naso con convessità nel labbro superiore. Si rovescia il naso in alto, si distrugge la parte inferiore del setto e la parte posteriore dell' etmoide; così si scopre lo sfenoide.

Via endonasale

Comprende due processi: di Hirsch e di West, che sono però adattabili al trattamento dei tumori maligni. Essi consistono, con lievi modificazioni uno dall' altro, nell' asportazione dei turbinati, nel vuotamento dell' etmoide, nella resezione della parte postero-superiore del setto.

Via bucco-nasale

Processo di Löwe.—Si rialza tutta la maschera cutanea della faccia con un' incisione del fornice gengivale superiore; indi si separano le branche montanti mascellari e le ossa nasali, in modo da rendere possibile il distacco del naso; si reseca infine la parete interna dei seni mascellari. Fatto ciò si pratica un taglio cutaneo a T con la branca orizzontale lungo le sopracciglia, la verticale sul dorso del naso. A questa maniera, per il distacco osseo fatto prima, si apre una larga breccia che permette il vuotamento del naso e l' esplorazione della sella turcica.

Via mascellare

1°. Processo di Kocher.—Non è mai stato praticato nell' uomo, ma consiste nella sezione media del naso e nella formazione di un lembo osteoplastico che corrisponde al mascellare superiore ed al malare di un solo lato.

2°. Processo di Hertle.—È uguale al precedente, ma bilaterale.

3°. Processo di Ruggi.—Pratica una resezione temporanea del mascellare superiore colla tecnica di Nelaton e ribatte all' esterno il lembo facciale del mascellare; a questo modo giunge allo sfenoide e nella sella turcica. Il processo riesce meglio se si demolisce un tratto di setto (vomere, lamina perpendicolare dell' etmoide).

Via palatina

1°. Processo Durante.—Si fa un' incisione a ferro di cavallo che aperta posteriormente costeggia la base dei processi alveolari; indi si distacca la mucosa col periostio e qualche isola ossea del palato duro e si ribatte tutto il lembo verso il palato molle; poi si demolisce il palato duro e si scoprono le fosse nasali. Si asportano i turbinati e si distacca il vomere, dopo aver sollevato la mucosa che lo riveste. Con uno scalpello quindi a curva adatta si aprono i seni sfenoidali.

2°. Processo di König.—Eseguito solo sul cadavere. Si fa un' incisione mediana nel palato duro, si demolisce il palato e poi si procede come nel processo di Durante.

3°. Processo di Kropus.—Disseca in mezzo la mandibola, poi delimita un lembo osteoperiosteo del palato duro che rovescia indietro, demolisce le ossa palatine e procede come sopra.

Via sopraioidea

Processo di Löwe.—Si pratica una faringotomia sopraioidea, s'incide il palato molle e si dominano così i seni sfenoidali.

Nei voluminosi tumori della base del cranio a facilitare la loro asportazione non solo converrà procedere alla divisione del velo palatino (praticata per la prima volta da Manne d'Avignone nel 1711), ma eseguire eziandio una preliminare tracheotomia con tamponamento della faringe.

Riguardo alle complicazioni intracraniche post-operative, dalle numerose osservazioni sparse in parecchie pubblicazioni sui tumori maligni nasali e delle cavità accessorie si può dedurre che le più frequenti si riferiscono al seno frontale, poi al seno sfenoidale ed infine alle cellule etmoidali.

Prognosi dei tumori maligni delle fosse nasali

In massima i tumori del naso hanno prognosi gravissima per la facile loro invasione nella cavità cranica.

I tumori, quando sono penetrati nelle cellule etmoidali, di rado possono esser rimossi totalmente anche con interventi ultra-demolitori.

La prognosi degli epiteliomi è assai più grave di quella dei sarcomi nasali. In genere le più ampie operazioni nei tumori nasali possono prolungare la vita di qualche anno.

Gli interventi nelle narici e nel cavo naso-faringeo hanno dato occasione ad un numero veramente grande di metodi, di cui volentieri si sono riportati i buoni risultati. Gli esiti infausti invece sono in genere taciuti, per cui acquistano sempre più importanza quei dati che pochi ebbero la pazienza di raccogliere nella letteratura attorno alla mortalità che raggiungono i tumori maligni naso-faringei operati, sia in rapporto alla natura istologica del neoplasma, sia in rapporto al momento scelto per agire chirurgicamente.

Se infatti in molti casi l' estirpazione di sarcomi e carcinomi risultò fatale a breve scadenza dall' intervento, ciò forse accadde perchè il tumore era così avanzato che era da aspettarsi una morte più o meno sollecita, e probabilmente essa fu affrettata dall' operazione. Francis R. Packard ebbe la pazienza di compilare una tabella istruttiva, completando quelle già pubblicate precedentemente da Holloway nel 1896 e da F. W. Hinkle nel 1898. Risulta anzitutto che sopra 26 morti per l'uso dell' anestesia generale 24 perirono per cloroformio, uno per la miscela di cloroformio ed etere, ed uno per l' etere. L' amministrazione del cloroformio sarà sopratutto controindicata nelle operazioni naso-faringee praticate sui bambini, avendo Hinkle osservato che queste morti erano avvenute con maggiore frequenza in pazienti di 'habitus linfaticus'. Tredici dei casi morti per cloroformio furono segnalati nella prima decade della vita.

Sopratutto nei bambini dovremo tener conto se esiste eredità emofillica, essendo l' emorragie post-operatorie una delle maggiori cause di mortalità nell' infanzia. In questi casi prima d' intervenire dovremo assoggettare i giovani pazienti all' uso del cloruro di calce.

Price-Brown ha raccolta la statistica di tutti i casi di sarcomi del naso operati tanto internamente quanto esternamente, in cui i pazienti furono potuti tenere lungo tempo sotto osservazione:

22—malato naso destro, turbinato.
29—malato naso sinistro, turbinato.
9—malati ambedue i cornetti.
11—malato il setto.
27—malate le cavità accessorie nasali.
19—operazioni endo-nasali.
13—recidive.
33—operazioni extra-nasali.
25—recidive.
38—morti.

In parecchi di questi casi l' intervento fu nello stesso tempo endo- ed extra-nasale. La guarigione permanente dopo interventi endo-nasali fu di 6 su 19, cioè del 31 %, la definitiva guarigione dopo operazioni extra-nasali 8 volte su 33, cioè del 24 %; la totale e permanente guarigione dopo operazioni di qualsiasi genere fu di 13 su 51, cioè 27 %.

Le statistiche sono concordi, è vero, nel giudicare fra i tumori maligni del naso-faringe assai più comuni i sarcomi degli epiteliomi (ad es. Laval ha raccolto in apposite tabelle 45 osservazioni dei primi e 25 dei secondi), ma in pari tempo nel riconoscere più favorevoli i risultati prognostici degli interventi radicali e solleciti nei sarcomi. Tanto che Laval è d'avviso che, vane essendosi dimostrate nei carcinomi le eseresi più larghe oltre i limiti del male, sia meglio accontentarsi di prolungare la vita al paziente ed attenuarne le sofferenze, asportando il più possibile del tumore per le vie naturali.

Anche nei casi semplicemente sospetti, come neoplasmi maligni, un sollecito e radicale intervento operativo sarà sempre giustificato, essendosi in questi ultimi tempi accresciuto il numero dei fautori che sostengono, più che rara, abbastanza frequente la degenerazione di forme neoplastiche benigne preesistenti. Tanturri ha descritto istologicamente un caso di lenta trasformazione carcinomatosa di un papilloma della fossa nasale sinistra, forse d' origine etmoidale, riconosciuto di tale natura soltanto dopo l' asportazione per la via endo-nasale. È probabile che la differenza di gravità prognostica in genere tra sarcomi ed epiteliomi del naso e della volta faringea non sia reale, dovendosi buona parte dei così detti sarcomi nasali falcidiare dalle statistiche ed ascriverli piuttosto per i loro caratteri istologici alle epulidi, ai granulomi, agli endoteliomi sarcomatosi, ai sarcoidi.

Al Congresso della Società Italiana di Laringologia tenutosi nel 1905 fu aperta una importante discussione a proposito di un mio lavoro di critica sui sarcomi endo-nasali, nel quale sostenevo che alcuni di quei tumori, che sorgono dai turbinati ed acquistano un certo volume, non sono in realtà sarcomi, ma granulazioni sviluppatesi da monconi residui d'un primo atto operativo, a cui la deficienza di tessuto interstiziale non ancora organizzato fa prendere l' aspetto sarcomatoso. Altre volte poi sono fibromi veri e propri in cui il tessuto interstiziale invece di subire la degenerazione mixomatosa ha subito quella jalina ; di modo che la durezza e vascolarizzazione loro può ingenerare il sospetto che si tratti di sarcomi, il che è poi smentito dall' esame istologico. È anche probabile che alcuni di questi tumori nasali, che all' esame istologico sembrano dei sarcomi, sieno epulidi, cioè tumori formati in prevalenza da tessuto embrionale.

Dall' osservazione clinica si deduce che nei sarcomi, dimostrati tali istologicamente, la malignità, oltre che dal tessuto su cui s' impiantano, dipende pure dalle regioni in cui si sviluppano. Forse i sarcomi prodottisi nelle cavità aperte riescono meno maligni di quelli sorgenti in cavità interne o nel parenchima dei tessuti, quantunque le condizioni sembrino più svantaggiose ai primi, per il più facile contatto con l' aria e con il pulviscolo atmosferico e quindi per la maggiore facilità di subire l'azione di elementi infettivi. Così, ad esempio, non si può certo paragonare la malignità del sarcoma nasale a quella del melanosarcoma della coroide, il quale, anche se aggredito in primo tempo, non garantisce dalle recidive nemmeno dopo parecchi anni.

Queste differenze di decorso e di comportamento ci fanno dubitare che alcuni dei così detti sarcomi nasali siano dei veri e propri sarcomi ; nella stessa guisa che il sarcoma idiopatico multiplo della cute, il quale per la sua relativa benignità ha molti punti di contatto con quello del naso, da molti si ammette che non sia veramente tale in origine, ma che le cellule fusate o rotonde provengano da un' infiltrazione flogistica che fa assumere al tumore la forma sarcomatosa. Talchè sarebbe più razionale includere tali neoformazioni in quella classe di tumori non

ancora ben definiti istologicamente ed etiologicamente, vari per il decorso e per i sintomi, cui alcuni patologi hanno dato il nome di sarcoidi.

Da ultimo i così detti sarcomi nasali potrebbero bene essere degli endoteliomi veri e propri sviluppatisi dalle cellule delle lacune linfatiche del connettivo. Infatti noi sappiamo che talora le cellule dello stroma di tali tumori si addensano in tal guisa da far pensare a prima vista ad un vero e proprio sarcoma e che tal' altra per proliferazioni dell' avventizia dei vasi del neoplasma si forma attorno ad essi un manicotto di cellule fusate che danno al tumore l' aspetto caratteristico del sarcoma.

Sulla benignità di alcuni neoplasmi nasali a carattere sarcomatoso fece egualmente al III° Congresso della Società Italiana di Laringologia un' interessantissima comunicazione Martuscelli, dimostrando che quasi sempre si tratta di tumori endoteliali. Egli osservò che essi si discostavano completamente da tutti gli altri neoplasmi così detti a triste indole, perchè in genere, anche quando si noti la recidiva, il novello tumore presenta sempre dimensioni minori del precedente, è impiantato nel medesimo punto ed ha del tutto identici al primo i caratteri istologici.

Martuscelli riassumendo le osservazioni di sarcomi del naso da lui raccolti in tre anni veniva alle seguenti conclusioni :

1°. che questi tumori non presentano quasi mai i caratteri di malignità, come i sarcomi di altre regioni ;

2°. che un tale fatto è dovuto in gran parte all' essere essi una lenta trasformazione dei tumori benigni del naso, maggiormente nei mixomi ;

3°. che questa trasformazione si effettua per una proliferazione endoteliale che parte dall' endotelio delle lacune linfatiche e dei capillari abbondanti quasi sempre nei tumori nasali ;

4°. che infine nei tumori di natura sarcomatosa l'operazione non deve essere mai differita e la prognosi deve essere sempre subordinata all' esito dell' esame microscopico.

La metaplasia dei neoplasmi da benigni in maligni, recisamente negata nel 1856 da Billroth, è ora una questione che non si mette più in dubbio. Massei fra i primi in Italia ed all' estero con accurate ricerche istologiche sostenne la metamorfosi delle neoplasie connettivali in sarcomatose. In realtà il primo a riferire un caso ben accertato di trasformazione dei comuni mixomi nasali in neoplasmi maligni fu Michel (1876), cui seguirono Hopmann, Bayer, Schmiegelow, Schaeffer, Gronbech.

Non solamente vi sono ancora molti punti oscuri riguardo alla trasformazione dei sarcomi benigni in maligni delle prime vie aeree, ma ci sono pure alcuni che sostengono aver osservata la spontanea scomparsa di neoplasmi di cattiva natura : ciò che sconvolgerebbe tutte le presenti teorie etiologiche di essi. Fra gli altri Robert Levy riferì l' interessante storia d'un paziente senza precedenti sifilitici, affetto da sarcoma del setto osteo-cartilagineo del naso con metastasi glandolari degli arti inferiori, specialmente del cavo popliteo, il quale abbandonato dopo ripetuti interventi come recidivo inoperabile fu riveduto un anno dopo

interamente guarito. Nel naso si riscontrava una semplice sinechia estendentesi dal setto alla volta nasale, e sugli arti inferiori la scomparsa completa delle tumefazioni glandolari. In seguito a tal caso sorprendente Robert Levy fece accurate ricerche nella letteratura, riscontrando che sono citati altri casi consimili, non però numerosi, e senza un conveniente esame istologico.

Che la prova clinica non possa essere considerata come positiva è ammesso da tutti, ma d' altro canto anche la diagnosi microscopica diede luogo a dibattute questioni. A proposito, p. es., dei casi di Senger, in cui i tumori avendo tutti i caratteri microscopici del cancro scomparvero, Freeman sostiene con Gussenbauer che essi probabilmente non erano dei veri sarcomi, il decorso clinico dovendosi forse valutare più degno d' attenzione che l' esame microscopico.

Una spiegazione scientifica di questa scomparsa di talune forme maligne, in specie sarcomatose, delle prime vie aeree fu quella data da Massei, Martuscelli ed altri allievi della Clinica di Napoli a proposito delle complicazioni nasali della sarcomatosi cutanea. Si tratta d' individui affetti da sarcoma cutaneo pigmentato emorragico, il quale, oltre che presentarsi in forma di noduli più o meno voluminosi su qualsiasi parte della superficie del corpo, può eziandio riscontrarsi sulla mucosa del setto, dei turbinati, dell' istmo delle fauci, dell' epiglottide e perfino del cavo laringeo.

Ora si sa che questa sarcomatosi cutanea subisce varie trasformazioni fino anche a scomparire spontaneamente. Si vedranno ad esempio alcuni noduletti di grandezza variabile, da quella di una noce a quella di un grano di miglio, di colorito rossastro, interessanti il derma, ora sporgenti, ora non estuberanti dalla superficie cutanea ; in pari tempo vedremo anche gli stessi noduli della grandezza, forma e colorito d' una lenticchia, ed in alcune parti del corpo, là dove una volta erano stati presenti questi noduli, delle macchie atrofiche di colorito bianco sporco con epidermide sottile, lucente, d' aspetto cicatriziale.

L'esame istologico di questi noduli fece rilevare la struttura identica dei sarcomi fascicolari, vale a dire cumuli di elementi fuso-cellulari disposti a forma di noduli contenenti ognuno più capillari. Tutti questi noduli sono tenuti fra loro assieme da un connettivo ricco di lacune vasali, tanto che in alcuni punti prende un aspetto cavernoso.

Nella prognosi dei tumori maligni delle prime vie respiratorie ed in specie in quelli del naso e del cavo si terrà pur conto, di fronte ai molti casi esistenti nella letteratura di guarigione o spontanea o consecutiva ad interventi chirurgici, eziandio dei legami etiologici fra sifilide e cancro, argomento trattato in una relazione del Massei nel Congresso Internazionale di Budapest nel 1909.

Anzichè un antagonismo fra sifilide e cancro, come si credeva nella metà del secolo passato, prevalse più tardi l' opinione che la lues potesse predisporre alle neoplasie epiteliali. Furono numerose le osservazioni specialmente di carcinomi svoltisi sopra punti precedentemente colpiti

da leucoplasie e di sarcomi sviluppatisi in organi per l' innanzi colpiti da gomme, venendosi a stabilire che vecchi focolai di lesioni terziarie potevano essere fertili sorgenti per lo sviluppo di tumori maligni sia epiteliali che connettivali.

Massei nel 1907 in una importante nota clinica sosteneva appunto la scomparsa di sarcomi e carcinomi delle prime vie aeree succeduti a lesioni terziarie sifilitiche, con esordio proprio delle forme gommose, per terminare con quella classica dei tumori maligni. Che meraviglia dunque che nei presunti tumori maligni, guariti o dietro intervento o spontaneamente, non si sia avverato un errore di diagnosi e cioè di avere scambiato per neoplasia maligna una pura lesione luetica, prima ancora che avesse subito questa metaplasia ?

Non è da meravigliarsi di questi errori nei quali possono cadere anche i più provetti clinici, per la ragione che non sempre è facile ricostruire la storia della sifilide sulle basi dell' anamnesi e dell' esame somatico. Nemmeno l' esame istologico riesce sempre facilmente dimostrativo ; aggiungi poi che talora neppure è possibile mettere in evidenza la concomitanza del cancro colla sifilide mercè la ricerca del treponema pallido e la sierodiagnosi col metodo di Wassermann, se il paziente fu a lungo assoggettato a cure specifiche.

La prognosi dei tumori maligni, specialmente delle fosse nasali, pecca di esagerato ottimismo stando a quelli che la presentano favorevole dopo interventi radicali, per la ragione che con essi spesso si confondono i pseudoneoplasmi a decorso maligno e che secondo Lermoyez sono di natura tubercolare. A giudizio di Garel, Collet e Gouedikt questi pseudoneoplasmi, che possono essere presi per sarcomi, talora assumono grande sviluppo senza grande reazione dei tessuti vicini. Si comprende come la terapia chirurgica di queste produzioni debba essere di esito fortunato.

Epiteliomi

Gli epiteliomi primitivi delle cavità nasali sono relativamente molto rari, tanto che Donogany e Lénart in tutta la letteratura non riuscirono a raccoglierne che ottanta casi ben confermati. Basterà a dimostrarne la rarità la statistica di Bondes relativa al cancro del naso, che quaranta volte erasi sviluppato esternamente, e solo due volte nella cavità interna delle narici. Nella statistica di Gurlt comprendente diecimila casi, in quattro si trattava di cancro ed in quattordici di sarcoma delle cavità nasali. Herzfeld sopra un totale di ventottomila infermi della sua ambulanza policlinica una sola volta riscontrò il cancro primitivo delle cavità nasali, e Finder nella Clinica di Fraenkel su quarantamila pazienti ne segnalò cinque.

La stessa eccezionale rarità dell' epitelioma primario delle cavità nasali fa sì che le nostre conoscenze sull' etiologia del cancro nasale siano quanto mai oscure, massime riguardo alla dibattuta questione, se esso possa essere la metamorfosi di un tumore benigno. Alcuni per

sostenere questa trasformazione di benigno a maligno di un tumore nasale attribuiscono un grande valore dimostrativo alla frequente presenza, nello stroma di polipi mixomatosi, di zone cancerigne. Bayer, Petersen, Kronpecher, come pure Donogany e Lénart, sostengono l' opinione che polipi propriamente benigni subiscono la degenerazione cancerigna.

Se ciò fosse vero avrebbero ben ragione quei rinologi, i quali, in presenza di polipi nasali, anzichè ricorrere alla semplice asportazione, cui più o meno tardi succede il riprodursi della neoplasia, consigliano di procedere per regola generale ad un intervento che garantisca l' ablazione completa del punto di partenza del tumore per ovviare appunto alla possibile, sebbene rara, trasformazione cancerosa. Siccome dai rilievi statistici di Donogany e Lénart risulterebbe che il carcinoma nasale prende origine più frequente in via decrescente, dalla regione del cornetto medio, del cornetto inferiore e del setto nasale, località che più spesso presentano vegetazioni polipoidi, ogni qualvolta ci si presenti un caso di sinusite cronica, o rinite ipertrofica a forma vegetante polipoide, non indugeremo a proporre un intervento radicale.

Sono così pochi nella letteratura i cancri primari delle cavità nasali che mancano anche speciali dettagli sulla prevalenza, dal punto di vista istologico, dell' una sull' altra forma di epitelioma ; ma sembrerebbe che in egual misura si abbiano cancri ad epitelio piano e cancri ad epitelio cilindrico.

Purtroppo i primi sintomi del carcinoma nasale alle volte sfuggono, e fanno perdere un tempo prezioso.

La letteratura non ricorda guarigioni di carcinoma primario delle cavità nasali e pochi in cui la recidiva venne a distanza di tempo (Bayer due anni, Bronner 10 anni).

Quando ci sia il sospetto di cancro ed esso sia ben localizzato al setto nasale e ai cornetti, delimitato alle parti circostanti e circondato ovunque da tessuto sano, non indugeremo qualsiasi operazione radicale per avere le maggiori probabilità di successo. Che se il cancro sarà diffuso alle cavità accessorie, alla volta faringea e nell' orbita, o ci limiteremo ai soliti palliativi o a quelle operazioni endo-nasali che possono arrecare un temporaneo allievamento alle sofferenze degli infermi.

Come nelle narici, ugualmente è rarissimo il carcinoma della faringe, tantochè nel 1901 Arslan sopra una statistica conglobare di 121 neoplasmi di questa regione registrava soltanto quattro casi di carcinoma. Già in un mio lavoro precedente a quello di Arslan io avevo accennato a tale rarità, del resto confermata da molti altri specialisti e chirurgi generici (Klebs, Koenig, Orth, Lublinski, Delie d'Ypres, Corradi, Cozzolino, Massei, Gradenigo, Roncalli, Citelli, Calamida, Rugani).

Riguardo alle cavità accessorie del naso molti casi, che furono descritti come tumori cancerosi guariti con intervento più o meno radicale e sopratutto colla completa resezione del mascellare superiore, non erano certamente epiteliomi, trattandosi invece di endoteliomi (Manasse).

È certo che non bisogna essere troppo corrivi nel far diagnosi di tumore maligno delle cavità nasali e delle cavità accessorie. Tissier in una sua memoria sui tumori delle vie nasali riporta la statistica di Gurlt nella quale sono segnalati solo 4 casi sopra un totale di 9554 casi di cancro di tutti gli altri organi. Newmann esprime l' opinione che due terzi dei così detti carcinomi del naso sono epiteliomi, e l' altro terzo semplici adenomi, i quali però da un momento all' altro possono trasformarsi in adeno-carcinomi.

Occorrerà però sempre stare in guardia quando l' esame istologico ci dimostri la presenza di un adenoma della mucosa naso-faringea poichè, come notò Thorner, certe forme di adenomi sono maligne fino dal principio e per lungo periodo talvolta non sono distinguibili nei dettagli anatomici delle varietà benigne. Sulla facilità con cui i papillomi e gli adenomi delle narici tendono a subire la metamorfosi maligna sarcomatosa o epiteliomatosa, lungamente discussero nell' Associazione Laringologica Americana nel 1897–99 parecchi rinologi (Wright, Hopkins, Leland e Newcomb).

Sarcomi

Forse clinicamente ha una certa importanza dal punto di vista della prognosi e del più o meno favorevole intervento chirurgico l'ubicazione del tumore maligno. Infatti vogliono alcuni che i sarcomi del setto nasale abbiano una gravità minore di quelli impiantati sui turbinati, nelle cavità accessorie nasali e sulla volta naso-faringea. Bosworth, Boylan, Payson-Clark, che raccolsero la più numerosa statistica di sarcomi del setto nasale, hanno concluso che la loro minore gravità dipende unicamente dalla situazione che permette di asportarli in toto e non perchè in essi esista una maggiore proporzione di elementi normali sui patologici nella trama del neoplasma.

Sarebbe anzi dimostrato che i fibro- e mixosarcomi del setto nasale, dopo un certo tempo apparentemente quiescenti, presero ad un tratto un rapidissimo sviluppo, per cui noi d' accordo con Payson-Clark, anzichè adottare mezze misure, consigliamo sempre in ogni caso sospetto di sarcoma di asportare immediatamente l' intiero setto osteo-cartilagineo nasale senza fare alcun trattamento conservativo.

Più difficile è alle volte la diagnosi dei sarcomi primitivi delle cavità accessorie nasali. Non è la prima volta che una sinusite cronica (sopratutto dell' antro di Higmoro e delle cellule etmoidali) con produzione di vegetazioni polipoidi, granulazioni fungose e voluminosa ipertrofia del turbinato medio o inferiore, venne presa per un sarcoma, avendo clinicamente presentato i medesimi sintomi, cioè la rapidità dello sviluppo, la protrusione degli occhi, emorragia spontanea o provocata, le intense nevralgie, l'invasione del cavo orbitale ecc.

Di tali casi ebbe a discutere l'Associazione Laringologica Americana, dando luogo ad un importante dibattito cui presero parte Price-Brown, Schurly, Hubbard, H. L. Swain, Ballenger, Cobb, Freer, John Mackenzie,

Coakley, Gordon Young, Robert Myles, Arthur Watson. Da questa discussione importante emerse sopratutto che la diagnosi di sarcoma dell' antro mascellare invadente il cavo naso-faringeo non è sempre facile, tanto che si cita il caso d'un paziente cui fu rifiutato qualsiasi intervento, avendo avuto un' emorragia, che lo aveva dissanguato. Ebbene dopo qualche tempo era guarito con semplici cure locali, essendo il tumore intieramente scomparso, salvo una piccola tumefazione sessile dell' estremo posteriore del turbinato.

Inoltre da questa discussione è risultato che si riscontra una notevole differenza di malignità fra i sarcomi nasali, dimodochè alcune volte la riproduzione del neoplasma è rapidissima nonostante le più vaste demolizioni, e tal' altra, con interventi assai limitati, la guarigione fu definitiva o la riproduzione avvenne tardivamente fino a cinque o sei mesi di distanza. A differenza dei sarcomi che insorgono in altre parti del corpo, quelli del naso hanno in genere una minore malignità e quindi anche gli interventi chirurgici sono coronati da maggiori successi.

Gli odontomi, di cui abbiamo una numerosa serie di casi interessantissimi illustrati da Forget, Robin, Broca, Magitot, Panas, Eames, Heath, Loyd, Sutton, A. W. de Roaldes, ecc., dopo un periodo di latenza possono rapidamente svilupparsi e dar luogo a tali deformazioni del mascellare superiore da essere scambiati con sarcomi, massime quando la superficie esterna dell' osso sia colpita da necrosi, e si manifestino fungosità attraverso i bordi alveolari ed il meato medio nasale con invasione del cavo orbitale.

Gli odontomi sono più rari nel mascellare superiore che non nell' inferiore ; forse sarebbero ancora in maggior numero se non fossero stati descritti come semplici tumori fibrosi od esostosi dell' antro di Higmoro. Il punto di partenza per lo più degli odontomi sono i denti molari e premolari.

Quando in un bambino di tenera età si riscontri la mancanza di uno o più denti, e contemporaneamente lo sviluppo di un tumore che deformi la cosmesi della faccia, devesi sempre sospettare prima un odontoma. Un tumore invece che si sviluppi nell' osso mascellare dopo una completa evoluzione dentaria farà invece sospettare, anzichè un odontoma, lo sviluppo di un sarcoma.

Ad ogni modo non si deve mai dimenticare, fra le probabilità di errori diagnostici, e quindi eccessive demolizioni del mascellare superiore, l' improvviso vuotamento d' un odontoma cistico nel seno mascellare donde un' infezione dell' antro di Higmoro ed una consecutiva sinusite fungosa a decorso più o meno rapido, che abbia tutte le parvenze d' un sarcoma.

Finder ha potuto dimostrare con la statistica alla mano che il sarcoma nasale è molto più frequente del carcinoma ; infatti egli potè riunire nella letteratura più di 100 casi di sarcoma e solo 17 di carcinoma. Su 28,000 malati iscritti al Policlinico di Charlottenburg notò 10 casi di sarcoma e due soltanto di carcinoma.

Prognosi dei tumori maligni del setto

Secondo i laringologi che si occuparono dei tumori maligni del setto (Gouguenheim e Hèlary, Dansac, D'Aguanno, Schmiegelow, Plicque, ecc.) essi avrebbero una riproduzione variabile a seconda della struttura istologica, e cioè molto rapida nell' epitelioma e sarcoma embrionale, più lenta negli endoteliomi e fibro-sarcomi. Delaux riferì osservazioni di sarcomi del setto il cui principio rimontava a più di 10 anni. In genere la fine di questi tumori, se non s' interviene presto, è fatale e la morte sopraggiunge o per cachessia dovuta al cancro, o per meningite, o per riproduzione del neoplasma negli organi interni.

Fra i tumori del naso ve ne sono alcuni creduti erroneamente maligni, e quindi operati con esito felice. Sovratutto i chirurgi generici, non obbligati a conoscere la nostra specialità, sono quelli che sovente nelle loro statistiche portarono per guariti radicalmente sarcomi del setto che non erano altro che i così detti polipi sanguinanti dapprima studiati da Schadewaldt, Lange, e poi da molti altri, tra cui Hasslauer, che ne ha fatto uno studio speciale e ne ha riassunto in una tabella tutti i casi pubblicati.

Questi polipi sanguinanti sono facili a scambiarsi con i sarcomi, potendo alle volte giungere fino al volume di una grossa noce, tanto da riempire le fosse nasali e deformare le narici. Anche la struttura istologica di questi tumori e la facile recidiva li fece prendere per tumori maligni. Allorchè si riuscirà a scoprire la caratteristica sede d' impianto di questi polipi sanguinanti e cioè la parte inferiore della cartilagine del setto, non cadremo nell' errore di fare un' operazione demolitrice, come sarebbe nel caso di un sarcoma.

La frequente comparsa di questi polipi sanguinanti sul setto cartilagineo, dove eziandio i sarcomi sono molto comuni a svilupparsi, ci lascia molto freddi dinanzi alle statistiche di alcuni chirurgi generici che sostengono aver guarito definitivamente i loro pazienti affetti da tumori maligni delle narici con vaste operazioni demolitrici. Significativa è la statistica di Arslan che su 80 tumori veri del setto segna, accanto a 29 sarcomi, 19 polipi e 8 papillomi che formano insieme una rispettabile cifra.

Prognosi dei tumori maligni epifaringei

Fra i tumori maligni del cavo naso-faringeo che fecero spesso, di fronte alle difficoltà operative, capitolare i più arditi chirurgi abbiamo i linfo-adenomi che, se più frequentemente furono visti sorgere dalle tonsille palatine, originarono anche in qualche caso dalla tonsilla di Luschka. Quando il linfo-adenoma, o malattia cosiddetta di Hodgkin, principia dalla volta naso-faringea, esso di rado resta limitato, ma invade l'uno e l'altro pilastro e spesso simultaneamente le tonsille palatine. Si distingue dal sarcoma, perchè ha un andamento molto più lento ed è costantemente accompagnato da ingrossamento delle glandole.

Un caso tipico di linfo-adenoma della volta faringea ebbi a seguire in tutte le sue fasi, diversi anni or sono, e si trattava di un giovane collega, che ebbe il coraggio di farsi operare ben cinque volte, aprendo da ultimo nella bocca una larga breccia mediante la resezione temporanea del mascellare inferiore. Sino dalla prima comparsa del tumore faringeo erano già invase dalla stessa neoplasia le glandole inguinali, le quali furono pure asportate. Or bene, nonostante le più ardite risorse dell' arte operatoria, il linfo-adenoma tornò sempre a riprodursi.

Nessuno meglio di questo caso infelice avrebbe potuto dimostrare il modo subdolo con cui i tumori maligni della faringe sovente insorgono ; una semplice ulcerazione superficialissima della tonsilla destra fu considerata in principio per tubercolosi e sifilide, mentre si trattava di un linfo-adenoma diffuso della faringe e già con invasione del sistema glandolare linfatico.

Se la medicina operativa non ha corrisposto nella cura dei linfo-adenomi, ha completamente fallito nei sarcomi primitivi della volta faringea. Mentre riguardo ai sarcomi delle tonsille Hayes Agnew osservò in una seduta dell' American Surgical Association (1889) che solo un caso è ricordato in cui non avvenne riproduzione e che qualunque operazione è semplicemente palliativa, noi affermiamo che per gli stessi neoplasmi, quando si svolgono sulla parete superiore faringea, la malignità è ancor più grave e del tutto inutile ogni tentativo chirurgico.

Basti pensare che non sono punto rari i sarcomi primitivi della base craniense comparsi in modo così rapido da avere in individui giovanissimi simulato la formazione di semplici ascessi che indussero il chirurgo a praticare una incisione del tumore !

Inoltre Sir James Paget ha avvertito che molti casi descritti come carcinomi midollari erano in realtà sarcomi. G. Hunter Mackenzie accenna pure alla loro rassomiglianza nei primi stadii con la semplice ipertrofia della tonsilla di Luschka, come ne raccogliemmo un esempio nella nostra Clinica e del quale conserviamo i preparati microscopici.

Hallopeau insiste sulla difficoltà diagnostica nelle prime fasi evolutive del sarcoma ; non v' è che l' esame istologico il quale possa dare qualche esatta notizia sulla natura del male. Bosworth ha trovato descritti nella letteratura diciannove casi in cui il sarcoma ebbe origine dal tessuto della volta faringea, e dalla sua statistica si rileva che può insorgere indifferentemente a tutte le età, in prevalenza nei maschi, con una durata della vita non superiore ad un anno, con minore tendenza a restare localizzato di quelli che nascono nelle cavità nasali.

Il sarcoma della volta naso-faringea costituisce, secondo Bosworth, una malattia maligna quanto il carcinoma. Mentre siamo pienamente della opinione di Bosworth, che nei sarcomi primitivi delle tonsille i migliori risultati si ottengono da una operazione radicale, cioè da una estirpazione completa di tutti i tessuti malati, invece crediamo che nei sarcomi primitivi della volta siano inutili tanto le più complete

demolizioni quanto i metodi operativi parziali, come l' allacciamento, il raschiamento, le cauterizzazioni galvaniche. Sulla volta faringea i sarcomi non hanno mai confini ben delimitati, i tessuti sono in estensione e profondità infiltrati, e, se un tentativo vogliamo ancor fare, non v' è che l' elettrolisi, la quale finora abbia corrisposto con una prognosi funesta meno precipitata.

Bosworth ha osservato che tutti i carcinomi della volta faringea, estirpati completamente assieme alle glandole cervicali sospette mediante la faringotomia e la sezione del mascellare inferiore, hanno recidivato dopo tre o cinque mesi. Secondo Trelat le operazioni cruente sono inutili nei casi di epitelioma faringeo ; su trenta pazienti da lui operati, quasi tutti soccombettero rapidamente. La maggioranza muore d' infiammazione flemmonosa del mediastino, di polmonite, di setticemia. Polallion dice che la mortalità è tra il 30,5 per cento e il 42 per cento.

Plicque sostiene del pari che nelle faringotomie, nonostante si faccia una rigorosa antisepsi della bocca, e si eseguisca la tracheotomia per applicare poi la canula di Trendelenburg, la prognosi è gravissima. In ventitrè operazioni da lui raccolte, la morte fu nove volte quasi immediata o per emorragia o per bronco-polmonite.

Quando la volta faringea è estesamente infiltrata dalla neoplasia, parlare d'operazione radicale sarebbe un' aberrazione ; però resterà ancora la possibilità di mitigare molto le sofferenze dovute al volume del tumore che ostruisce il cavo naso-faringeo e provoca disfagia e soffocazione ; sarà ancora il caso di frenare l' emorragia, di diminuire la secrezione icorosa. Non crediamo che tra i metodi palliativi sia finora stato suggerito nulla di meglio della elettrolisi.

I tumori maligni del naso-faringe hanno una prognosi funestissima quanto più è tenera l' età dei pazienti. Sebbene rarissimi nella prima infanzia pure ne sono stati registrati due casi. Brocet ebbe un sarcoma in un bambino di tre anni e mezzo, Scheimann in un bambino di tre anni, e la nostra Clinica ne ricorda uno. Quando anche si tratti della trasformazione di tumori adenoidei (Roncalli, Castex, Monboyran) è meglio non intervenire.

Prognosi dei tumori maligni nei bambini

Nei tumori maligni dei giovani l' esito immediato di qualsiasi atto operativo può essere più o meno buono a seconda del metodo prescelto. La via nasale è incerta e pericolosa, e parimenti la via buccale non è consigliabile, compromettendo essa la dentizione, la deglutizione, la fonazione. Nonostante i gravi danni della cosmesi è preferibile la via esterna, con speciale riguardo a rispettare il pavimento del naso. I tumori maligni del naso nei bambini furono alle volte confusi con l' encefalocele. La letteratura rinologica a tal proposito è quasi muta e ricordiamo soltanto i lavori di Fraenckel e Schvetz.

Nella letteratura chirurgica si conosce invece qualche caso di encefalocele in cui si ebbe un esito fortunato dall' intervento operativo che consistette per lo più nella resezione osteoplastica del mascellare. Nei bambini adunque, prima di abbandonare alle sole risorse della natura il paziente in cui si sospetti un tumore maligno, si faccia attenzione alla possibilità di non confondere con esso un encefalocele, di cui i segni distintivi principali saranno l' esistenza di un canale osseo e la possibilità di una riproduzione parziale o totale del tumore.

Prognosi dei tumori maligni dell' antro d' Higmoro

Nei tumori dell' antro di Higmoro è sempre giustificabile in massima un intervento operativo, ma occorre osservare alcune regole speciali. Al loro inizio la diagnosi è in genere incerta, ma, siccome nel dubbio è regola generale d' operare sollecitamente, sorge la necessità di operazioni economiche a scopo esplorativo. Quando si è sicuri dell' esattezza della diagnosi (rapido sviluppo del neoplasma, caduta dei denti, adenopatie, epistassi), allora bisogna seguire le regole generali del cancro, operare molto largamente e sollecitamente ; fare cioè una resezione tipica, estesa quanto è necessario, e non omettere di levare i gangli. La recidiva è frequente, ma ogni chirurgo ha visto dei malati stare bene per parecchi anni e ciò è sufficiente per invitarci a tentare un intervento precoce.

Sembrerebbe che fra i tumori maligni delle cavità accessorie nasali quelli dell' antro d' Higmoro dovessero rimanere lungo tempo isolati e quindi facili ad una definitiva guarigione con l' asportazione completa del mascellare superiore. Heymann su 250 soggetti presi a caso nell' anfiteatro anatomico e morti per cause diverse trovò 31 tumori di varia natura nei seni mascellari, senza che in vita avessero dato luogo ad alcun disturbo.

Di questi tumori per lungo tempo latenti e di natura benigna è abbastanza comune la trasformazione in epiteliomi e fibro-sarcomi a cellule polimorfe. Non sarà perciò mai vano ogni tentativo per giungere in casi sospetti ad una diagnosi sollecita che ci permetta di procedere ad un radicale intervento operativo che, dato il caso che il neoplasma non abbia ancora varcato i limiti del cavo mascellare, ritarderà una riproduzione troppo sollecita.

I tumori maligni primitivi del seno mascellare vanno assolutamente distinti da quelli svoltisi originariamente nelle narici e da queste diffusisi alle cavità accessorie, tanto dal punto di vista operativo quanto dal lato prognostico.

Col progredire delle cognizioni rinologiche il ristretto concetto della patologia chirurgica, che tiene soltanto conto delle alterazioni del mascellare in quanto si riferiscono all' apparecchio masticatorio, fu sorpassato dal riconoscimento che il corpo intero di quest' osso non solo costituisce la parte più essenziale dello scheletro nasale della faccia, ma racchiude altresì una delle più importanti cavità accessorie nasali. Spetta alla

rinologia di avere, col perfezionamento dei suoi metodi di esplorazione clinica, notevolmente migliorata la prognosi sinora così disastrosa dei tumori maligni, potendosi al presente in tempo far la diagnosi differenziale di quelli nati dalla mucosa di rivestimento del seno, dagli altri primitivi della piramide ossea che costituisce il mascellare superiore.

Ci sono argomenti tali da credere che il maggior numero dei casi di tumori maligni della chirurgia classica antica, sottoposti alla resezione totale del mascellare superiore, coi mezzi attuali sarebbero stati assai prima riconosciuti, almeno per due terzi, come neoplasmi insidiosamente svoltisi nell' ombra dell' antro d' Higmoro.

Non è chi non veda l' importanza che clinicamente ha una sollecita scoperta di un tumore maligno primitivo del seno mascellare, sia per l' indicazione dell' intervento più o meno energico da scegliersi, sia per l' esito medesimo. È certo che i dati prognostici più confortanti riferiti dalla letteratura dei tumori maligni dell' ultimo ventennio dipendono dalla tecnica diagnostica perfezionata che permette di scoprire i tumori all' inizio della loro formazione. Senza dubbio i tumori maligni dell' antro d' Higmoro, quando sono in tempo diagnosticati, offrono una prognosi relativamente meno grave di quelli primitivi della sostanza ossea del mascellare superiore, perchè possono essere soggiogati, anzichè col sacrifizio della totalità di questo osso, colla resezione parziale dell' antro.

Comprenderemo bene l' importanza di rispettare il pavimento dell' orbita e la volta palatina, riducendosi di molto lo schock operatorio, i pericoli di sepsi, la deformità della faccia, i disturbi della deglutizione e dell' emissione della parola (Faure, Bichanton, Jacques, Gaudier). Allorchè però il tumore intrasinusale abbia già invaso le pareti ossee dell' antro, l' estirpazione totale classica del mascellare s' impone, tanto più che ormai è raro, con la tecnica moderna, che i pazienti muoiano immediatamente in seguito a questo grave atto operativo, come molte volte succedeva in passato. È certo che con la totale asportazione del mascellare si è più lungamente tranquilli, essendo in genere la recidiva più tardiva che nelle resezioni parziali dell' antro, come del resto lo hanno di recente dimostrato in una statistica personale Jacques e Gaudier.

Dicono i partigiani della eseresi totale del mascellare superiore che la deformità ed i disturbi funzionali consecutivi a questa vasta demolizione della faccia siano in buona parte riparabili con una protesi, di cui hanno opinione favorevole Martin, Schlasler, Habl, Fritsche, Schröder. Non occorre molta intuizione per sostenere la tesi contraria e cioè che qualunque apparecchio di protesi, per quanto ingegnoso e leggero, sarà sempre irritante e mal sopportato, contrario alla disinfezione del cavo e quindi un' esca permanente alla recidiva dei tumori maligni. Non sarà che dopo una lunga sopravvivenza decorsa dall' atto operativo che si consegnerà il paziente ad un abile meccanico che provveda alla protesi, la quale, secondo Lebilean e Delair, non dovrà essere eseguita col caoutchouc ma con la porcellana.

Nei cancri primitivi del seno mascellare, anche dopo gl' interventi più radicali, malgrado tutte le precauzioni, la recidiva è quasi costante. Secondo Richon i tumori maligni del seno mascellare hanno una tendenza particolare ad invadere le cavità vicine, in specie l' etmoide, donde la necessità di esplorare anche in primo tempo il labirinto etmoidale.

Prognosi dei tumori maligni del seno sfenoidale

I tumori maligni del seno sfenoidale, sebbene la cerchia delle cognizioni sul'e malattie delle cavità accessorie nasali abbia fatto passi giganteschi per merito dei rinologi, rappresentano ancora un punto oscuro. La letteratura ne segnala un numero assai scarso e forse inferiore alla realtà, quando si pensi che le osservazioni di flogosi acute e croniche di tale seno si vanno moltiplicando. Si può anzi affermare che il più delle volte i tumori maligni sfenoidali vengono in principio scambiati con semplici sinusiti, vuoi primitive dello sfenoide, vuoi secondarie a lesioni iniziatesi nelle cellule etmoidali posteriori.

Naturalmente, i tumori dei seni sfenoidali formando fino adesso un capitolo quasi ignorato della rinologia, ben poco a noi è concesso dire della prognosi. A priori però dobbiamo affermarla fra le più disastrose della chirurgia delle prime vie aeree, sia per gl' interventi operativi che richiedono, sia per la natura stessa del processo morboso che, latente per lungo tempo e quindi troppo tardi diagnosticato, lascia poco adito alle speranze di una completa asportazione, anche nel semplice intento di ritardare le inevitabili complicazioni intracraniche.

Sono perfino registrati dei casi nei quali la diagnosi fu fatta solo al tavolo anatomico (Cimoroni ecc.). Bosworth fin dal 1896 in una sua interessante memoria presentata al Congresso annuale dell' Associazione Laringologica Americana sosteneva che tanto le affezioni sfenoidali di natura flogistica, quanto quelle di natura neoplastica, erano rarissime e per lo più consecutive a lesioni consimili delle cellule etmoidali, confondendosi facilmente la loro sintomatologia. Da allora non si può disconoscere che i mezzi diagnostici, fra cui la radiografia, sono al caso di renderci prezioso ausilio nello stabilire le affezioni primitivamente originatesi nel seno sfenoidale con esclusione di altre cavità come il labirinto etmoidale. L'argomento si è complicato di fronte ad altri tumori che non hanno punto di partenza dal seno sfenoidale, ma vi si possono successivamente affacciare, avendo avuto origine nell' ipofisi.

Prognosi dei tumori maligni etmoidali e frontali

La prognosi è sempre sfavorevole riguardo agli esiti lontani, perchè in genere queste cavità non vengono colpite primitivamente ed anche gl' interventi più radicali si trovano di fronte a vaste disseminazioni.

Nei tumori maligni etmoido-frontali ed etmoido-sfenoidali bisogna tener conto della possibile contemporanea presenza di ascessi cerebrali, avendo

presente non solo la possibile invasione endocranica del tumore ma il passaggio dei piogeni.

Alle affezioni del seno frontale si associano più spesso gli ascessi latenti del lobo frontale, a quelli etmoidali la meningite, ed a quelli sfenoidali la meningite e la trombosi del seno cavernoso.

Un esempio tipico di diffusione di tumori maligni a tutti i seni ed all' endocranio l' abbiamo avuto in un paziente di cui riportiamo la figura dimostrativa. Questo infermo, che pure non accusava ancora alcun sintomo cerebrale, morì improvvisamente mentre si recava ad una sezion ospitaliera chirurgica nella speranza di poter subire un altro intervento.



Statistica personale

Pongo fine a questo lavoro, nel quale ho cercato di riunire in forma sintetica tutte le attuali cognizioni sull' argomento, con una statistica dei tumori maligni del naso e dell' epifaringe osservati in Roma nell' ultimo decennio al Policlinico Umberto I°. Ho potuto raccogliere in tal guisa un numero rilevante di casi che, se non ci autorizzano a deduzioni cliniche come le statistiche conglobali raccolte da altri autori, pure serviranno di ammaestramento, sopratutto perchè furono trattati con unico indirizzo clinico basato esclusivamente sulla chirurgia.

Purtroppo i risultati sono per la mia statistica disastrosi come per le altre, e devo confessare che di molti casi segnati come guarigioni non è ancora detta l' ultima parola in quanto siamo troppo vicini all' intervento.

Una constatazione di fatto è che i migliori successi si riferiscono ai sarcomi limitati alle fosse nasali ; è questa forse una vittoria della rinologia che permette diagnosi precoci ed interventi solleciti.

Riepilogando la statistica, dirò : che su 65 tumori maligni 44 furono sarcomi nelle molteplici varietà istologiche (67 per cento), 19 furono epiteliomi (29 per cento) ; vi furono poi un endotelioma ed un adenoma (1,30 per cento). Relativamente agli esiti si ebbero : guariti sarcomi 27 (57,35 per cento), epiteliomi 8 (42 per cento), adenomi 1. Morti, sarcomi 17 (38 per cento), epiteliomi 11 (57 per cento), endoteliomi 1.

Di fronte a così poco lusinghieri risultati sta oggi l' affaticarsi delle migliori energie a risolvere l' assillante problema del cancro e non deve sembrare vana la speranza che una buona volta si giunga a conoscerne la patogenesi per dedurne la terapia.

Statistica dell' Istituto Chirurgico di Roma

1. B. R. 17 (u.)	Mix. sarc. dell' antro d' Higmoro	Resez. mascel. sup. alla Durante	Guar.
2. F. G. 8 (u.)	Sarc. mascel. sup.	Rifiuta inter.	Morte
3. P. M. 50 (d.)	Sarc. mascel. sup. sin.	Resez. mascel. sup. alla Durante	Guar.
4. A. T. 50 (u.)	Mix. sarc. mascel. sup. d.	Resez. mascel. sup. alla Durante	,,
5. C. A. 13 (d.)	Sarc. mascel. sup. sin.	Resez. mascel. sup. alla Durante	,,
6. C. G. 23 (u.)	Sarc. mascel. sup. d.	Resez. mascel. sup. alla Durante	,,
7. G. L. 40 (u.)	Sarc. mascel. sup. d.	Resez. mascel. sup. alla Durante	,,
8. M. P. 39 (d.)	Sarc. dei due mascel. sup.	Resez. totale dei mascel.	,,
9. G. L. 30 (u.)	Sarc. mascel. sup. d.	Resez. mascel. sup. alla Durante	Morte
10. P. C. 72 (u.)	Sarc. mascel. sup. d.	Resez. mascel. sup. alla Durante	,, ,,
11. R. A. 65 (u.)	Epit. antro d' Higmoro d.	Resez. parz. mascel. sup.	Guar.
12. G. G. 20 (u.)	Sarc. etmoide e seno front. s.	Vuotamento	,,
13. M. O. 55 (u.)	Endot. seno front. s. diffuso all' orbita, all' etmoide e al seno mascellare	Exorbitatio e vuotamento dei seni	Morte
14. C. T. 41 (u.)	Sarc. corn. inf. s. e pal. duro	Res. del naso alla Chassaignac. Vuotamento	Guar.

Statistica del I° Padiglione chirurgico al Policlinico

1. B.M.D. 48 (u.)	Epit. della porz. post. del mascel. sup. con diff. alla fossa pterigo-mascel.	Resez. del mascel. sup. e dell' osso malare. Resez. metà sup. branca montante mascel. inf. Dissez. del tum. fino alla base cranica	Morte
2. P. D. 44 (u.)	Epit. dell' epif. con diff. alla coana d. e porz. d. e med. del pal. molle	Resez. del mascel. sup., asport. del tumore	Guar.
3. C. S. 38 (d.)	Epit. epifaring. e retromascel.	Resez. temp. del mascel., asport. del tumore	Guar.
4. G. M. 47 (d.)	Epit. della base del cranio	Resez. temp. del mascel. sup., asport. del tumore	Guar.
5. D.F.M. 48 (d.)	Tumore mal. del mascel. sup. d.	Resez. mascel. sup.	Guar.

Statistica del III° Padiglione chirurgico al Policlinico

1. S. A. 60 (u.)	Sarc. della base del cranio diff. alla fossa nasale sin.	Resez. osteoplastica alla Ollier, asport. del tumore	Morte
2. F. A. 25 (u.)	Sarc. della base del cranio	Non operabile	,,
3. C. L. 57 (d.)	Sarc. della metà d. dell' epif.	Resez. mascel. sup. d., asport. del tumore	,,
4. M. G. 14 (u.)	Sarc. della base del cranio diff. ai seni ed alla faringe	Insufflazione intratracheale alla Meltzer. Resez. mascel. sup., asport. del tum., vuotamento dei seni	,
5. C. E. 12 (d.)	Mix. sar. fossa nas. sin. con usura della parete int. dell' orbita. Recid. 4 volte	Sempre eseguita la rinotomia temporanea	Guar.
6. E. I. 15 (u.)	Fibr. sar. pedunc. fossa nas. d.	Rinotomia temporanea, asport. del tum.	,,
7. C. S. 40 (d.)	Sarc. dell' etmoide d. diff. alla fossa nas., faring., cavo orbit. e seno mascellare	Legatura provv. della carot. esterna, rinotomia temp., aspor. del tumore, vuotamento dei seni	,,
8. C. M. 28 (d.)	Sarc. dell' etmoide, recid. 2 volte	Vuotamento dell' etmoide	Morte

STATISTICA DELLA CLINICA OTO-RINO-LARINGOIATRICA DI ROMA

1. R. G. 16 (u.)	Sarc. della volta faring.	Spaccamento del palato	Morte
2. B. P. 18 (u.)	Fibr.-sarc. dell' epifaring.	Resez. del mascel. sup.	,,
3. A. T. 60 (u.)	Epit. naso-faring., metastasi gland. multiple	Inoperabile	,,
4. S. R. 50 (u.)	Epit. naso-faring., metastasi gland. multiple	,,	,,
5. F. S. 50 (u.)	Sarc. del rino-faring.	,,	,,
6. A. T. 2 (u.)	Sarc. rino-faring.	Rinotomia temp.	Guar.
7. R. B. 35 (u.)	Sarc. fossa nasale d.	,, ,,	,,
8. V. E. 30 (d.)	Sarc. fossa nasale s.	,, ,,	,,
9. T. G. 40 (u.)	Sarc. fossa nasale d.	,, ,,	,,
10. D. S. 30 (u.)	Fibro-sarc. fossa nasale s.	,, ,,	,,
11. M. C. 40 (d.)	Mixo-sarc. fossa nasale d.	,, ,,	,,
12. E. P. 30 (u).	Fibro-sarc. fossa nasale s.	,, ,,	,,
13. D. D. 40 (u.)	Fibro-sarc. fossa nasale s.	,, ,,	,,
14. M. S. 30 (d.)	Sarc. fossa nasale d.	Rifiuta intervento	Morte
15. A. D. 55 (u.)	Sarc. fossa nasale destra diff. all' etmoide, sfenoide, endocranio	Inoperabile	,,
16. C. B. 40 (u.)	Sarc. melan. fossa nasale d. limitato alla parte ant., recidivato 4 volte	Rinotomia temporanea e folgorazione, res. del mascel. sup.	Morte
17. D. P. 45 (u.)	Sarc. fossa nasale s.	Rinotomia temporanea	Guar.
18. V. T. 50 (u.)	Epit. fossa nasale s.	,, ,,	,,
19. M. C. 41 (d.)	Epit. fossa nasale d., metastasi gland. multiple	Inoperabile	Morte
20. M. C. 45 (d.)	Epit. diff. del naso, met. gland. multiple	,,	,,
21. D. R. 70 (u.)	Epit. diff. del naso, met. gland. multiple	,,	,,
22. S. G. R. 56 (u.)	Epit. fossa nasale d. consecutivo a mixoma	Rinotomia temporanea	Guar.
23. E. A. 66 (d.)	Epit. diff. del naso, metastasi glandolari multiple	Inoperabile	Morte
24. A. R. 45 (u.)	Epit. diff. del naso, metastasi glandolari multiple	,,	,,
25. D. M. 50 (d.)	Epit. diff. del naso, metastasi glandolari multiple	,,	,,
26. M. D. 60 (u.)	Epit. diff. del naso, metastasi glandolari multiple	,,	,,
27. T. C. 46 (u.)	Sarc. del pavimento del naso	,,	,,
28. G. C. 30 (u.)	Sarc. del pavimento del naso	Rifiuta intervento	,,
29. A. P. 50 (u.)	Epit. del palato duro, adenite cervicale	Trattamento col radium	,,
30. G. A. 30 (d.)	Sarc. turbin. inf. d.	Rinotomia temporanea	Guar.
31. G. T. 20 (u.)	Sarc. turb. inf. d.	,, ,,	,,
32. T. C. 35 (u.)	Sarc. del setto nasale	,, ,,	,,
33. L. F. 40 (u.)	Sarc. del setto	Resezione del setto	,,
34. D. E. 27 (u.)	Sarc. mascel. sup. sin.	Resez. del mascel.	,,
35. L. R. 19 (d.)	Osteo-sarc. mascel. sup. s.	Resez. parziale	,,
36. C. D. R. 50 (u.)	Epit. seno-mascel. s.	,, ,,	,,
37. E. T. 52 (d.)	Adenoma seno mascel. d.	,, ,,	,,
38. I. C. 20 (u.)	Sarc. dell' etmoide	Vuotamento dell' etmoide	,,

SECTION XVI

Otology

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OTOLOGY

DISCUSSION No. 1

THE SURGICAL ANATOMY OF THE MASTOID

Rapport par M. le Dr. JULES MOURET, Professeur adjoint à la Faculté de Médecine de Montpellier

QUE DOIT-ON ENTENDRE PAR RÉGION MASTOÏDIENNE CHIRURGICALE ?

Bloc Mastoïdien

On appelle mastoïde la saillie que forme extérieurement l'os temporal en arrière du conduit auditif externe. Cette saillie ou apophyse proémine en dehors et en bas, en forme de cône renversé. Au point de vue embryologique, l'os pétreux et l'os écailleux concourent à sa formation. L'os écailleux forme la couche superficielle de sa partie supéro-antérieure, la plus voisine du conduit auditif externe ; l'os pétreux forme tout le restant de la mastoïde. La démarcation entre ces deux parties est indiquée par une ligne de suture, dite pétro-squameuse externe, qui part de l'angle squamo-mastoïdo-pariétal et descend très obliquement, en bas et en avant, vers le sommet de l'apophyse. Au point de vue chirurgical, il importe peu que la mastoïde soit constituée, à la fois, par l'os écailleux et par l'os pétreux, de même qu'il importe peu que le sommet de la mastoïde proémine plus ou moins sous la peau de la région rétro-auriculaire. Ce qu'il importe au chirurgien de bien connaître ce n'est pas la mastoïde, saillie anatomique, mais la mastoïde, région chirurgicale, c'est-à-dire la *région mastoïdienne*, que les suppurations de l'oreille envahissent si souvent et que le chirurgien fouille habituellement au cours de ses interventions. C'est la délimitation de la *région mastoïdienne chirurgicale*, telle que je la comprends, que je décrirai tout d'abord. Chacun des auteurs, qui se sont occupés de la mastoïde chirurgicale, en a donné une description un peu différente de celle des autres. On ne saurait donc m'en vouloir de faire comme eux. Mais ce n'est pas pour faire œuvre personnelle que je trace à la *région mastoïdienne* des limites un peu différentes de celles qu'on lui donne habituellement : c'est uniquement parce que l'étude des suppurations mastoïdiennes me paraît d'accord avec la conception que je me fais des limites qu'il convient de donner à cette région.

FIGURE I

Temporal droit vu par la face externe. C'est la pièce, qui a servi à faire les préparations, qui sont représentées dans les figures II, III, IV.—Sur cette figure, j'a schématisé les zones de la face externe de la mastoïde, que je décris dans le texte. J'ai représenté en projection les différentes positions du sinus latéral, ainsi que la position, que l'antre a dans cette pièce, et celle qu'il devrait avoir normalement.

1, ligne de section marquant la limite antérieure du bloc mastoïdien. Comme je l'ai dit dans le texte, cette ligne, qui passe à travers la spina-supra-méatum devrait, en réalité, passer en avant d'elle et passer à 2 mm. plus en avant.—2, ligne marquant, sur la face externe du temporal, le niveau auquel correspond, dans cette pièce, le plancher de la cavité crânienne.—3, linéa temporalis. Sur cette pièce elle est très basse et située à 8 mm. au-dessous de la ligne 2, qui marque la situation du plancher de la cavité crânienne.—4, conduit auditif externe.—5, spina-supra-méatum.—6, projection en pointillé de l'antre, de l'aditus et de la cavité épitympanique, telles que seraient ces cavités, si elles occupaient une situation normale, c'est-à-dire si leur voûte ne dépassait pas la racine horizontale de l'apophyse zygomatique et si l'antre occupait la région, qu'il doit occuper normalement en arrière du $\frac{1}{3}$ supérieur de la paroi postérieure du conduit.—7, extrémité postérieure de la rainure digastrique.—8, partie postérieure de la bulle digastrique.—9, 9′, suture pétro-squameuse externe. Cette suture est marquée légèrement à la surface de l'os, mais elle s'étend en arrière, plus loin que normalement, de même qu'on peut la suivre, en bas, jusqu'au milieu du sommet de l'apex.—10, apophyse zygomatique.—11, projection de la cavité épitympanique telle qu'elle est dans cette pièce.—12, projection de la cavité antrale, telle qu'elle est reéllement dans cette pièce. L'antre est petit, très élevé, en position dite fœtale, et son plancher est presque sur le même niveau que la paroi supérieure du conduit auditif externe.—13, projection du sinus latéral sur la surface extérieure de la mastoïde.—14, ligne pariéto-apicale. Cette ligne marque, dans les dispositions normales, la limite antérieure de la portion descendante du sinus latéral et, aussi, la situation de la suture pétro-squameuse externe ; celle-ci se trouve, ici, exceptionnellement, en arrière de la ligne pariéto-apicale.—15, sinus latéral, tel qu'il serait en légère procidence.—16, sinus latéral, tel qu'il serait en procidence extrême.—17, angle squamo-mastoïdo-pariétal.—18, suture mastoïdo-occipitale postérieure.—19, rond d'attaque de l'angle supéro-postérieur du méat auditif dans la trépanation trans-spino-méatique.

La zone mastoïdo-pétreuse antérieure correspond au quadrilatère formé par les lignes *a*, *a′*, *d*, *b*.

La zone antrale correspond au quadrilatère *a*, *a′*, *c′*, *c*.

La zone sous-antrale correspond au quadrilatère *c*, *c′*, *d*, *b*.

La zone mastoïdo-pétreuse postérieure ou intersinuso-antrale correspond au triangle *e*, *d*, *a′*.

La zone sinusienne correspond à l'espace compris entre les deux lignes brisées marquées par le chiffre 13.

La partie terminale de la portion horizontale du sinus correspond à S_1.

La région du coude correspond à S_2.

La portion descendante correspond à S_3.

La zone cérébelleuse correspond à *C*.

La ligne *b*–*b′* marque la limite de la base de l'apex.

La zone apicale, *A*, est au-dessous de cette ligne.


17
14
15
16
3
12
1
11
2
e
a'
a
13
S1
S2
9
S3
C
18
4
10
5
19
6
8
A
7
9'

Les auteurs ne décrivent à la région mastoïdienne qu'une face externe et une face interne. Encore celle-ci n'est-elle décrite que dans la partie postérieure, dite écaille de la mastoïde, qui s'étale, vers l'occipital, à la surface du cervelet, en arrière de l'apophyse proprement dite. A la partie principale de la mastoïde, qui, dans la profondeur, fait corps avec la pyramide pétreuse, on ne donne pas de limites. Or, il me semble juste de mieux préciser la délimitation de la région mastoïdienne, particulièrement au niveau de cette partie, qui paraît, anatomiquement, indivise entre la mastoïde et le rocher. C'est là, en effet, que se trouve sa partie principale, *l'antre mastoïdien*. Le manque de précision dans la délimitation de la région mastoïdienne fait que l'antre est appelé par les uns *antre pétreux*, par d'autres *antre mastoïdien*, par d'autres encore *antre pétro-mastoïdien*. D'autres enfin considérant surtout la situation de cette cavité entre deux os embryologiquement distincts, l'os pétreux et l'os écailleux, l'appellent *antre pétro-squameux*. Tout cela, sans doute, n'est que convention et affaire de mots. La dénomination importe peu.

Au point de vue clinique et chirurgical, la mastoïde forme une région distincte. L'antre est non seulement le centre pneumatique, qui commande toutes les autres cavités de la région, mais aussi le centre des suppurations d'origine tympanique ; il est aussi le centre opératoire de toute intervention sur la mastoïde. Pour toutes ces raisons, me semble-t-il, il est assez rationnel de négliger son *origine pétreuse et squameuse* pour ne considérer que son *importance mastoïdienne*. Aussi je l'appellerai simplement antre *mastoïdien* et je placerai la limite profonde de la région mastoïdienne immédiatement en dedans de la paroi interne de l'antre.

On pourra, peut-être, m'objecter, que cette délimitation empiète beaucoup sur la *pyramide pétreuse*, mais, en définitive, puisque la mastoïde n'est qu'une *région conventionnelle* et que ce n'est pas la *saillie apophysaire* de la mastoïde, qui est intéressante pour nous, mais bien l'ensemble des cavités, qui sont creusées dans son intérieur, et dont l'antre est le centre de rayonnement, peu importe que la délimitation de la mastoïde s'étende plus ou moins loin dans la profondeur de l'os. D'ailleurs, cela est plus en rapport avec les faits cliniques et les actes chirurgicaux : c'est la chose essentielle.

Pour bien voir la *région mastoïdienne chirurgicale*, il faut l'isoler de l'os temporal. Pour cela, je la sépare d'abord de *l'écaille* par une section horizontale, qui rase la face supérieure de la pyramide pétreuse. Puis, je fais deux autres sections perpendiculaires l'une à l'autre. L'une de ces deux sections est vertico-transversale et rase la face postérieure du conduit auditif externe ; l'autre est verticale et antéro-postérieure, parallèle à l'axe antéro-postérieur du crâne. Celle-ci tombe sur la face postérieure de la pyramide, en passant par la partie la plus inférieure de la suture occipito-mastoïdienne, à l'intérieur du crâne. Les deux coupes ainsi faites sont perpendiculaires entre elles et se rencontrent au niveau du trou stylo-mastoïdien. La troisième portion (partie descendante) du canal de Fallope se trouve au voisinage du sommet de l'angle dièdre formé pas les deux plans de section.

Le tracé de ces deux coupes est donné dans les figures I et II. Dans la figure I la ligne 1 représente, à la surface extérieure de l'os, la section faite dans le plan vertico-transversal. Dans la figure II, la ligne 8 représente cette même section dans l'intérieur du crâne, à la face supérieure du rocher ; la ligne 8′ représente aussi dans l'intérieur du crâne la section faite dans le sens vertical et antéro-postérieur. Le résultat de cette double section est représenté dans les figures III et IV. La figure III montre le *bloc mastoïdien* détaché du restant du temporal : le bloc osseux est vu par l'angle qui sépare les deux plans de section. La figure IV montre le restant du temporal, vu aussi par les surfaces de section, après séparation du *bloc mastoïdien*.

Ainsi isolée, la région mastoïdienne est formée par un bloc osseux, auquel il convient de distinguer six faces : une face externe, une face interne, une face antérieure, une face postérieure, une face supérieure et une face inférieure.

§ I. **Face externe.** — La face externe du bloc mastoïdien forme une surface légèrement convexe, qui, en bas, se prolonge par une saillie de forme conique, laquelle constitue l'apophyse mastoïde proprement dite ou *apex*. Cette face représente, au point de vue opératoire, la partie la plus importante de la région mastoïdienne, aussi convient-il d'en connaître les détails tant au point de vue purement descriptif qu'au point de vue topographique. J'entends, par ce dernier mot, les rapports que la surface extérieure de la mastoïde présente avec les parties qui lui sont sous-jacentes, et que le chirurgien peut avoir à rechercher. Je ne m'occuperai en ce moment que du côté purement descriptif, réservant pour la fin de ce travail l'étude topographique, à laquelle je rapporterai les indications opératoires qu'elle pourra nous fournir. D'ailleurs l'étude topographique se comprendrait mal ici, tant que les autres parties du bloc mastoïdien n'auront pas été décrites.

Au point de vue descriptif la surface extérieure de la mastoïde nous présente à considérer : 1° ses limites, 2° sa conformation.

En avant, elle est limitée, de bas en haut, par le bord antérieur de l'apex mastoïdien, par le bord postérieur du méat auditif, et, au-dessus de celui-ci, par une ligne fictive, qui prolongerait ce bord jusqu'à la racine horizontale de l'apophyse zygomatique. Tout cela se trouve sur le bord externe de la coupe vertico-transversale (figs. III et IV), qui nous sert à isoler le bloc mastoïdien : sur cette coupe on voit que la partie de son bord externe, qui est sus-jacente à la lèvre postérieure du méat auditif, correspond, dans la profondeur, à une région dans laquelle se trouve la paroi externe de l'aditus et de la cavité épitympanique. En bas, elle est limitée par le sommet de l'apex. La limite postérieure (fig. I) correspond : 1° en bas, au bord postérieur de l'apex, 2° en haut, à la suture mastoïdo-occipitale, 3° entre ces deux parties, à une ligne fictive qui s'étend à peu près horizontalement de l'un à l'autre.

La limite supérieure est marquée, au milieu, par l'angle de suture

pariéto-squamo-mastoïdien (17) ; en arrière de celui-ci, par la suture pariéto-mastoïdienne ; en avant de lui, par une légère crête, très mousse, obliquement ascendante d'avant en arrière, qui représente le prolongement de la racine horizontale de l'apophyse zygomatique au delà du méat auditif externe : on l'appelle *linéa temporalis*. Elle donne insertion à l'aponévrose temporale et sépare la fosse temporale de la face externe de la région mastoïdienne. Les chirurgiens accordent à la linéa temporalis une importance très grande : on la considère comme correspondant au plancher de la cavité crânienne et, par suite, comme étant la limite extrême, que la trépanation mastoïdienne ne doit pas dépasser, au risque de pénétrer dans la fosse moyenne du crâne. La linéa temporalis est un bon point de repère pour le premier temps de la trépanation mastoïdienne, il faut même s'en tenir d'abord un peu éloigné, car il arrive, parfois, que le plancher de la cavité crânienne descend plus bas que le niveau de la linéa. Mais elle peut aussi être sur un point moins élevé que l'endocrâne (fig. I, 3).

Sur la face extérieure de la mastoïde se trouvent : (*a*) une dépression ; (*b*) une surface d'insertions musculaires ; (*c*) une saillie en forme de crête ou d'épine et une fossette rétro-spinale ; (*d*) des orifices vasculaires ; (*e*) une ligne suturale, dite suture pétro-squameuse externe.

(*a*) *Dépression mastoïdienne.* — Dans son ensemble la surface extérieure de la mastoïde est convexe de haut en bas et d'avant en arrière. Dans le tiers supérieur de la moitié antérieure, au-dessous de la linéa temporalis et en arrière de la moitié supérieure du méat auditif, la surface est déprimée sur une hauteur de 1 cm. environ. Cette dépression est plus ou moins prononcée, suivant que l'apex est plus ou moins volumineux et que la linéa temporalis est plus ou moins saillante à la surface de l'os. Elle correspond à ce que j'appellerai plus loin la zone antrale.

(*b*) *Insertions musculaires.* — Les insertions supérieures des muscles sterno-cléido-mastoïdien, splénius et petit complexus se font sur la face externe de la région mastoïdienne, suivant une ligne oblique de bas en haut et d'avant en arrière, qui va vers l'angle de suture mastoïdo-pariéto-occipitale. L'étendue de ces insertions musculaires recouvre toute la face externe de l'apex proprement dit et a une largeur de plus de 1 cm. dans la partie qui s'étend de l'apex à l'angle supéro-postérieur de la région mastoïdienne ; elle a un aspect rugueux, qui est dû à l'implantation des fibres tendineuses sur l'os (fig. XI, 10). Cette ligne divise la surface externe de la mastoïde en deux parties : l'une antérieure, l'autre postérieure. La partie antérieure est pour le chirurgien la partie la plus importante. C'est sur elle que porte presque toujours l'acte opératoire. Ce n'est que plus rarement, lorsque les lésions osseuses sont très étendues en arrière, qu'on est obligé d'attaquer la partie postérieure, cérébelleuse, après avoir ruginé les insertions des muscles mastoïdiens. Ces insertions musculaires protègent, à la fois, la zone cérébelleuse de la partie externe de l'écaille de la mastoïde et la partie postérieure (postéro-externe) de ce que j'appellerai *face inférieure* du bloc mastoïdien. Pour aborder celle-ci il faut d'abord désinsérer les muscles mastoïdiens, comme pour découvrir

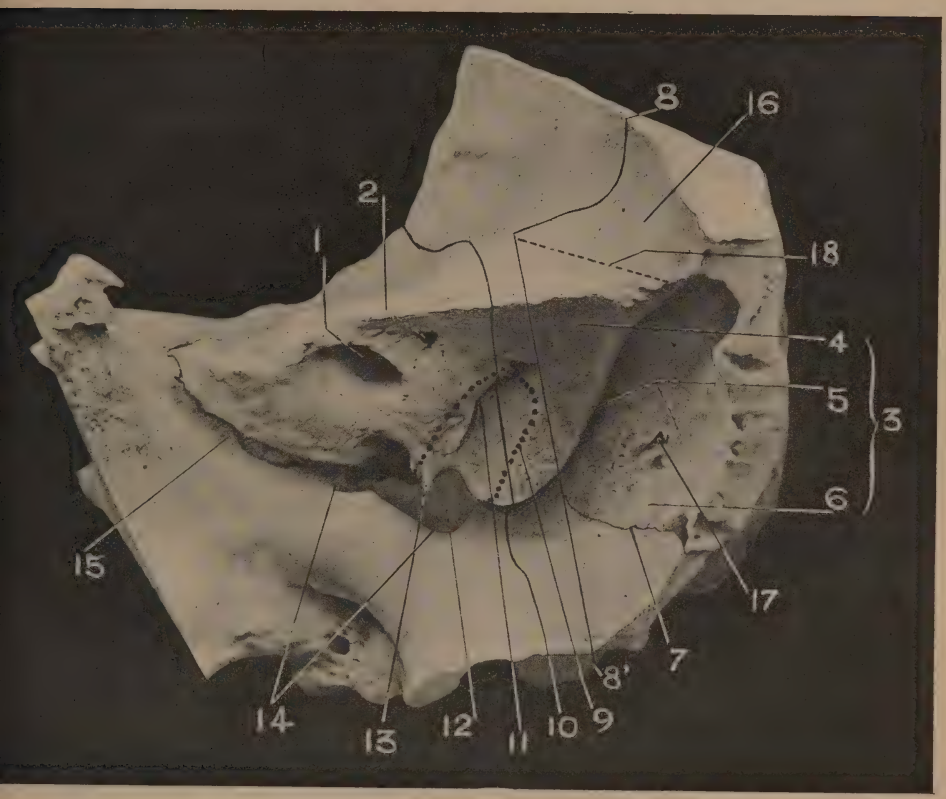


FIGURE II

Même temporal vu par sa face postéro-interne, endocrânienne.—1, trou auditif interne.—2, fossa subarcuata.—3, ensemble de la face postéro-interne du bloc mastoïdien, qui se décompose en trois zones secondaires, 4, 5 et 6.—4, zone rétro-pétreuse.—5, zone sinusienne.—6, zone cérébelleuse.—7, suture mastoïdo-occipitale.—8, ligne de section correspondant au bord supérieur, intra-crânien, du plan vertico-transversal suivant lequel le bloc mastoïdien est séparé du restant du temporal, en avant. Cette ligne correspond à 8 de la fig. III.—8′, ligne de section correspondant au plan de section vertical et antéro-postérieur, suivant lequel le bloc mastoïdien est séparé de la pyramide pétreuse en dedans. Cette ligne 8′ correspond au 18′ de la fig. III.—9, ligne pointillée indiquant l'étendue de la procidence du golfe de la jugulaire dans cette pièce.—10, ligne correspondant à un autre plan de section antéro-postérieur, qui a été fait aussi sur cette pièce.—11, fente unguéale.—12, partie du trou déchiré postérieur correspondant à la jugulaire.—13, épine jugulaire.—14, ensemble du trou déchiré postérieur.—15, sillon du sinus pétreux inférieur.—16, face supérieure du bloc mastoïdien appartenant à la face supérieure de la pyramide pétreuse.—17, orifice du canal de la veine mastoïdienne.—18, ligne représentant, à la face supérieure du bloc mastoïdien, l'orientation de l'axe antéro-postérieur de la cavité adito-antrale.

la zone cérébelleuse. En suivant alors le bord postérieur saillant de l'apex, on pénètre dans la face inférieure de la mastoïde ; mais pour la mettre bien à découvert il faut, en outre, réséquer l'apex mastoïdien qui la protège en dehors, en formant, sous les muscles, un mur solide et résistant.

(*c*) *Épine de Henle.* — *La spina-supra-méatum* de Henle est un point de repère des plus importants pour la trépanation mastoïdienne. Elle est située au voisinage de l'angle supéro-postérieur du méat auditif ; quelquefois au niveau même de cet angle, empiétant sur le bord supérieur du méat (ou tout entière sur le bord supérieur) ; plus souvent au-dessous de l'angle, le long de la partie supérieure du bord postérieur du méat. Sa forme est assez variable : tantôt elle a la forme d'une épine (fig. V, 5) tantôt celle d'une crête allongée (figs. I, 5 ; X, 6). Sa longueur varie de 2 à 10 mm. Quelquefois elle est à peine ébauchée (fig. IX, 10), parfois elle manque complètement (fig. XI). En arrière de l'épine, surtout lorsqu'elle est longue et très saillante, on trouve une surface de forme ovalaire ou triangulaire, déprimée en fossette : fossette rétro-spinale. Cette fossette existe parfois en l'absence de l'épine (fig. XI). Sur la coupe vertico-transversale, représentée dans la figure III, l'épine de Henle et la fossette rétro-spinale correspondent au plancher de l'aditus. Ce rapport n'est pas absolu. Il varie d'abord avec l'étendue et la position de l'épine ; il varie aussi suivant que le toit de l'antre, de l'aditus et de la cavité épitympanique est élevé ou abaissé. La distance, qui les sépare, varie de 5 à 10 mm. suivant que l'os est plus ou moins épais ; chez le jeune enfant cette distance est très minime : quelques millimètres à peine.

(*d*) *Orifices vasculaires.* — La face externe de la mastoïde présente deux zones riches en vaisseaux : la zone criblée rétro-méatique et la zone des grandes veines mastoïdiennes ; les vaisseaux de ces deux zones réunissent la circulation profonde (intra-osseuse et intra-crânienne) à la circulation du périoste et des parties molles en général. Ces deux zones vasculaires occupent, l'une, la partie de la face externe, qui est en avant de la ligne des insertions tendineuses, l'autre, la partie qui est en arrière d'elles.

La *zone criblée rétro-méatique* se trouve en arrière de l'épine de Henle, dans la fossette rétro-spinale (fig. X, 5) et autour d'elle, lorsque cette fossette existe. Elle occupe une surface d'un centimètre carré environ, qui est criblée de tout petits orifices, dont chacun donne passage à un vaisseau, qui réunit la circulation du périoste à la circulation intra-osseuse et tout particulièrement à la circulation périantrale. Chez certains sujets ces pertuis osseux sont peu apparents ; chez d'autres, au contraire ils sont très nettement dessinés et donnent à la région l'aspect d'un crible très fin. Ils sont surtout très visibles au cours des mastoïdites, par suite de l'inflammation du tissu conjonctif et des veines qui le traversent. Ils forment un des principaux lieux de passage de l'infection endo-mastoïdienne vers le périoste et favorisent la formation des abcès périostiques, sans que l'os lui-même soit fatalement malade.

Les *grandes veines mastoïdiennes* constituent une voie de dérivation pour la circulation intra-crânienne de la plus haute importance. Tout d'abord, dans les premiers temps de la vie fœtale, elles représentent la principale voie de sortie du sinus latéral hors du crâne. Puis, la circulation sinusienne prenant la voie du trou déchiré postérieur, la voie des veines mastoïdiennes devient d'une importance secondaire : ces veines s'atrophient en partie et ne représentent parfois que des canaux de petit calibre. Mais, chez certains sujets, les veines mastoïdiennes sont quelquefois très volumineuses et, lorsque la veine est unique, elle peut avoir un calibre de 6 à 8 millimètres de diamètre. Le nombre des veines mastoïdiennes est, en effet, variable. La veine peut être unique, double ou triple ; chacune d'elles passe à travers la surface extérieure de l'os par un orifice spécial ; les orifices sont, en général, très rapprochés l'un de l'autre ; dans l'épaisseur de l'os ils convergent en un canal unique, dans lequel les veines se réunissent en un seul tronc, qui va se jeter dans le bord postérieur du sinus latéral, au-dessous de son coude. Les dimensions des orifices et du canal intra-osseux varient suivant le volume même de la veine. Leur situation, à la surface de l'os, se trouve toujours en arrière de la ligne des insertions musculaires. Mais, dans cette partie postérieure de la face externe de la mastoïde, leur position est très variable : la veine peut pénétrer dans l'os à la partie la plus déclive de la face externe, au voisinage de la limite postérieure de la région digastrique (fig. X) ; l'orifice extérieur du canal veineux peut aussi se trouver très haut, au voisinage de l'angle supéro-postérieur de la mastoïde, ou bien occuper une position intermédiaire à ces points extrêmes. Le canal intra-osseux a toujours une direction oblique et n'est jamais franchement transversal. Si l'orifice extérieur est haut placé, la direction du canal intra-osseux est oblique en bas et en avant vers le sinus ; s'il est très bas placé, sa direction est, au contraire, oblique en haut et en avant. La longueur du canal varie suivant cela même ; il peut avoir quelquefois deux centimètres de long. Les veines mastoïdiennes sont rarement blessées au cours des trépanations mastoïdiennes ordinaires, car le plus souvent le champ d'opération reste limité en avant de la ligne des insertions musculaires, en arrière de laquelle elles se trouvent. Mais, lorsque les lésions osseuses obligent à étendre le champ opératoire dans la partie reculée de l'écaille de la mastoïde, la rugination du périoste peut amener l'ouverture de ces vaisseaux. Si la veine est peu volumineuse l'hémorragie est de peu d'importance et s'arrête facilement sous la compression.

Mais si la veine est de gros calibre, l'hémorragie ressemble à celle que produit l'ouverture du sinus latéral lui-même et devient fort génante, surtout s'il faut réséquer les parties osseuses, qui entourent le trajet intra-osseux de la veine.

Les rapports directs, qui existent entre les veines mastoïdiennes et le sinus latéral, expliquent l'importance des hémorragies de ces veines, ainsi que la gravité des inflammations, qui peuvent les atteindre au cours

de lésions mastoïdiennes : la phlébite mastoïdienne se propage avec la plus grande facilité au sinus latéral.

(*e*) *Suture pétro-squameuse externe.* — A la surface extérieure de la mastoïde, on voit souvent une ligne de suture de même ordre que celles qu'unissent entre eux les os du crâne. Elle indique la formation primitive de cette région aux dépens de deux et même trois os : écailleux, tympanal, pétreux. On l'appelle suture pétro-squameuse externe (je l'ai, dans un autre travail, appelée plus exactement pétro-squameuse externe et postérieure).[1] Il convient au chirurgien de bien connaître la valeur et les rapports de cette ligne, car elle peut être un lieu très important pour la propagation des suppurations endomastoïdiennes vers le périoste, en même temps qu'elle peut servir de ligne d'orientation au cours des interventions sur la mastoïde. Mais sa présence est très inconstante. Très souvent, chez l'enfant comme chez l'adulte, elle a disparu, ne laissant aucune trace. D'autrefois, elle persiste sous forme de tracé sutural plus ou moins bien marqué à la surface de l'os. Ce tracé sutural existe rarement dans toute son étendue. Le plus souvent cette ligne est fragmentée. Parfois il n'en reste que quelques points à peine marqués. D'autrefois sa place est indiquée par des trous à bords déchiquetés, creusés dans la corticale osseuse. Quelquefois elle se manifeste par une encoche osseuse à bords irréguliers de 1 cm. à 1½ cm. de long (fig. X). Plus rarement une encoche profonde parcourt la mastoïde de haut en bas (fig. XI). Très exceptionnellement, enfin, il a été donné de voir cette suture bien complète et bien conservée dans toute son étendue, chez l'adulte : on ne connaît de ceci que de très rares exemples, dont j'ai récemment rapporté un cas, qui appartient à la collection du professeur Brühl, de Berlin, et que j'ai reproduit dans mon récent travail sur la structure de la mastoïde.[2]

La suture pétro-squameuse externe et postérieure se trouve dans la région opératoire habituelle, c'est-à-dire en avant de la ligne rugueuse, qui donne insertion aux muscles mastoïdiens. Elle s'étend de l'angle squamo-mastoïdo-pariétal au milieu de la base de l'apex, mais elle peut aussi descendre jusqu'au sommet même de l'apex ; de même, en haut, elle peut partir de la suture mastoïdo-pariétale, en arrière de l'angle squamo-mastoïdo-pariétal (fig. I, 9). Ceci dépend de la façon dont la partie de l'os écailleux, que j'ai appelée *aile squamo-mastoïdienne*, s'étale à la surface de la base du rocher, pour prendre part à la constitution de la région mastoïdienne. L'étude du temporal chez le fœtus et l'étude du temporal, que le professeur Brühl possède dans sa collection, montrent bien quelle est sa place réelle et quels sont les endroits où, chez l'adulte normal, on peut trouver des vestiges plus ou moins importants de cette suture. Cette ligne occupe, dans la pièce du professeur Brühl,

[1] Mouret, 'Étude sur la structure de la mastoïde et sur le développement de cellules mastoïdiennes. — Influence de la constitution de la mastoïde sur l'évolution des suppurations antro-cellulaires,' *Annales des maladies de l'oreille*, 1913, et *Congrès français d'O.-R.-L.*, mai 1912.

[2] Mouret, loc. cit.

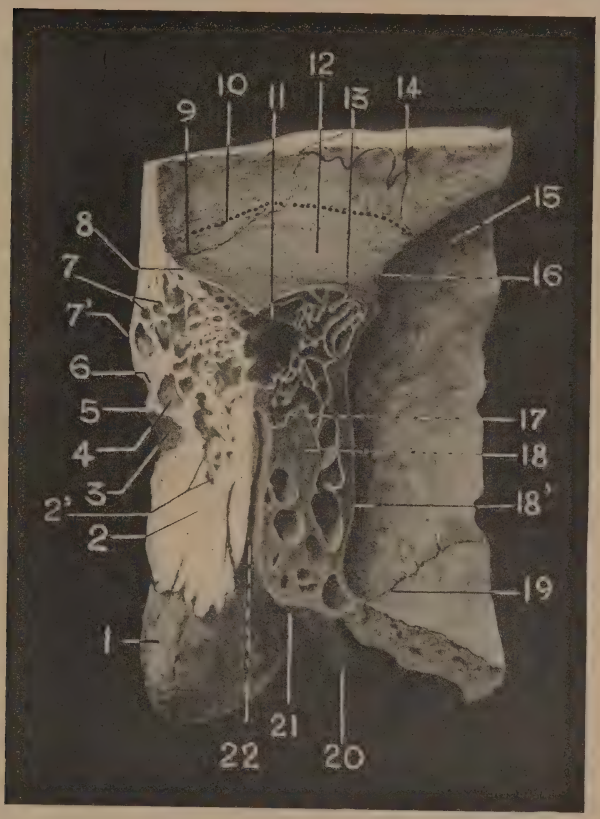


FIGURE III

Bloc mastoïdien détaché du temporal et se montrant par l'angle, qui sépare la surface de section antérieure de la surface de section interne.

1, apex mastoïdien : vue de son bord antérieur et de sa face digastrique.—2, face antérieure du bloc mastoïdien. La coupe a rasé un peu trop la paroi postérieure du conduit auditif.—2′, cellules limitrophes ouvertes par la coupe.—3, partie externe de la paroi postérieure du conduit auditif externe, non-touchée par le trait de scie.—4, cellule de la paroi externe, située en dedans de 5, spina-supra-méatum.—6, fossette sus ou rétro-spinale.—7, cellules de la paroi externe de l'antre situées au-dessus de la linéa temporalis.—7′, linéa temporalis très basse, sur cette pièce, par rapport à la cavité crânienne.—8, bord supérieur de la face antérieure du bloc, ou bord antérieur de la face supérieure.—9, suture pétro-squameuse interne.—10, ligne pointillée indiquant l'angle latéral de la base du crâne.—11, aditus ad antrum.—12, face supérieure du bloc mastoïdien.—13, bord interne de la face supérieure ou bord supérieur de la face interne.—14, suture pariéto-mastoïdienne.—15, gouttière du sinus latéral.—16, bord postérieur de la face supérieure du bloc.—17, coude de l'aqueduc de Fallope.—18, face interne (pétreuse) du bloc mastoïdien.—18′, bord intra-crânien de la face interne.—19, suture mastoïdo-occipitale inférieure.—20, bulle digastrique.—21, rainure digastrique.—22, dernière portion de l'aqueduc de Fallope.

le *pourtour* de l'aile squamo-mastoïdienne. Elle part de l'angle de suture squamo-mastoïdo-pariétal, descend, presque verticalement, jusque vers le *milieu* de la base de l'apex, se dirige ensuite horizontalement à la surface de cette base jusqu'au niveau du bord externe de l'arc postérieur de l'os tympanal, puis elle remonte le long de celui-ci. En réalité cette dernière partie n'est plus la suture pétro-squameuse externe, mais plutôt une suture squamo-tympanale. L'une et l'autre, lorsque l'aile squamo-mastoïdienne n'est pas soudée à la partie du rocher qu'elle recouvre (cas de Brühl, Frey), conduisent dans l'interstice, qui sépare ces deux os. La ligne de suture, tracée à la surface de l'os, conduit alors dans une profonde *fissure interosseuse* (*intersquamo-mastoïdienne*), dont les deux parois sont formées par une lame osseuse de tissu compact du genre cortical. Ces deux lames sont complètement indépendantes l'une de l'autre, dans la pièce du professeur Brühl. Elles représentent les deux parties constituantes de la cloison intercellulaire (intersquamo-mastoïdienne), décrite chez le fœtus par Schwartze et Eyselle. La lame squameuse et la lame pétreuse de cette cloison aboutissent, en haut, au plancher de *l'antre*, et la fissure interosseuse (pétro-squameuse), qui les sépare l'une de l'autre, arrive aussi jusqu'à la cavité antrale. L'antre se trouve donc communiquer avec l'extérieur de l'os par l'intermédiaire de la fissure pétro-squameuse, plus ou moins comblée par du tissu conjonctif. Cette communication permet ainsi de comprendre quelle importance il convient d'accorder à toutes parties de cette fissure pouvant persister chez l'adulte : bien que comblées par le tissu conjonctif, ces parties n'en restent pas moins un passage plus facile pour les suppurations antrales vers l'extérieur. C'est ainsi que, maintes fois, les abcès périostés de la surface extérieure de la mastoïde se produisent facilement, grâce au passage des germes infectieux des suppurations antrales le long des vestiges de la fissure pétro-squameuse externe. On peut, dans ces cas, en l'absence même de toute lésion osseuse apparente, voir, au moment de la trépanation, que le tissu conjonctif, qui comble ces vestiges suturaux, est rouge et particulièrement enflammé ; quelquefois même, en pressant sur l'os avoisinant, on peut voir en sortir quelques gouttes de pus. L'étude d'un temporal, dans lequel l'os écailleux est indépendant de l'os pétreux et de l'os tympanal (cas de Frey, Brühl, Guyes), nous montre donc l'importance de la suture pétro-squameuse externe, comme voie de propagation des suppurations antrales à la surface extérieure de la mastoïde, en même temps qu'elle précise la théorie émise par Schwartze et Eyselle sur l'indépendance, qui existe primitivement entre les cellules d'origine écailleuse et celles d'origine pétro-mastoïdienne. Les cas de ce genre sont, d'ailleurs, excessivement rares et correspondent toujours à un certain arrêt de développement des parties osseuses. Aussi ne donnent-ils pas, pour les cas normaux, une indication absolue sur la place qu'il convient d'assigner à la suture pétro-squameuse externe, lorsqu'elle n'est représentée que par des vestiges rares et peu apparents à la surface de l'os. Le tracé de cette suture à la surface de l'os varie, suivant que l'aile

squamo-mastoïdienne s'est plus ou moins développée à la surface de la mastoïde.

Ligne pariéto-apicale. — Mais l'étude d'un grand nombre de mastoïdes permet de tracer à la surface de l'os une ligne conventionnelle, au niveau de laquelle on peut repérer, tout à la fois, les vestiges de la suture pétro-squameuse externe et la situation normale du sinus latéral. Cette ligne s'étend de l'angle pariéto-squamo-mastoïdien au milieu du sommet de l'apex : je l'appelle ligne *pariéto-apicale* (fig. I, *e–d*). Elle est oblique de haut en bas et d'arrière en avant. Elle correspond au point de jonction de la partie pétreuse de la mastoïde avec sa partie cérébelleuse. La suture pétro-squameuse externe se trouve, normalement, sur le trajet même de cette ligne (fig. XI). Elle est très rarement en avant d'elle, mais elle peut se trouver plus loin en arrière (fig. I).

Lorsque la suture se trouve sur le trajet de la ligne pariéto-apicale, elle apparaît parfois comme une large entaille creusée profondément dans l'os (figs. X et XI). Lorsqu'elle se trouve en arrière de cette ligne, elle est toujours réduite à de simples vestiges, peu apparents et marqués très superficiellement (fig. I). Ceci tient à ce que l'aile squamo-mastoïdienne de l'os écailleux s'est alors largement étalée à la surface de la mastoïde. Or, plus cette aile s'élargit, plus son bord postérieur s'amincit : la *fissure* primitive paraît alors très superficielle, bien que, en réalité, son étendue entre la surface de l'os et l'antre soit plus grande.

La suture peut arriver, en bas, jusqu'au sommet de l'apex (fig. XI), mais le plus souvent elle ne dépasse guère la ligne horizontale, qui indique la limite de la base de l'apophyse. Ceci est encore en rapport avec l'étendue de l'aile squamo-mastoïdienne, qui, en général, ne se développe qu'à la surface des régions antrale et sous-antrale (voir plus loin ce que j'entends par régions antrale et sous-antrale).

La situation du sinus latéral est aussi indiquée par le tracé de la ligne pariéto-apicale à la surface de la mastoïde. Cette ligne correspond à l'union de la base de la pyramide pétreuse avec la partie postérieure (cérébelleuse) de la mastoïde, le sinus se trouve en arrière et le long de son bord postérieur, sauf dans les cas anormaux où le sinus est plus ou moins procident (fig. I).

Enfin la ligne pariéto-apicale forme la limite postérieure de la zone de trépanation de l'antre, dans laquelle on peut opérer librement, si le sinus n'est pas procident et si l'antre est normalement développé. Le tracé de cette ligne à la surface des mastoïdes petites montre combien, dans ces cas, est étroite la zone antrale et combien doit, alors, être prudemment mesurée l'étendue de la surface d'attaque de l'os, car les mastoïdes petites sont celles où l'on trouve le plus souvent le sinus procident et l'antre en position dite fœtale.

§ II. **Face antérieure.** — La face antérieure du bloc mastoïdien est tracée par un plan vertico-transversal, perpendiculaire à l'axe médian et antéro-postérieur du crâne. Ce plan passe immédiatement en avant de l'apex, de la paroi postérieure du conduit auditif externe et de la

paroi postérieure de la caisse. Pour que la paroi postérieure du conduit, qui est légèrement oblique de dehors en dedans et d'arrière en avant, soit contenue tout entière à la surface de la coupe, il faut que le trait de scie passe, à la surface extérieure de l'os, un peu avant de la spina-supra-méatum et du bord postérieur du méat auditif externe. Or je tiens à faire remarquer que sur la pièce, qui sert à faire ma démonstration, le trait de scie (*a–b*, fig. I) se trouve à 1 millimètre environ un peu trop en arrière : il entaille le bord postérieur du méat et l'extrémité supérieure de la spina supra-méatum. Il en est résulté que la coupe, au lieu de passer en avant de la paroi postérieure de conduit et de la caisse, est passée à travers l'épaisseur de l'arc postérieur de l'os tympanal (fig. III, 2) et, qu'au lieu de montrer toute la face postérieure du conduit, elle n'en présente qu'une minime portion (3). C'est là une faute de préparation de peu d'importance. Il eût été facile de la réparer et de reproduire une coupe plus exactement orientée, mais j'ai préféré la donner telle qu'elle est, parce que, après avoir fait ces remarques, je lui trouve deux avantages. 1° En entaillant la couche superficielle de la paroi postérieure du conduit, elle permet de voir combien les cellules qui se trouvent dans son épaisseur (2′), et qu'on appelle cellules limitrophes du conduit, arrivent très superficiellement sous la mince corticale osseuse, que forme la lame postérieure de l'os tympanal. 2° En coupant l'extrémité supérieure de la spina supra-méatum, elle permet de retrouver sur son bord externe la trace de l'épine (5) et de la fossette rétrospinale (6), ce qui permet aussi de voir quelle est, par rapport à la cavité adito-antrale, la situation de ces deux parties, qui forment deux points de repère précieux pour l'attaque de la surface extérieure de la mastoïde, lors de la trépanation antrale. L'étude de cette coupe (figs. I et III) nous montre que la partie supérieure de l'épine de Henle (5) correspond au plancher de l'aditus ad antrum, au-dessous duquel se trouve, immédiatement, le deuxième coude de l'aqueduc de Fallope (17) avec le nerf facial. Elle nous montre aussi la richesse de la paroi externe de l'aditus en cavités pneumatiques (7).

Il convient encore de faire remarquer que, sur cette pièce, la linéa temporalis (3, fig. I) était très basse, par rapport à la cavité crânienne et que l'antre (12) était très haut placé.

§ III. **Face interne.** — J'entends par face interne de la région mastoïdienne une surface artificielle obtenue en faisant une coupe verticale et antéro-postérieure, qui sépare la région mastoïdienne de la pyramide pétreuse. Cette coupe tombe verticalement sur celle qui sert à marquer la limite antérieure du bloc mastoïdien et dont je viens de parler. La surface de cette coupe est sensiblement parallèle à l'axe médian du corps et est diamétralement opposée à la surface extérieure de la mastoïde : c'est pour cela que je l'appelle face ou limite interne du bloc mastoïdien. On peut aussi l'appeler face ou limite pétreuse du bloc, car le plan de section passe à travers le rocher, immédiatement en arrière des canaux semi-circulaires. Les figures II, 8′ et III, 18 montrent que ce plan de

section passe à travers la gouttière du sinus latéral, au niveau de la ligne de suture mastoïdo-occipitale, et qu'elle entaille la face postérieure de la pyramide pétreuse à une certaine distance de la fente unguéale. La coupe passe à travers le rocher et longe le bord convexe des canaux demi-circulaire externe et demi-circulaire postérieur. Elle tombe perpendiculairement sur le plan de section, qui sert à délimiter la face antérieure du bloc (8′ et 8, fig. II). Le plan de section passe, ainsi, en dedans de l'antre et de l'aditus. Toutefois la paroi interne de ce dernier, qui correspond au canal demi-circulaire externe lui-même, se trouve laissée hors du bloc mastoïdien : on ne peut faire autrement sans ouvrir le labyrinthe.

La troisième portion de l'aqueduc de Fallope se trouve contenue dans la région mastoïdienne au voisinage de l'arrête osseuse, que forment, en se rencontrant, la *face antérieure* et la *face interne* du *bloc mastoïdien*. Dans la figure III l'aqueduc se montre ouvert sur toute son étendue : cela tient à ce que le plan de section, qui sert à marquer la limite interne du bloc, est passé à 1 mm. trop en dehors. En revanche cette petite erreur de coupe a l'avantage de montrer la situation de l'aqueduc dans l'intérieur même de la région mastoïdienne.

L'aspect que présente la surface interne du bloc mastoïdien est variable, suivant que la constitution de l'os est spongieuse, compacte ou pneumatique. Sur la pièce (fig. III), qui sert ici à ma démonstration, elle est creusée de nombreuses et grandes cellules, qui s'étagent de haut en bas entre la face supérieure et la face inférieure (bulle digastrique). On retrouve ces cellules sur l'autre face de la même section dans la figure IV, où elles se prolongent dans le rocher (zone cellulaire intersinuso-auditive).

§ IV. **Face postérieure ou postéro-interne.** — La face postérieure de la région mastoïdienne se moule sur le cervelet et forme une surface concave dans son ensemble, qui borde la partie correspondante de la fosse cérébelleuse. Elle n'est donc pas diamétralement opposée à la face antérieure : dans son ensemble elle regarde en arrière et en dedans, elle est donc plutôt postéro-interne. Elle correspond à la partie intra-crânienne du temporal, qui se trouve au-dessous du plan horizontal qui sert à isoler l'écaille de la région mastoïdienne, en arrière et en dehors du plan vertico-transversal qui sert à délimiter la face interne ou pétreuse du bloc mastoïdien. La figure II montre bien cette paroi *postéro-interne* de la région mastoïdienne. Elle se divise en trois parties bien nettement marquées sur le squelette : une partie *rétropétreuse*, une partie *sinusienne* et une partie *cérébelleuse*.

(*a*) *Partie rétropétreuse.* — (fig. II, 4). Cette zone correspond à la partie la plus externe de la face postérieure (cérébelleuse) de la pyramide pétreuse, située dans l'angle que forme la portion descendante du sinus latéral avec le bord supérieur du rocher. Elle a une forme triangulaire ; son sommet est dirigé en bas, vers le point de rencontre de la ligne de suture mastoïdo-pétreuse (7) avec le rocher. L'étendue de ce triangle rétropétreux est variable, suivant les sujets, et ceci est lié au volume même de la pyramide pétreuse. La base est tournée en haut et correspond

au bord supérieur du rocher. Sur la pièce qui a servi à faire les photographies II, III, IV son diamètre vertical, étendu du sommet à la base du triangle, mesure 18 mm. Ce diamètre varie entre 15 à 20 mm. La base du triangle mesure 11 mm., elle varie de 10 à 15 mm. Des trois bords qui limitent le triangle rétro-pétreux, deux sont formés naturellement : le supérieur par le bord supérieur de la pyramide pétreuse, le postérieur par la lèvre antérieure de la gouttière du sinus latéral. Le troisième, bord antérieur, est conventionnel et correspond à la ligne de section (8'), qui, passant à travers la pyramide, sert à marquer la limite interne (pétreuse) du bloc mastoïdien. Ce bord antérieur passe en arrière de la fente unguéale, tantôt rasant cette fente, tantôt en restant distante de quelques millimètres : Ceci dépend de la forme même que peut présenter la fente unguéale. Le bord supérieur, formé par le bord supérieur du rocher, subit les variations de forme propres à ce dernier : tantôt rectiligne, tantôt incurvé, quelquefois mince et tranchant, d'autres fois mousse et arrondi, ou bien encore saillant vers la cavité cérébelleuse et surplombant la surface même du triangle rétro-pétreux (fig. XIII). Ce bord correspond à la ligne d'insertion de la tente du cervelet et au trajet du sinus pétreux supérieur. Le bord postérieur correspond à la lèvre antérieure du sinus latéral. Il est oblique de haut en bas et d'arrière en avant, comme le sinus lui-même. C'est de la situation de ce bord, c'est-à-dire de la position occupée par le sinus latéral, que dépend, tout particulièrement, la largeur du triangle mastoïdo-pétreux. Ce bord est d'autant plus marqué que la gouttière du sinus latéral est plus creusée dans l'os. Chez certains sujets, il est à peine indiqué, la gouttière du sinus étant elle-même à peine marquée à la surface de l'os. Chez d'autres il peut former une arête vive, en forme de crête ou d'éperon parfois très saillant, surtout lorsque l'os est petit, très peu pneumatique, et que le sinus occupe la position dite procidente (figs. XIII et XV).

La surface du triangle rétro-pétreux peut être plane ou légèrement bombée, lorsque l'os est volumineux, surtout lorsque sous la corticale se trouvent développées de grandes cellules comme dans la figure VI. Elle peut être légèrement concave, reproduisant l'empreinte de la convexité correspondante du cervelet. Cette forme concave atteint son maximum lorsque la paroi interne de l'antre est très mince et que la pyramide est étroite et dépourvue de cellules pneumatiques. Le triangle mastoïdo-pétreux forme la partie la plus antérieure et la plus profonde de la face postéro-interne de la région mastoïdienne.

Au point de vue chirurgical, cette partie de la face postéro-interne du bloc mastoïdien a une très grande importance. Sa moitié supérieure correspond à l'antre, lorsque celui-ci a des dimensions normales. Sa moitié inférieure correspond à ce que j'appellerai plus loin la zone mastoïdienne sous-antrale de la face externe. Cette zone rétro-pétreuse représente le lieu d'élection pour aborder le cervelet en avant du sinus latéral. Sa partie supérieure, antrale, correspond à la région dite bloc de Janssen, que cet auteur abat pour ouvrir les collections suppurées de la fosse



FIGURE IV

Vue du temporal après ablation du bloc mastoïdien.—1, cellules pétreuses comprises entre la face postérieure du rocher et la paroi interne de l'aditus.—2, ligne pointillée indiquant la procidence du golfe de la jugulaire.—3, fente unguéale.—4, épine jugulaire.—5, fosse jugulaire.—6, ligne d'une section faite dans le sens vertical et antéro-postérieur correspondant à 10 de la fig. II.—7, partie terminale de la gouttière du sinus latéral.—8, cellules pétreuses.—9, suture occipito-mastoïdo-inférieure.—10, apophyse jugulaire de l'occipital.—11, portion descendante de l'aqueduc de Fallope. Comme je l'ai expliqué dans le texte, ce canal se trouve intéressé par la coupe, parce que le plan de section passe à 1 ou 2 mm. trop en dehors. —12, canal vasculaire accessoire de l'aqueduc de Fallope.—13, angle de section.—14, conduit auditif externe, dont la partie externe de la paroi postérieure est seule restée dans le bloc mastoïdien. Ceci, comme je l'ai dit aussi dans le texte, est dû à ce que le plan de section aurait dû être fait un peu plus en avant.—15, spina supra-méatum.—16, fossette sus ou rétro-spinale.—17, 2e coude de l'aqueduc de Fallope.—18, apophyse zygomatique.—19, racine horizontale de cette apophyse.—20, cellules de la paroi supérieure du conduit auditif externe.—21, cavité épitympanique.—22, saillie hémisphérique du canal demi-circulaire externe, située sur la paroi interne de l'aditus, laquelle reste hors du bloc mastoïdien.—23, face supérieure de la pyramide pétreuse.—24, bord supérieur (supéro-postérieur) de la pyramide pétreuse.

Les figures V, VI, VII, VIII représentent les faces externe, postéro-interne, supérieure et inférieure de la région mastoïdienne d'un temporal très pneumatique.

cérébelleuse. C'est une des voies les plus importantes pour la propagation des suppurations antrales vers le cervelet. C'est par elle qu'on doit pénétrer dans la loge cérébelleuse pour ouvrir l'empyème du sac endolymphatique et pour pénétrer dans le cervelet par voie présinusienne. Sa partie inférieure doit, aussi, être enlevée pour mettre à nu le bulbe de la jugulaire, surtout lorsque ce bulbe est procident dans la profondeur du rocher, comme dans la figure II.

(*b*) *Partie sinusienne.* — (fig. II, 5). — La partie sinusienne de la face postéro-interne de la région mastoïdienne correspond à la gouttière que dessine, sur la face endocrânienne du temporal, la portion descendante du sinus latéral. Cette gouttière est oblique de haut en bas, d'arrière en avant et de dehors en dedans. Étant donnée cette courbure générale, sa moitié supérieure regarde en dedans, sa moitié inférieure regarde en dedans mais aussi en haut.

La première partie correspond à la face externe de la mastoïde, la deuxième court au-dessus de sa face inférieure. Sa largeur, sa profondeur, ses modalités de situation tiennent, tout à la fois, au volume et à la situation du sinus et à la constitution elle-même de la région mastoïdienne.

Sa largeur est en général de 1 cm., mais ceci est lié au volume du sinus latéral, qui est assez souvent inégal de sujet à sujet et même d'un côté à l'autre, sur un même sujet. Le sinus gauche est souvent plus petit que le droit. La gouttière sinusienne siège dans l'angle que forme la face postérieure du rocher avec la face cérébelleuse de l'écaille mastoïdienne, et empiète à peu près également sur l'une et l'autre de ces parties. Le long de cet angle, elle s'étend depuis le coude du sinus jusqu'au trou déchiré postérieur. Il peut arriver que la gouttière n'existe que dans sa partie supérieure, jusqu'au niveau du trou que traverse la veine mastoïdienne, comme si la partie inférieure du sinus latéral manquait. Dans ce cas l'orifice de la veine mastoïdienne est très volumineux. Sur une de mes pièces et sur une des pièces de la collection du professeur Brühl, cet orifice a un diamètre de 7 mm. N'ayant pu voir que le squelette de pareilles pièces, je ne puis dire si la dernière portion du sinus latéral manquait réellement. Mais, comme dans ces cas, le trou déchiré postérieur est beaucoup plus petit que normalement, j'en conclus que la partie inférieure du sinus devait être, tout au moins, rudimentaire. Il n'y aurait d'ailleurs rien d'étonnant à ce que cette partie manquât complètement, car il en est ainsi dans les premiers temps du développement chez le fœtus : au début, la circulation sinusienne sort du crâne non par le trou déchiré postérieur, mais par la grande veine mastoïdienne. De telles dispositions correspondent donc à un arrêt plus ou moins accentué du développement du système veineux sinuso-jugulaire.

La gouttière du sinus latéral ne suit pas toujours exactement l'angle pétro-mastoïdien de la face cérébelleuse du temporal. Le sinus peut occuper une position dite procidente, c'est-à-dire s'avancer plus ou moins en avant de l'angle pétro-mastoïdien. Dans ce cas la gouttière du sinus latéral est déplacée en avant, et empiète plus ou moins sur la portion

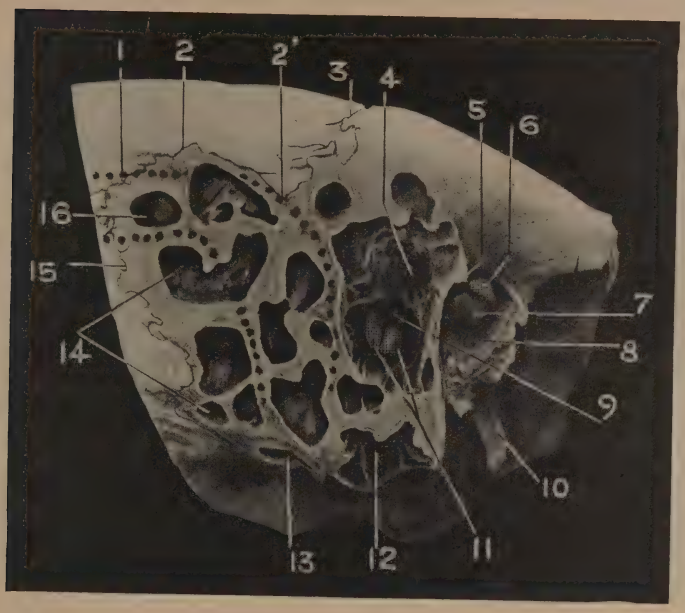


FIGURE V

Face externe d'une mastoïde très pneumatique. Les cellules sont très grandes et ont été mises à nu par perforation de la corticale externe. Au niveau de l'antre les cellules de sa paroi externe ont été complètement enlevées pour mettre à jour l'antre lui-même. Dans la région sous-antrale les cellules ont été, aussi, complètement évidées dans la profondeur, jusqu'au niveau des cellules, qui se trouvent sous la paroi postérieure du rocher, que l'on voit marqué d'une + sur la fig. VI.

1, gros pointillé noir indiquant la projection de la gouttière du sinus latéral à la surface extérieure de la mastoïde.—2, suture mastoïdo-pariétale.—2', angle squamo-mastoïdo-pariétal.—3, suture squamo-pariétale.—4, antre mastoïdien.—5, spina-supra-méatum.—6, ouverture artificielle éclairant la voûte de la caisse, due à ce que le tegmen-tympani a été enlevé (voir fig. VII).—7, paroi interne de la caisse, éclairée par l'ouverture 6.—8, conduit auditif externe.—9, région sous-antrale évidée ; dans la profondeur on voit : 11, deux orifices éclairant le fond de la région sous-antrale et dus à l'ouverture large des cellules qui sont marquées par + et ++ dans la fig. VI.—10, apophyse styloïde.—12, cellules de l'apex, ouvertes par ablation du sommet de l'apex.—13, cellules de la bulle digastrique. Pour faire voir la bulle (13), les cellules de l'apex (12) et l'orifice de la voûte de la caisse (6), la pièce a été un peu inclinée de bas en haut et en dedans.—14, cellules de la zone cérébelleuse de la mastoïde.—15, suture mastoïdo-occipitale postérieure.—16, cellules de la zone sinusienne.

rétro-pétreuse du bloc mastoïdien : Le sinus peut être procident à la surface de l'os ou bien creuser une loge profonde à la base de la pyramide.

Dans la première disposition le sinus glisse en avant de l'angle pétro-mastoïdien à la surface même de la pyramide. La gouttière sinusienne est alors tracée à la surface de la zone, que je viens d'appeler *triangle rétro-pétreux*. Dans ce cas la gouttière est superficielle,[1] mais la zone rétro-pétreuse se trouve bien diminuée et le sinus se trouve alors en rapport avec la paroi interne de l'antre (voir fig. XVI, loc. cit. in *Annales des Mal. de l'Oreille*, n° 2, 1913).

Dans la deuxième disposition le sinus procide en avant et en dehors. La gouttière sinusienne est alors très profondément creusée à la base de la pyramide pétreuse. Son bord pétreux est très saillant et peut même former une arête volumineuse, qui le surplombe à la façon des tuiles qui sont au bord d'un toit (fig. XIII). Lorsque le sinus est très procident en avant et en dehors, la gouttière du sinus latéral peut n'être séparée de la paroi postérieure du conduit auditif externe que par une lame osseuse excessivement mince. Le sinus procident en avant et en dehors occupe, ainsi, ce que j'appellerai plus loin, la zone pétreuse de la surface extérieure de la mastoïde, c'est-à-dire la zone opératoire (figs. XIII, XV, XIX). De telles dispositions correspondent à un arrêt de développement de la base de la pyramide pétreuse : la saillie mastoïdienne est alors très petite et la structure de l'os est assez anormale ; les cellules mastoïdiennes manquent ; l'antre est rudimentaire, haut placé, en position fœtale et a, à peine, le volume d'un antre fœtal du 5e mois, et représentant un simple diverticule de la cavité épitympanique. L'antre est alors cantonné au niveau de la région, qui correspond à l'angle supéro-postérieur du conduit auditif. Il en est ainsi sur les pièces représentées par les mêmes figures.

Le sinus peut encore présenter une autre modalité de rapports avec la pyramide pétreuse. Celle-ci n'est d'ailleurs qu'une variante du cas précédent, mais plus dangereuse encore au point de vue chirurgical. Cette disposition est heureusement fort rare. J'en ai vu un exemple remarquable dans la collection du professeur Brühl (figs. XIX et XX). Je reviendrai plus loin sur ce point, à propos des anomalies de la région mastoïdienne.

(*c*) *Partie cérébelleuse.* — (fig. II, 6). — Cette partie de la face postéro-interne du bloc mastoïdien en est la partie la plus reculée. Elle correspond à la portion rétro-sinusienne de la face interne de ce qu'on appelle lame cérébelleuse ou écaille de la mastoïde. Elle est limitée en arrière et en bas par la suture mastoïdo-occipitale (7) ; en haut, par le coude du sinus latéral. Elle n'est pas plus large que la région sinusienne, quelquefois même plus étroite. Elle a, en général, 1 cm. de largeur. Elle s'étend moins haut que la région sinusienne et n'arrive pas jusqu'à la suture pariéto-mastoïdienne, car le coude du sinus latéral recouvre la partie supérieure de l'écaille mastoïdienne à ce niveau. Aussi la hauteur de la zone mastoïdienne cérébelleuse est-elle diminuée de toute la largeur

[1] Du côté de la cavité crânienne.

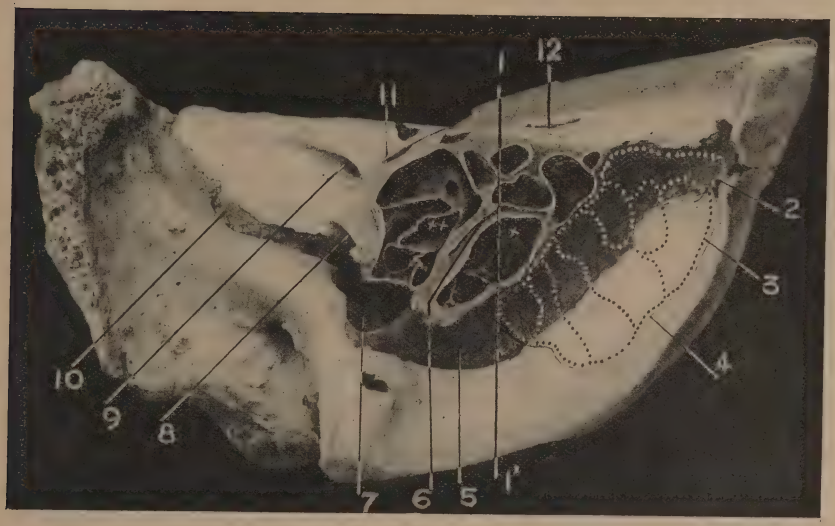


FIGURE VI

C'est la même pièce représentée dans la fig. V, qui est vue par la fosse cérébelleuse. Les cellules de la pyramide ont été ouvertes par le même procédé, par perforation de la corticale pétreuse. Les cellules de la zone sinusienne et de la zone cérébelleuse ont été seulement représentées en pointillé à la surface endocrânienne de l'os. Les cellules de cette pièce sont peu nombreuses, mais très grandes et ne forment qu'une couche entre la corticale externe et la corticale intra-crânienne, dans les régions sinusienne et cérébelleuse, de sorte que si on avait perforé celles-ci on n'aurait pas eu de cavités cellulaires, mais seulement des trous communiquant avec l'extérieur, puisque déjà la corticale avait été enlevée à la surface extérieure de la mastoïde (fig. V).

1–1′ est une ligne factice, correspondant au plan de section 8′ de la fig. II, et marquant la limite entre le bloc mastoïdien et la région pétreuse proprement dite.—2, pointillé blanc indiquant les cellules de la zone sinusienne.—3, pointillé noir indiquant les cellules de la zone cérébelleuse.—4, suture mastoïdo-occipitale.—5, gouttière du sinus latéral.—6, fente unguéale.—7, fosse jugulaire.—8, fossette pyramidale.—9, trou auditif interne.—10, gouttière du sinus pétreux inférieur.—11, fossa subarcuata. Le canalis subarcuatus (canal pétromastoïdien) est ouvert et aboutit à cette fossa.—12, toit tympano-antral ouvert. La face supérieure de la pyramide a subi la préparation, qui est représentée dans la fig. VII.

du coude de la gouttière sinusienne, et n'a guère plus de 2 cm. à 2½ cm. d'étendue. D'ailleurs cette hauteur varie un peu, suivant que la gouttière du sinus est plus ou moins haut placée et qu'elle empiète plus ou moins sur la face interne du pariétal. Cette partie de la face postéro-interne de la région mastoïdienne regarde directement en dedans.

§ V. **Face supérieure.** — La face supérieure de la région mastoïdienne (fig. II, 16 ; fig. III, 12) fait partie de l'étage moyen de la base du crâne et correspond à la partie externe de la face supérieure de la pyramide pétreuse. Elle a la forme d'un quadrilatère. Elle est limitée, en avant, par la ligne (8, fig. II), qui correspond au plan de section vertico-transversal, qui sert à tracer la limite antérieure du bloc mastoïdien ; en dedans, par la ligne (8′) qui correspond au plan vertical et antéro-postérieur, qui sert à tracer sa limite interne ou pétreuse (fig. III, 13) ; en arrière, par le bord supérieur du rocher (16) ; en dehors, par une ligne correspondant à l'angle, que forment, en se réunissant, la face interne de l'os écailleux et de l'os pariétal avec la face supérieure de la pyramide pétreuse (angle latéral de la base du crâne). La moitié postérieure de cet angle est bordée par la suture pétro-pariétale (fig. III, 14), qui est presque toujours assez nettement marquée ; la moitié antérieure est bordée par la suture pétro-squameuse interne (fig. III, 9), qui au contraire, est souvent, chez l'adulte, peu marquée en cet endroit. Cette face correspond au lobe temporal du cerveau et présente l'empreinte de la face inférieure de la troisième circonvolution temporale.

Le long de l'angle latéral pétro-squameux on trouve, parfois, un sillon assez net, qui correspond à la situation du sinus pétro-squameux, lequel peut persister chez l'adulte (Cheatle). Mais ce sillon est, le plus souvent, à peine marqué le long de la suture pétro-squameuse interne, et ne se prolonge pas, en arrière, jusqu'au bord de la pyramide. Le sillon aboutit, en effet, à un petit orifice osseux, qui mène dans un canal intra-osseux, dont on retrouve l'autre extrémité (fig. XIV, 24) ouverte dans la gouttière du sinus latéral, immédiatement au-dessous du bord supérieur de la pyramide. La face supérieure du bloc mastoïdien se trouve ainsi en rapport direct avec le sinus pétro-squameux, lequel collecte les veines qui viennent de l'intérieur de la caisse, de l'antre et des cellules susantrales. La phlébite de ces veines, au cours des suppurations mastoïdiennes, doit être une des causes fréquentes, bien que souvent méconnue, des thrombophlébites du sinus latéral. La face supérieure du bloc mastoïdien peut présenter souvent des déhiscences de la corticale osseuse. Les unes se trouvent sur la ligne de la suture pétro-squameuse interne et représentent des vestiges de cette fissure, vestiges plus ou moins grands, dans lesquels passent des vaisseaux, qui font communiquer la circulation de toit de l'antre avec la circulation intra-crânienne ; les autres peuvent être disséminées sur tous les points de la face supérieure du bloc mastoïdien. Celles-ci sont des déhiscences en *trou de mite* et conduisent soit dans les cellules sous-jacentes à la corticale pétreuse, soit dans l'intérieur de l'antre (fig. XXI, 5).

L'antre, en effet, est situé au-dessous de la face supérieure du bloc mastoïdien. Le grand axe de l'antre se trouve sur le prolongement du grand axe de la caisse et de la trompe d'Eustache. Comme celui-ci il a une direction oblique d'avant en arrière et de dedans en dehors. Projeté sur la face supérieure du bloc mastoïdien, l'axe antéro-postérieur de l'antre se trouve sur le trajet de la bissectrice de l'angle, que forment, en se rencontrant, les plans verticaux qui servent à délimiter, respectivement, la limite antérieure et la limite interne (pétreuse) de la région mastoïdienne (fig. II, 18).

§ VI. **Face inférieure.** — Cette face n'est, que je sache, décrite dans aucun livre d'anatomie. Elle constitue cependant une zone des plus importantes de la région mastoïdienne. Au point de vue anatomique cette face du bloc mastoïdien a une conformation et des limites bien précises ; au point de vue pathologique et chirurgical, elle est de la plus haute importance. Pour la voir dans son ensemble il suffit de regarder en face une base de crâne (fig. XII).

Sa forme générale est celle d'un triangle dont le sommet, tourné en avant, correspond à la base de l'apophyse styloïde (2). Un des côtés, l'externe, présente une forte saillie (l'apex mastoïdien (5), qui proémine en bas, formant une sorte de rempart extérieur, qui protège la face inférieure de la mastoïde, comme la malléole externe protège et recouvre l'interligne tibio-tarsien. La limite externe ou bord externe de cette face correspond donc au sommet et aux bords antérieur et postérieur de l'apex. Entre le bord antérieur de l'apex et l'apophyse styloïde, le bord externe de la face inférieure de la mastoïde correspond, en outre, à la portion de l'os tympanal, qui forme la paroi inférieure (3) du conduit auditif externe. La limite interne est formée par la suture mastoïdo-occipitale inférieure (9). La limite postérieure, qui forme la base du triangle, n'est pas tournée directement en arrière, mais regarde aussi un peu en dehors. Elle correspond à une ligne horizontale passant à la base de l'apex mastoïdien, sur la face extérieure de l'os (fig. I, *b'*, *d*, *b*).

La présence de l'apex mastoïdien donne à la face inférieure de la région mastoïdienne une forme spéciale. On peut lui considérer deux parties : une verticale, l'autre sensiblement horizontale. La première est formée par la face profonde (interne) de l'apex ; la deuxième par une lame osseuse, qui est comprise entre la base de la saillie apicale et la suture mastoïdo-occipitale (comparer la figure XII avec la figure III). Cette dernière en est la partie la plus importante. Dünn l'appelle lame occipitale de l'apophyse mastoïde, Pérez l'appelle crête ou bulle digastrique. Le nom de lame occipitale peut prêter à confusion avec la partie postérieure de la face externe de la mastoïde, qui est, elle aussi, lamelleuse et s'articule avec l'occipital, ou bien alors faudrait-il l'appeler lame occipitale *inférieure* de la mastoïde. Les noms de crête ou bulle digastrique me paraissent plus appropriés, mais comme ils préjugent trop de la forme extérieure de cette lame, je l'appellerai simplement *région digastrique*.

La face inférieure de la mastoïde se divise, ainsi, en deux parties : l'une apicale, externe et verticale, formée par la face interne de l'apex, l'autre interne et sensiblement horizontale, région digastrique. L'une et l'autre ont une forme à peu près triangulaire. Le sommet de l'une (pointe de l'apex) est dirigé en bas. Le sommet de l'autre est tourné en avant, vers la base de l'apophyse styloïde.

Le triangle apical ne nous présente rien d'important. Sa base correspond à la rainure digastrique et au bord externe du triangle digastrique. Ses dimensions sont d'autant plus grandes que l'apex est plus volumineux et que la rainure digastrique est plus profonde. Sa surface est lisse. La corticale osseuse qui la recouvre est souvent très mince ; aussi, lorsque l'apex est pneumatique, cette corticale forme une barrière très peu résistante et se laisse facilement détruire par la suppuration endo-apicale, qui se répand alors dans l'espace sous-mastoïdien. La fragilité de la corticale de la face interne de l'apex est ainsi une cause fréquente des ' abcès de Bezold '.

Le triangle digastrique est situé dans un plan sensiblement horizontal, mais légèrement convexe d'avant en arrière. Ses dimensions sont variables, suivant les sujets. Sur la pièce, qui a servi à faire la photographie XII, le triangle digastrique mesurait 3 cm., du sommet à la base, et avait 18 mm. de largeur, au niveau de la base elle-même. La forme du crâne influence tout particulièrement ces dimensions. Chez les Dolichocéphales, le triangle digastrique est plus petit et plus allongé. La présence de cellules dans l'épaisseur de l'os lui donne une forme plus ou moins globuleuse, tandis que, quand il n'est pas pneumatique, il a un aspect plutôt lamelleux. Au sommet du triangle digastrique se trouve le trou stylo-mastoïdien, qui représente l'orifice extra-crânien de l'aqueduc de Fallope et donne passage au nerf facial et aux vaisseaux stylo-mastoïdiens. Au fond de ce trou, à trois millimètres de profondeur sur sa paroi antérieure, se trouve un autre petit orifice, qui conduit dans un fin canal que parcourt la corde du tympan jusqu'à la paroi postérieure de la caisse. Tout autour du trou stylo-mastoïdien se trouve, enfin, une zone criblée de petits orifices, qui servent de passage aux vaisseaux, qui vont du périoste dans la profondeur de l'os. Tous ces divers petits orifices peuvent, dans certains cas, servir de voie de passage à la suppuration de l'oreille moyenne vers la face inférieure de la mastoïde. Le long du bord externe du triangle digastrique se trouve un sillon, parfois très accentué, appelé rainure digastrique (6) ; le long de son bord interne, en dehors de la suture mastoïdo-occipitale inférieure, se trouve un autre sillon dans lequel glisse l'artère occipitale (8).

Entre la rainure digastrique et le sillon de l'artère occipitale se trouve comprise la majeure partie (7) du triangle digastrique, dont la forme varie suivant les sujets : quelquefois elle est plate ou lamelleuse, plus souvent elle a la forme d'une crête osseuse ; d'autres fois elle est globuleuse, ovoïde à grosse extrémité tournée en arrière vers la base du triangle digastrique : c'est la lame, crête ou bulle digastrique proprement



FIGURE VII

Même pièce vue par la face supérieure du rocher. Un trait de scie, tangentiel à cette face, a enlevé la couche superficielle, mettant ainsi à découvert l'antre, l'aditus, la caisse, la trompe d'Eustache et les deux premières portions de l'aqueduc de Fallope. La ligne *A* indique la limite antérieure du bloc mastoïdien, la ligne *B* indique sa limite interne. La ligne *B* laisse toute la cavité antrale dans le bloc et longe la paroi interne de l'aditus, laquelle ne saurait être comprise dans le bloc, sans que le labyrinthe (canal demi-circulaire externe) ne fût intéressé. La face supérieure du bloc mastoïdien ainsi décortiquée montre les cellules sous-jacentes à la corticale intra-crânienne tout autour de l'antre. Ces cellules sont plus petites et plus nombreuses que celles qui correspondent à ses autres faces (figs. V, VI, VIII).

1, antre mastoïdien.—2, caisse du tympan.—3, trompe d'Eustache.—4, conduit du muscle du marteau se terminant en crochet : bec de cuiller.—5, trou déchiré antérieur.—6, 1re portion de l'aqueduc de Fallope.—7, son 1er coude.—8, sa 2e portion.—9′, sillon du grand nerf pétreux superficiel.—9, branche antérieure (ampullaire) du canal demi-circulaire supérieur.—10, branche postérieure de ce même canal.—11, golfe de la jugulaire.—12, partie horizontale du sinus latéral.—13, partie descendante du sinus latéral.—14, suture mastoïdo-occipitale postérieure.—15, suture mastoïdo-pariétale.—16, suture pétro-squameuse interne ; un fin pointillé longe ces lignes de suture pour mieux les indiquer.—17, canalis subarcuatus, que j'ai appelé, aussi, canal pétro-mastoïdien (Congrès d'Otologie de Bordeaux, 1904). Ce canal mince et long a été heureusement ouvert dans toute son étendue. D'un côté, il aboutit aux cellules qui sont dans l'angle dièdre, que forment le canal demi-circulaire supérieur et le canal demi-circulaire externe. De l'autre côté, il s'ouvre à la face postérieure du rocher, dans la fossa subarcuata (voir fig. VI, 11).

dite. La forme de lame ou de crête correspond à une structure spongieuse de l'intérieur de cette région. La forme bulleuse correspond à une forme pneumatique (fig. VIII, 14). Plus la bulle est volumineuse, plus la cavité pneumatique correspondante est grande.

Du côté de la cavité crânienne, le triangle digastrique correspond à la moitié inférieure de la zone sinusienne et de la zone cérébelleuse de la face postéro-interne du bloc mastoïdien. La gouttière de l'artère occipitale correspond plus particulièrement à la zone sinusienne. Ces rapports indiquent ainsi quelle peut être l'importance du triangle digastrique. Il forme le plancher de la partie inférieure du sinus latéral, au voisinage du golfe de la jugulaire, et n'est séparé de celui-ci que par l'apophyse jugulaire de l'occipital (11). La présence très fréquente des cavités pneumatiques dans la bulle digastrique, qui parfois s'étendent au delà de cette région, jusque dans l'apophyse jugulaire de l'occipital elle-même, donne à cette région une importance toute particulière, tant au point de vue clinique qu'au point de vue chirurgical. Au point de vue clinique, la thrombose du bulbe de la jugulaire est souvent liée aux suppurations des cellules digastriques ; au point de vue chirurgical, l'ouverture du golfe thrombosé doit être précédée de la résection du triangle digastrique et de l'apophyse jugulaire. Cette résection seule permet de découvrir largement la portion terminale du sinus latéral et le bulbe de la jugulaire, en même temps qu'elle supprime les foyers d'ostéite ou de cellulite digastrique, qui le plus souvent sont cause de l'infection du bulbe.

D'autre part si, au lieu de se propager à l'intérieur du crâne, l'ostéite ou la cellulite digastrique gagne la surface extérieure de l'os, le périoste sous-mastoïdien sera envahi par l'infection. L'abcès sous-mastoïdien (forme Bézold) sera la conséquence des lésions de la région digastrique.

Cependant la périostite et l'abcès sous-mastoïdien peuvent se produire sans qu'il y ait nécrose osseuse du triangle digastrique : il y a, en effet, dans cette région de nombreux orifices vasculaires, qui servent de passage à des vaisseaux intra-osseux, qui unissent la circulation périostique à la circulation intra-osseuse, au même titre que les nombreux trous vasculaires que l'on décrit en arrière de l'épine de Henle sous le nom de zone criblée de Chipault. Ces orifices, déjà mentionnés au pourtour du trou stylo-mastoïdien, sont aussi échelonnés le long de la rainure digastrique et du sillon de l'artère occipitale (fig. XII, 6 et 8). Ils forment ainsi une *double zone criblée digastrique*, dont il convient de ne pas méconnaître l'importance, comme lieu de passage de la suppuration mastoïdienne dans la profondeur du cou. Mais, en considérant ces canalicules vasculaires comme pouvant servir à la propagation des infections mastoïdiennes à la face inférieure de la mastoïde, il y a lieu de se demander si la suppuration de la région sous-mastoïdienne, au cours des otites moyennes, ne peut pas se produire aussi par des voies plus longues encore, et servant de trait d'union direct entre la caisse du tympan et la face inférieure de la mastoïde. Il y a, en effet, des cas,

rares il est vrai, où la périostite sous-mastoïdienne peut se faire au cours d'une otite moyenne en l'absence de toute lésion osseuse ou intra-cellulaire de la région digastrique. La propagation de l'infection peut s'expliquer, alors, par la présence des canaux, tels que l'aqueduc de Fallope et le canal de la corde du tympan, qui s'étendent directement de la paroi postérieure de la caisse à l'extrémité antérieure du triangle digastrique. J'ai observé un cas semblable et je n'ai pu trouver que cette explication, en l'absence de toute lésion de la mastoïde et des cavités intra-mastoïdiennes. Les germes infectieux avaient dû passer de la caisse dans l'aqueduc de Fallope ou dans le conduit que parcourt la corde du tympan et arriver par le trou stylo-mastoïdien à la partie profonde du sommet du triangle digastrique.

Structure de la Région mastoïdienne

Sans vouloir entrer ici dans les détails de structure de la mastoïde, il n'est cependant pas sans intérêt de jeter un rapide coup d'œil sur la constitution du bloc mastoïdien et de voir quels sont les rapports directs des diverses faces de ce bloc avec les parties intra-osseuses.

Si la délimitation du bloc et la description de ses faces peuvent permettre au chirurgien de mieux orienter ses interventions, l'étude des parties intra-mastoïdiennes est aussi de la plus haute importance, car c'est la structure même de l'os qui fournit à la mastoïde son cachet spécial et donne aussi une allure toute particulière aux suppurations de l'oreille.

Le bloc mastoïdien est caractérisé par la présence de cavités pneumatiques dans son intérieur : antre mastoïdien et cellules mastoïdiennes. Ces cavités communiquent entre elles et avec la cavité tympanique. L'antre et les cellules sont creusés au sein du bloc osseux et ont, suivant les individus, des dispositions variables de forme, de volume et d'orientation. Le tissu osseux qui les entoure est aussi variable en structure et en épaisseur.

Le tissu osseux mastoïdien présente trois zones différentes : 1° Une zone superficielle, qui recouvre toutes les faces naturelles du bloc mastoïdien et qui constitue la *corticale extérieure ou périphérique* de l'os ; 2° une zone centrale, qui entoure directement les cavités antrale et cellulaire, que j'ai appelée[1] *corticale centrale* ; 3° une zone intermédiaire à la corticale périphérique et à la corticale centrale, qui constitue le tissu osseux fondamental de la mastoïde.

La corticale périphérique et la corticale centrale sont formées par du tissu compact. La corticale périphérique est beaucoup plus épaisse que la corticale centrale, mais son épaisseur est variable, suivant la zone de la surface osseuse que l'on envisage. Elle varie de 1 à 7 millimètres. Le maximum d'épaisseur correspond, en général, à la zone opératoire,

[1] Mouret, ' Étude sur la structure de la Mastoïde, etc.,' *Annales des maladies de l'oreille*, février 1913.

c'est-à-dire à la zone antrale de la surface extérieure du bloc mastoïdien. Dans certains cas elle est partout d'une minceur extrême et peut alors présenter des déhiscences du genre de celles que j'ai appelées *déhiscences en trou de mite.*

La corticale centrale est, le plus souvent, très mince, parfois réduite à un mince liseré, qui délimite le pourtour des cavités pneumatiques.

La masse osseuse intermédiaire aux deux corticales peut présenter quatre aspects :

(*a*) Chez certains sujets elle est formée par du tissu osseux spongieux à larges mailles.

(*b*) Chez d'autres elle est formée par du tissu très finement spongieux.

(*c*) Chez d'autres, enfin, ce tissu intercortical est formé par du tissu compact, ayant le même aspect et la même densité que le tissu cortical périphérique et central, avec lequel il se continue alors sans aucune ligne de démarcation.

Cette dernière variété d'os présente très peu de cavités pneumatiques ; on l'a improprement appelée mastoïde scléreuse. Mais elle n'est pas, comme on l'a cru, le résultat d'un état pathologique consécutif aux suppurations des cavités pneumatiques : elle constitue simplement une variété spéciale, mais *naturelle*, de l'os mastoïdien.

(*d*) Chez d'autres sujets le tissu osseux intercortical n'a pas une structure régulière partout : en certains endroits il est formé par du tissu spongieux à larges mailles, en d'autres par du tissu finement spongieux, en d'autres encore par du tissu compact.

Cavités pneumatiques, Antre et Cellules

L'antre et les cellules donnent à la région mastoïdienne toute son importance. Ces cavités sont une dépendance de l'oreille moyenne : les maladies de la caisse se propagent aux cavités mastoïdiennes et y évoluent, souvent, de façon telle que l'endomastoïdite aiguë ou chronique, consécutive à la tympanite, réclame, plus que la caisse elle-même, l'intervention du chirurgien.

Antre

De toutes les cavités mastoïdiennes l'antre est la principale. L'antre est constant : mais son volume, et, par suite, ses rapports sont variables. L'antre normal forme une cavité, qui occupe une bonne partie de l'intérieur de bloc mastoïdien. Il est creusé dans cette partie du bloc, qui est comprise, d'une part, entre la paroi postérieure du conduit et la ligne *pariéto-apicale* ; d'autre part, entre la linéa temporalis et une ligne horizontale passant par la paroi postérieure du conduit, à l'union de son tiers supérieur avec ses deux tiers inférieurs. Il est souvent plus petit et cantonné au-dessus d'un plan tangentiel au bord supérieur du méat (fig. I).

On distingue à l'antre six parois : une externe, correspondant à la surface extérieure de la mastoïde, une interne et une supérieure, correspondant toutes deux à la cavité endocrânienne, une inférieure, une

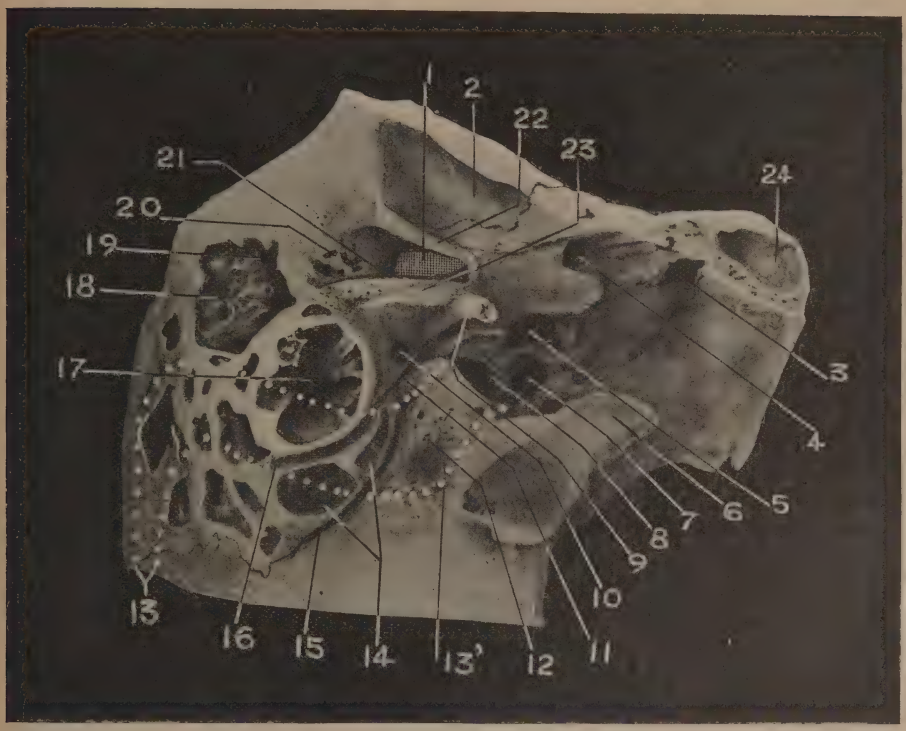


FIGURE VIII

Même pièce que V, VI, VII, vue par sa face inférieure, montrant la face inférieure de la région mastoïdienne. La face externe de la mastoïde est vue de profil. La paroi antérieure du conduit auditif externe a été largement réséquée : en 22 se voit la surface de section, qui l'a séparée de la paroi supérieure du conduit, en 23 celle qui l'a séparée de la paroi inférieure. Cette résection permet de voir la paroi postérieure avec ses cellules limitrophes et montre la large ouverture faite au toit de la caisse. La gouttière du sinus latéral a été projetée à la surface en pointillé blanc.

1, ouverture faite au toit de la caisse (fig. VII).—2, cavité glénoïde du temporal.—3, trou déchiré antérieur.—4, orifice extra-crânien de la partie osseuse de la trompe d'Eustache.—5, orifice d'entrée dans la partie ascendante du canal carotidien.—6, condyle occipital.—7, trou déchiré postérieur.—8, grande cellule siégeant au-dessus du golfe de la jugulaire. Cette cellule a été ouverte par le même procédé que les autres. Elle correspond à la cavité qui est dans la profondeur de la cellule, qui surmonte le golfe de la jugulaire, dans la fig. VI.—9, apophyse styloïde.—10, trou stylo-mastoïdien.—11, suture de la mastoïde avec l'apophyse jugulaire de l'occipital.—12, apophyse jugulaire de l'occipital.—13, gouttière du sinus latéral, projetée en pointillé blanc à la surface extérieure de la mastoïde.—13', partie terminale du sinus latéral, projetée à la face inférieure de l'apophyse jugulaire de l'occipital.—14, deux grandes cellules du triangle digastrique.—15, suture mastoïdo-occipitale inférieure.—16, rainure digastrique.—17, grandes cellules de l'apex mastoïdien.—18, région des cellules sous-antrales.—19, région de l'antre (se reporter pour ces deux régions à la fig. V).—20, cellules limitrophes de: 21, paroi postérieure du conduit auditif externe.—22, surface de section entre la paroi supérieure et la paroi antérieure du conduit.—23, surface de section entre la paroi inférieure et la paroi antérieure du conduit.—24, sinus sphénoïdal.

antérieure et une postérieure. Nous étudierons les rapports particuliers à chacune de ces parois. Mais, avant cela, il convient de jeter un coup d'œil sur le développement de l'antre.

Développement de l'antre.—L'antre se développe de bonne heure. Il commence à apparaître chez le fœtus de quatre à cinq mois. Il se forme aux dépens de la cavité épitympanique, c'est-à-dire, aux dépens de la partie de la caisse qui est au-dessus des osselets. La cavité épitympanique s'allonge en arrière, au-dessus du deuxième coude du canal de Fallope, et en dehors du canal demi-circulaire externe, dans l'espace qui sépare l'os pétreux de l'os écailleux. Dans ce premier stade l'antre se présente sous la forme d'un cône creux, dont le sommet est dirigé en arrière et dont la base, tournée en avant, se continue avec la cavité épitympanique : le grand axe de l'antre a, alors, une direction antéro-postérieure, parallèle à la paroi supérieure du rocher, et se trouve sur le prolongement du grand axe de la cavité épitympanique. La partie de sa cavité, qui correspond à l'aqueduc de Fallope et au canal demi-circulaire externe, constitue l'aditus ad antrum, la partie, qui est en arrière de celui-ci, constitue l'antre proprement dit.

Chez certains sujets, le développement de l'antre s'arrête à ce stade et ne s'agrandit pas davantage. Dans ce cas, l'antre de l'adulte se présente, alors, comme un simple diverticule de la cavité épitympanique, de forme conique, n'ayant guère plus (antre et aditus réunis) de 6 à 8 millimètres de longueur. Le plancher de la cavité adito-antrale ne dépasse pas un plan antéro-postérieur passant par la lèvre supérieure du méat auditif, au-dessus de l'épine de Henle. On appelle cette disposition de l'antre chez l'adulte : *antre en position fœtale.* On le recontre plus particulièrement dans les mastoïdes compactes et petites, il est très difficile à trouver par la trépanation rétro-méatique classique, les chirurgiens avertis utilisent alors le procédé de Stacke, qui consiste à abattre d'abord le mur de la logette des osselets et la paroi externe de l'aditus, allant ainsi, à rebours, de la caisse vers l'antre. Mais on peut la rencontrer aussi dans les mastoïdes pneumatiques, bien que plus rarement. Cependant, chez la plupart des sujets, le cône antral des premiers stades de développement s'allonge et, par sa paroi inférieure, descend en arrière du coude de l'aqueduc de Fallope et en arrière du $\frac{1}{3}$ supérieur de la branche postérieure de l'anneau tympanal. Cela se voit clairement dès les septième ou huitième mois de la vie fœtale sur un rocher dont on sépare l'os écailleux de l'os pétreux (fig. III in loc. cit. *Annales des maladies de l'oreille,* février 1913). De sorte que l'antre a, dès la fin de la vie fœtale, un volume plus grand et des rapports directs avec l'arc postérieur de l'os tympanal (futur conduit auditif externe osseux), à peu près identiques à ceux qu'il a chez l'adulte. Toutefois, si l'antre descend, déjà chez le fœtus, en arrière de l'extrémité supérieure de l'arc postérieur de l'os tympanal, qui représente le futur conduit auditif externe osseux, il convient de remarquer que cela ne permet pas de dire que l'antre se trouve, comme chez l'adulte, en arrière de l'ébauche de ce conduit.

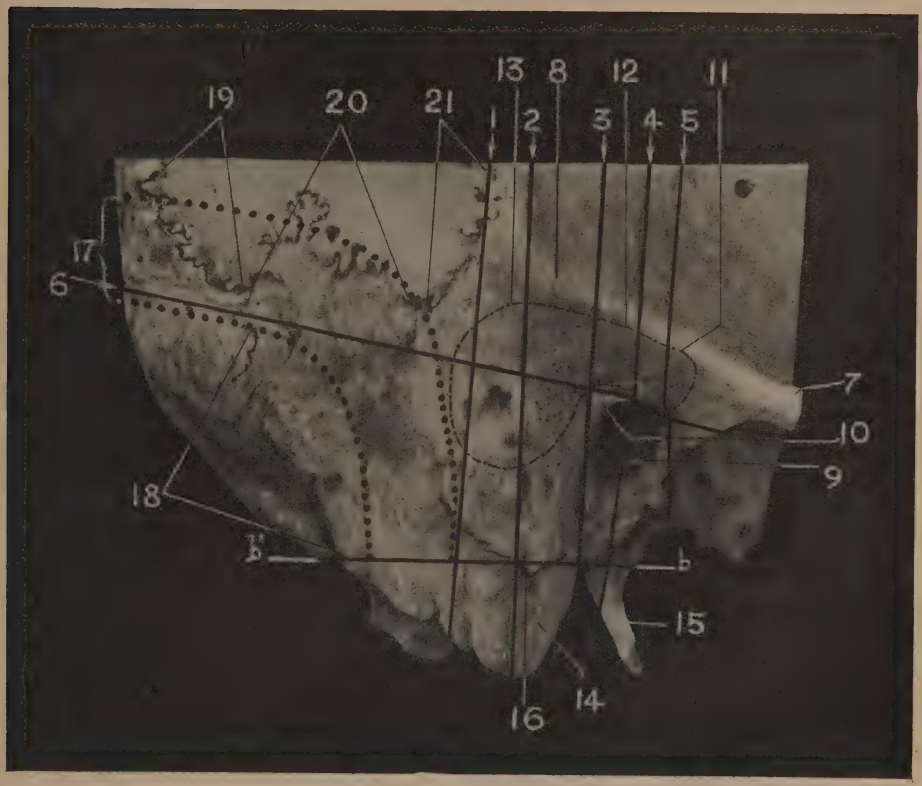


FIGURE IX

Face externe d'un temporal extrêmement pneumatique, ainsi que j'ai pu m'en rendre compte par les diverses coupes, que j'en ai faites suivant les lignes de section 1, 2, 3, 4, 5, 6. La coupe correspondant à la ligne 6 est représentée par la fig. XVIII. L'antre mastoïdien est très développé. J'ai représenté en projection à la surface extérieure de l'os la cavité épitympanique et la cavité antrale, ainsi que la gouttière du sinus latéral.

b–b', ligne horizontale passant sous la lèvre inférieure du méat auditif externe. Cette ligne marque la base de l'apex mastoïdien proprement dit.—7, apophyse zygomatique.—8, linéa temporalis.—9, conduit auditif externe.—10, spina-supra-méatum.—11, cavité épitympanique (attique), projetée à la surface de l'os, au-dessus du conduit auditif externe.—12, cavité de l'aditus, projetée à la surface de l'os.—13, cavité de l'antre, projetée à la surface de l'os.—14, apex mastoïdien.—15, apophyse styloïde.—16, suture pétro-squameuse externe, marquée, ici, principalement à la base de la région de l'apex.—17, projection du trajet du sinus latéral en pointillé noir.—18, suture mastoïdo-occipitale externe.—19, suture occipito-pariétale.—20, suture mastoïdo-pariétale.—21, suture squamo-pariétale.

En effet, non seulement le conduit est à peine ébauché, mais le rocher lui-même est loin d'avoir sa forme et son orientation définitives et la cavité tympano-antrale a, par suite de cela, une orientation différente de celle qu'il présente chez l'adulte. Ce n'est qu'au cours du développement de la base du crâne, pendant les premières années de la vie, que les rapports définitifs de l'antre et de la caisse avec le conduit auditif externe et avec les autres parties du temporal s'établissent peu à peu.

Il est classique de dire que chez le nouveau-né l'antre est *au-dessus* du conduit auditif externe, que chez le jeune enfant il *est au-dessus et en arrière* de l'angle supéro-postérieur du conduit, que chez l'adulte il *est en arrière* de la moitié supérieure de la paroi postérieure du conduit.

Cela est vrai. Mais il ne faut point croire que cela tienne uniquement à l'agrandissement de la cavité antrale, car si l'antre peut parfois continuer à s'agrandir après la naissance en même temps que la base du rocher, son volume est souvent presque définitif à la naissance. Ce changement de rapports tient principalement aux modifications des parties osseuses qui l'entourent.

Chez l'adulte la mastoïde et le conduit auditif externe sont bien développés. La caisse et la membrane du tympan sont un peu inclinées en dehors et en avant, mais cette inclinaison est assez peu importante pour qu'on n'en tienne pas compte dans la dénomination de leurs parois et de leurs bords. C'est ainsi que la paroi de la caisse, qui est formée par la membrane du tympan et par le cadre tympanal, est dite paroi externe, bien qu'en réalité cette paroi soit aussi inclinée en dehors et en avant. De même, la face du rocher, qui correspond à la fosse cérébrale moyenne est dite supérieure, bien qu'elle soit aussi un peu inclinée en avant. La caisse elle-même présente une inclinaison semblable.

Chez le fœtus et chez le nouveau-né cette double inclinaison est, au contraire, très accentuée. L'apophyse mastoïde n'existe pas encore, la base du rocher est petite et regarde en bas, au lieu de regarder en dehors. L'anneau tympanal et la membrane du tympan, qui sont sur cette partie du rocher, paraissent ainsi être situées, de chaque côté, à la face inférieure de la base du crâne et regardent en bas. L'anneau tympanal est couché presque horizontalement, l'extrémité de ses deux branches regardant en dehors et non en haut. La face supérieure du rocher est, de même, fortement inclinée en avant et est bien plus *supéro-antérieure* que simplement *supérieure*.

La caisse du tympan et la cavité épitympanique subissent l'influence de la forme et de l'orientation du rocher. La caisse est couchée presque horizontalement comme la membrane du tympan et comme l'os tympanal, au-dessus d'eux et au-dessous de l'oreille interne. La paroi supérieure est inclinée en dehors et le plancher est tourné en dedans.

La cavité épitympanique subit, en outre, l'influence de l'orientation de la paroi supérieure du rocher, qui en forme la voûte. Comme elle, cette cavité est très inclinée en avant, de sorte que son axe antéro-postérieur est ascendant d'avant en arrière et de bas en haut. L'antre

se trouve, ainsi, placé en arrière de la cavité épitympanique, mais dans un plan un peu plus élevé.

En conséquence, la position couchée de la caisse au-dessus du conduit fait que la cavité épitympanique est *au-dessus* du mur de la logette et que l'antre lui-même est dans un plan plus élevé que le conduit auditif.

En outre de cela, il faut encore tenir compte de l'orientation générale du grand axe de l'antre, de la caisse et de la trompe d'Eustache, qui n'est pas simplement antéro-postérieur et oblique de bas en haut et d'arrière en avant ; cet axe est aussi très oblique de dedans en dehors, du pharynx vers l'extérieur. Cette obliquité fait que l'antre est non seulement en arrière et un peu au-dessus de la cavité épitympanique, mais qu'il est aussi plus extérieur qu'elle : si la cavité épitympanique est couchée au-dessus du mur de la logette, l'antre est, plus en dehors, couché sur un plan passant au-dessus du conduit, lequel est encore très court, et le déborde même en dehors. Tout cela est bien démontré par une série de coupes d'un temporal d'enfant de 1½ mois, telles qu'elles sont représentées dans la figure XXII. Ces coupes sont faites dans le sens vertical et antéro-postérieur. Elles montrent clairement ce que je viens de dire. La coupe la plus superficielle (*a*) n'ouvre que les cellules de la paroi externe de l'antre. La deuxième coupe (*b*) ouvre l'antre. Le conduit auditif externe n'est touché que dans la troisième coupe (*c*). L'étude des coupes, qui sont représentées dans cette figure, permet de dire :

1° La face dite *supérieure* du rocher est plutôt *antérieure* ; le bord, qui sépare cette face de la face postérieure, lequel est d'ailleurs large et arrondi, mérite seul le qualificatif de supérieur (figs. *c*, *d*, *e*, *f*, *g*).

2° La caisse est couchée au-dessus du conduit auditif et au-dessous de l'oreille interne (figs. *d*, *e*).

3° Le mur de la logette est, lui aussi, couché presque horizontalement et la cavité épitympanique se trouve au-dessus de lui (fig. *c*).

4° L'antre et la cavité épitympanique sont inclinés de bas en haut et d'avant en arrière, comme la face supérieure du rocher, qui forme leur voûte, et l'antre est plus élevé que la cavité épitympanique (fig. *c*).

5° L'antre est plus près de la surface extérieure de l'os que la cavité épitympanique et déborde, en dehors, la paroi supérieure du conduit auditif externe, qui est encore très court. Dans la coupe *b*, l'antre seul est intéressé.

6° L'antre s'allonge en bas, du côté de sa paroi inférieure, derrière la région, qui sera plus tard occupée par le conduit auditif, lorsque l'anneau tympanal se sera élargi (fig. *b*).

7° La coupe *a* montre enfin que la paroi externe de l'antre est recouverte par de nombreuses cellules et que celles-ci, comme l'antre lui-même, sont entourées par une couche osseuse, dense, formant une corticale centrale, périantrale et péricellulaire (voir *Études sur la structure de la Mastoïde*, etc., cité plus haut).

L'antre n'a donc pas tout à fait la même orientation ni les mêmes rapports chez le fœtus, chez le nouveau-né et chez l'enfant. Il subit un déplacement notable, qui est dû non à sa propre évolution, mais à l'évolution même de l'os temporal. A mesure que la base du crâne grandit, le rocher s'allonge et s'élargit ; la région mastoïdienne s'épaissit et le conduit auditif externe s'allonge. Le résultat de cela est double : 1° l'axe vertical de la caisse, de l'antre, de la membrane du tympan se redresse : ces organes ne restent plus couchés au-dessus du conduit auditif externe et paraissent s'enfoncer dans l'os qui s'élargit ; 2° l'axe antéro-postérieur de la cavité épitympanique, qui est parallèle à la face supéro-antérieure du rocher, se redresse aussi et tend, comme celle-ci, à prendre une position horizontale : l'antre ne paraît plus situé au-dessus de la cavité épitympanique, ni au-dessus de la paroi supérieure du conduit auditif, il se montre, au contraire, en arrière de la cavité épitympanique, au niveau de l'angle postéro-supérieur du conduit et même en arrière du tiers supérieur de la paroi postérieure, si l'antre est suffisamment grand.

Les rapports intrinsèques de l'antre avec la caisse ne changent donc pas, mais les rapports avec les parties qui l'entourent se modifient. Cela a une grande importance pour l'orientation qu'il faut donner aux coupes destinées à l'étude du développement de l'antre : lorsqu'on veut voir les rapports de l'antre avec la cavité épitympanique, ces coupes doivent être faites parallèlement à la face supérieure du rocher et non parallèlement à un plan horizontal. Cela a aussi une grande importance au point de vue opératoire, car, chez le nouveau-né, l'antre doit être recherché non en arrière du méat auditif mais au-dessus de son angle postéro-supérieur.

Chez l'adulte, l'antre normal occupe la profondeur du bloc mastoïdien, dans la zone qui, à la surface extérieure de l'os, est limitée, en avant, par le bord postérieur du méat auditif, en arrière, par la ligne pariéto-apicale, en haut, par la linéa temporalis, en bas, par une ligne horizontale qui passe par le bord postérieur du méat à l'union de son $\frac{1}{3}$ supérieur avec ses $\frac{2}{3}$ inférieurs. Ses dimensions sont ordinairement les mêmes que chez le fœtus à terme. Le diamètre antéro-postérieur = 6 à 7 mm. ; le diamètre vertical = 6 à 8 mm. ; le diamètre transversal = 5 mm. Mais il peut être beaucoup plus grand et s'allonger, soit dans le sens vertical, soit dans le sens antéro-postérieur, et avoir plus de deux centimètres suivant sa plus grande longueur. Mais ce sont là des cas exceptionnels. D'une façon générale un antre volumineux se trouve dans les grandes mastoïdes très pneumatiques ; un antre petit, de forme fœtale, se trouve plus spécialement dans les mastoïdes petites, dures et compactes. Mais cela n'est pas absolu et certaines mastoïdes très pneumatiques peuvent n'avoir qu'un antre tout petit. L'antre présente six parois, dont les rapports sont importants à connaître.

Paroi externe. — La paroi externe est pour le chirurgien de la plus haute importance, car c'est sur elle que porte la trépanation mastoïdienne.

Au point de vue embryologique la paroi externe de l'antre est uniquement formée par l'aile squamo-mastoïdienne, que l'os écailleux envoie à la surface de la mastoïde : l'orientation de cette paroi par rapport à cette surface, l'épaisseur et la structure du mur osseux, qui la sépare de l'extérieur, enfin l'étendue des rapports, qui existent entre elle et la surface extérieure, sont des points qu'il convient de bien connaître. Cette étude doit être faite à l'aide de coupes horizontales et de coupes vertico-transversales. Ces deux sortes de coupes permettent de bien voir l'orientation, l'épaisseur et la structure de la paroi externe de l'antre.

Soit une coupe horizontale, comme celle qui est représentée dans la figure XIV. Si sur cette coupe nous menons deux lignes perpendiculaires l'une à l'autre, l'une *B* longeant la paroi postérieure du conduit auditif externe, l'autre *A* passant contre les canaux demi-circulaires, ces deux lignes nous indiquent, respectivement, la limite antérieure (tympanale) et la limite interne (pétreuse), que j'ai déjà assignées au bloc mastoïdien. Cette figure montre clairement quelle est l'orientation de l'axe antéro-postérieur de l'antre, et quelle est aussi l'orientation de sa paroi externe par rapport à la face antérieure (conduit auditif) et à la face externe de la mastoïde. Elle montre, en même temps, l'orientation de sa paroi interne et de sa paroi postérieure par rapport à la cavité crânienne.

L'axe antéro-postérieur de l'antre est sur le prolongement de l'axe antéro-postérieur de la caisse (22). Cet axe, prolongé en arrière, aboutit au milieu de la gouttière sinusienne, lorsque celle-ci est en position normale. Il peut, aussi, passer en dehors de la gouttière, lorsque celle-ci est très peu creusée. L'antre de cette pièce a les dimensions suivantes : hauteur = 13 mm. ; longueur = $9\frac{1}{2}$ mm. ; largeur = $5\frac{1}{2}$ mm. La coupe passe par l'extrémité inférieure de l'épine de Henle. Nous avons affaire à un antre de volume et de situation ordinaires.

Orientation de la Face externe de l'Antre. — La paroi externe de l'antre regarde, tout à la fois, la face externe du bloc mastoïdien et sa face antérieure (paroi postérieure du conduit auditif). Une ligne *D*, perpendiculaire au milieu de la face externe de l'antre, passe par l'angle, que forment, en se rencontrant, la face externe et la face antérieure du bloc mastoïdien, c'est-à-dire au niveau du point, qui est occupé par la spina-supra-méatum. Ici, sur la coupe, la ligne *D* passe par l'extrémité inférieure de la spina.

Épaisseur de la Paroi externe de l'Antre. — Sur cette pièce l'épaisseur de la paroi externe, c'est-à-dire la distance, qui sépare l'antre de la surface extérieure de l'os, est aussi de dimensions moyennes. Mesurée au niveau de la ligne *D*, qui, partant de la spina, tombe perpendiculairement sur le milieu de la face externe de l'antre, l'épaisseur est de 6 millimètres. Si on la mesurait au niveau du champ d'attaque classique, cette distance serait beaucoup plus grande. Le carré d'attaque classique mesurant 1 cm. de côté se trouve à la surface de la mastoïde à $\frac{1}{2}$ centimètre en arrière de la spina, et son milieu correspond à la flèche qui est marquée sur la figure. Une ligne, partant du milieu de ce carré d'attaque

et allant vers le milieu de la face externe de l'antre, est sensiblement parallèle à la paroi postérieure du conduit auditif : sa longueur, sur cette même pièce, mesure 1½ cm. Dans la trépanation classique, la paroi externe de cet antre serait donc considérée comme ayant 1½ cm. d'épaisseur. Mais remarquons bien, que dans cette dernière façon de mesurer la paroi externe de l'antre, cette mesure est fausse, puisqu'elle est prise très obliquement par rapport à l'antre. Pour le moment je tiens seulement à faire remarquer ce fait. J'y reviendrai dans le paragraphe destiné à indiquer quel doit être rationnellement le véritable lieu d'élection de la trépanation de l'antre.

La lame squamo-mastoïdienne, qui forme la paroi externe de l'antre, est très mince au début, mais s'épaissit avec l'âge. Chez le nourrisson, elle peut n'avoir encore que quelques millimètres d'épaisseur et l'on peut facilement avec une simple curette effondrer la paroi externe de l'antre. En grandissant l'os s'épaissit et la lame osseuse acquiert, chez l'adulte, des dimensions qui sont fréquemment de 1½ cm., et même de 2 cm. Mais je ferai encore remarquer que ces dimensions, qu'on trouve données dans tous les traités classiques, sont basées sur la trépanation dite aussi classique : or nous venons de voir que, dans ce cas, l'épaisseur indiquée mesure la paroi externe suivant une ligne parallèle à la paroi postérieure du conduit, c'est-à-dire oblique par rapport à la face externe de l'antre et non suivant une ligne perpendiculaire à la surface antrale, comme cela doit être. L'épaisseur réelle doit, en effet, être prise suivant une ligne, qui tombe perpendiculairement à la face externe de l'antre, c'est-à-dire au niveau de la spina. Or l'épaisseur ainsi prise est en réalité beaucoup moindre que ne l'indiquent les classiques, elle peut avoir 1 centimètre, mais, en moyenne, elle n'a que 6 à 7 millimètres.

Structure. — La structure de la paroi externe de l'antre est variable, suivant les sujets et suivant leur âge. Cette paroi comprend deux parties : une partie, qui regarde en dehors, derrière le méat auditif, et une partie, qui regarde le conduit auditif. Au point de vue embryologique elle est constituée par l'aile squamo-mastoïdienne, que l'os écailleux envoie à la surface de la mastoïde. Du côté de la paroi postérieure du conduit, cette lame osseuse est doublée de l'arc postérieur de l'os tympanal. L'étude de l'antre, chez le fœtus, montre que c'est à la face antrale de l'aile squamo-mastoïdienne que se fait toujours l'apparition des premières cellules péri-antrales. La paroi externe de l'antre est donc *toujours* pourvue de cellules, qui s'ouvrent dans la cavité antrale. Mais ces cellules peuvent pneumatiser complètement toute l'épaisseur de l'os ou bien ne tapisser que sa surface antrale. En effet, pendant que l'os s'épaissit, les cellules *fœtales* de la face externe de l'antre augmentent, en général, de volume et creusent complètement l'os jusqu'au niveau de la corticale périphérique : la paroi est alors complètement pneumatique, c'est le cas dans la figure XIV. D'autrefois, les cellules fœtales s'arrêtent dans leur développement, restent toutes petites et cantonnées



FIGURE X

Crâne vu par sa face externe, mais légèrement incliné de bas en haut, ce qui permet de voir, en partie, la région digastrique. L'apophyse zygomatique a été réséquée pour bien mettre à nu le fond de la fosse temporale, où est bien marquée la ' zone criblée de la fosse temporale '.

1, zone criblée de la fosse temporale.—2, cavite glénoïde.—3, zone criblée susméatique.—4, suture squamo-tympanale ou de Glaser.—5, fossette rétro-spinale, avec zone criblée rétro-méatique de Chipault.—6, spina-supra-méatum, elle a ici la forme d'une crête.—7, fissure pétro-squameuse externe bien marquée dans une partie de son trajet descendant.—8, partie postérieure de la rainure digastrique montrant de nombreux pertuis vasculaires.—9, surface digastrique de forme lamelleuse.—10, sillon de l'artère occipitale présentant de nombreux pertuis vasculaires.—11, triple orifice pour trois branches de la grande veine mastoïdienne. Ces orifices sont, sur cette pièce, très bas, à la limite de la face externe de la mastoïde avec sa face inférieure (digastrique).—12 à 12', surface rugueuse sur laquelle s'insèrent les muscles mastoïdiens.—13, apophyse styloïde.—14, angle postérieur de l'os malaire, isolé de l'apophyse zygomatique.—15, aile externe de l'apophyse ptérygoïde.—16, suture mastoïdo-occipitale.—17, suture pariéto-occipitale.—18, suture mastoïdo-pariétale.—19, angle pariéto-mastoïdo-occipital.—20, suture squamo-pariétale.—21, angle squamo-mastoïdo-pariétal.—22, partie horizontale de la gouttière du sinus latéral, projetée en pointillé sur la surface externe du crâne.—23, sa portion descendante.—24, linéa temporalis.—25, surface de section de l'apophyse zygomatique.

à la face antrale de l'aile squamo-mastoïdienne. L'os, devenu plus épais, paraît alors dépourvu de cavités pneumatiques et sa propre constitution varie encore suivant les sujets : il peut être formé par du tissu spongieux à larges mailles, ou bien par du tissu spongieux à très fines mailles, ou bien encore être dense, compact, éburné. Dans ce cas, la paroi externe de l'antre est dite, improprement, à mon avis, scléreuse (structure de la mastoïde : loc. cit.). Cette variabilité de structure rend la trépanation antrale délicate et parfois difficile, surtout lorsqu'on la fait au lieu dit classique et qu'on est obligé de creuser, à travers un os éburné, jusqu'à 2 cm. de profondeur pour arriver jusqu'à l'antre.

Étendue des Rapports de la Face externe de l'Antre avec la Surface extérieure de l'os. — La face externe de l'antre regarde en dehors et en avant vers le bord postérieur du méat auditif. Les rapports de l'antre avec la surface extérieure de la mastoïde sont intéressants à connaître au point de vue chirurgical. L'étendue de ces rapports est variable suivant le volume même de l'antre. Plus un antre est grand, plus il s'étend dans la profondeur du bloc mastoïdien et plus étendus, aussi, sont ses rapports avec la surface de l'os. J'ai dit plus haut que certains antres sont très grands dans le sens antéro-postérieur, que d'autres le sont dans le sens vertical : le maximum que j'ai observé dans l'une ou l'autre de ces directions est de 2 cm. Mais, en général, les dimensions ordinaires ne dépassent pas un centimètre ; souvent même elles sont inférieures à ce chiffre. Lorsque l'antre est très volumineux, comme dans les figures IX et XVIII, les rapports de sa face externe avec la surface extérieure de la mastoïde sont tellement étendus que la trépanation est très facile. Aussi n'est-ce pas des cas de ce genre qu'il convient de choisir pour une démonstration. Il faut prendre les cas où l'antre a des dimensions normales, ou, mieux encore, les cas où l'antre est petit, c'est-à-dire, ceux où les rapports de l'antre avec la surface extérieure sont très réduits, afin de localiser le plus exactement possible l'endroit où doit se faire la trépanation. Prenons un cas ordinaire : antre de dimensions normales ayant environ 8 à 10 mm. carrés de surface. Suivant le schéma classique, la limite supérieure de la face externe de l'antre ne dépasse pas, en haut, la linéa temporalis ; en bas, la limite inférieure correspond à un plan horizontal passant à l'union du $\frac{1}{3}$ supérieur avec le $\frac{1}{3}$ moyen de la paroi postérieure du méat auditif. Nous venons de voir que le milieu de la face externe regarde la spina-supra-méatum. Il n'y a donc que la moitié postérieure de cette face qui regarde la face externe du bloc mastoïdien. Il semble, ainsi, que pour tomber sur la paroi externe de l'antre le plus directement possible, on doive passer tout à la fois par le conduit et par la mastoïde, c'est-à-dire à travers la partie supérieure du bord postérieur du méat. Or ce n'est pas ainsi que l'on fait d'après l'ordonnance classique. On respecte le conduit, on trépane à $\frac{1}{2}$ cm. en arrière de son $\frac{1}{3}$ supérieur. Une trépanation ainsi faite et menée *perpendiculairement* à la surface de l'os ne conduirait pas sur l'antre. Elle passerait en arrière de lui et irait vers la cavité



FIGURE XI

Temporal de la collection du professeur Brühl, de Berlin. Scissure pétro-squameuse externe bien marquée.

1, conduit auditif externe.—2, apophyse styloïde.—3, cavité glénoïde.—4, apophyse zygomatique.—5, racine tranverse de cette apophyse.—6, sa racine horizontale.—7, linéa temporalis.—8, fossette rétrospinale (la spina-supra-méatum manque).—9, zone criblée rétroméatique.—10, surface rugueuse, sur laquelle prennent insertion les muscles mastoïdiens.—11, suture pétro-squameuse externe très bien marquée sur toute la ligne pariéto-apicale, depuis l'angle squamo-mastoïdo-pariétal (12), jusqu'au sommet de l'apex.—13, zone criblée sus-méatique.

crânienne. Pour éviter cela, il est recommandé de creuser l'os *parallèlement* à la paroi postérieure du conduit : de cette façon on prend le chemin *le plus long* et, très souvent, on ne trouve l'antre qu'à une profondeur de 1½ cm. à 2 cm.

D'autre part, la voûte de l'antre correspond au plancher de la fosse cérébrale moyenne, dont elle n'est souvent séparée que par une épaisseur d'os très minime. Pour éviter de pénétrer dans la fosse temporale de la cavité crânienne, on prend comme ligne de repère la linéa temporalis, qui est supposée indiquer la limite supérieure, au delà de laquelle il ne faut pas trépaner, au risque d'ouvrir la cavité crânienne. Dans beaucoup de cas ce repère supérieur peut être suffisant. En s'orientant ainsi, à la fois sur la linéa temporalis et sur la paroi postérieure du conduit, on mène souvent à bien la trépanation antrale, le but du chirurgien étant d'ouvrir l'antre, sans ouvrir la cavité crânienne ni du côté de la ligne temporale, ni du côté du sinus latéral, et sans léser, aussi, le nerf facial. On n'a que l'ennui d'être obligé de creuser l'os profondément avant d'arriver à l'antre. Mais il faut savoir que maintes fois cette opération est plus délicate :

(*a*) La linéa temporalis ne correspond pas toujours au plancher crânien, elle peut occuper un plan situé soit plus haut, soit plus bas que lui.

(*b*) L'antre n'a pas toujours le même volume et n'occupe pas toujours la même situation par rapport à la linéa temporalis.

(*c*) Le sinus latéral et la cavité crânienne peuvent présenter certaines anomalies de disposition et le ciseau de l'opérateur a grandes chances de les ouvrir, s'il suit très simplement les données classiques.

(*A*) La linéa temporalis ne constitue pas un point de repère précis et constant, qui puisse servir à fixer la situation du plancher de la cavité crânienne par rapport à la surface externe de la mastoïde. Elle peut coïncider exactement avec la situation du plancher. C'est le cas dans la pièce représentée dans la figure IX. Elle peut aussi être située à plus de 8 mm. au-dessus de lui (fig. I). Mais, encore, le plancher crânien, procident en bas et en dehors, peut descendre au-dessous de la linéa temporalis, comme c'était le cas dans la pièce, qui est représentée dans la figure XIII.

(*B*) L'antre, même s'il a un volume normal, peut ne pas être toujours placé dans la région qui correspond au ⅓ supérieur de la paroi postérieure du conduit auditif. Dans certains cas, en effet, le plancher de la cavité crânienne est comme surélevé. Il existe entre ce plancher et un plan horizontal passant par la paroi supérieure du conduit auditif externe, une épaisseur d'os, telle que la cavité antrale, de moyen volume, puisse s'y loger sur le prolongement direct de la cavité épitympanique, de laquelle il émane, sans être obligé de se déployer en bas, derrière le conduit. L'antre occupe alors une position élevée au-dessus de l'angle supéro-postérieur du conduit auditif. C'est la disposition, qui se présente dans la pièce qui est représentée dans la figure I, où le bord inférieur de l'antre arrive à peine *au-dessus* de l'épine de Henle, tandis que son

bord supérieur remonte à 7 mm. au-dessus d'un plan horizontal passant par le bord supérieur du méat auditif. Dans la figure III on voit, en coupe, les rapports du plancher de cet antre (11) avec la spina-supra-méatum (5). Dans ce cas, l'antre se trouve presque tout entier au-dessus du champ de la trépanation classique.

(*C*) L'antre n'a pas un volume normal et s'est arrêté dans son développement. Il est alors représenté par une petite cavité, située en arrière et sur le prolongement de la cavité épitympanique ; son plancher ne descend pas non plus au-dessous d'un plan passant par le bord supérieur du méat auditif. Il se trouve encore au-dessus du champ classique de la trépanation. Dans ce cas, comme dans le précédent, en suivant le procédé classique, l'opérateur court le risque de passer au-dessous de l'antre et d'aller blesser le facial, comme cela est arrivé à Schwartze (cas rapporté par Duplay, ' De la Trépanation de l'apophyse mastoïde,' in *Archives générales de Médecine*, 1888).

(*D*) Lorsque l'antre est petit, la cavité crânienne peut être procidente en bas et descendre en dehors de sa face externe, *au-dessous du plan* dans lequel se dessine la linéa temporalis à la surface de l'os. Si la gouge se rapproche trop de celle-ci, on ouvre la cavité crânienne.

(*E*) Lorsque l'antre est petit, le sinus latéral peut encore, lui aussi, être très procident en avant (fig. XV) et arriver au contact de la paroi postérieure du conduit auditif. Dans ce cas, la gouge, correctement appliquée dans la zone classique, pénètre sûrement dans le sinus et n'ouvre pas l'antre. Il ne faut pas croire que ce soient là des cas très exceptionnels. On les rencontre, il est vrai, moins souvent dans les trépanations faites pour mastoïdite aiguë, mais on les trouve souvent dans la cure radicale des otorrhées chroniques. J'en ai donné les raisons ailleurs (*Structure de la Mastoïde*, etc., loc. cit.).

La plupart de ces faits sont d'ailleurs bien connus et c'est pour cela qu'on fait encore les recommandations suivantes :

1° Sur le bord postérieur du carré d'attaque de l'os, donner le coup de ciseau tangentiellement à sa surface, afin d'éviter de blesser le sinus, au cas où il serait procident.

2° Obliquer la pointe du ciseau un peu en haut, lorsqu'on est arrivé à 1 cm. de profondeur sans trouver l'antre, ce faisant on fuit le nerf facial et l'on se rapproche de l'antre, s'il est haut placé.

Tout cela est très bien pour qui a une grande habitude des opérations sur la mastoïde, mais il faut bien reconnaître que pour ceux qui ne sont pas complètement familiers avec les variétés de disposition que peut présenter l'antre mastoïdien, sa recherche est difficile et, parfois, l'opérateur n'osant aller trop loin, s'arrête et cache son incompétence en disant que l'antre n'existe pas.

Comme tout le monde, je me suis trouvé bien des fois aux prises avec de pareilles difficultés. Aussi me suis-je demandé, s'il n'y aurait pas avantage à modifier le champ d'attaque de la trépanation antrale. Pour cela, je me suis basé sur l'étude du développement de l'antre à

l'extrémité postérieure de la cavité épitympanique et sur l'étude des coupes horizontales de la mastoïde, qui nous montrent (fig. XIV) que la face externe de l'antre regarde, tout à la fois, la paroi postérieure du conduit, l'angle postéro-supérieur du méat, et la partie rétro-méatique de la face extérieure de la mastoïde. Je me suis aussi basé sur l'étude des coupes vertico-transversales (fig. III, et des coupes horizontales, fig. XV) qui montrent que l'extrémité supérieure de la spina-supra-méatum et l'angle supéro-postérieur du méat sont sur un même plan horizontal que le plancher de l'aditus et de l'antre, lorsque celui-ci, étant rudimentaire, ne forme qu'un petit diverticule à l'extrémité postérieure de la cavité épitympanique. Depuis plusieurs années, je ne trépane plus la mastoïde à un demi-centimètre en arrière du bord postérieur du méat, j'ai porté plus haut et plus en avant le champ d'attaque de la trépanation : je fais une *trépanation transspino-méatique.* Avec une gouge de 8 mm. de large je trace un rond d'attaque au niveau de l'angle supéro-postérieur du méat. Le bord supérieur de la ligne d'attaque remonte au-dessus de cet angle d'environ 3 mm. ; le bord postérieur passe en arrière de l'épine de Henle ; le bord inférieur passe au-dessous d'elle et le bord antérieur en avant d'elle. L'épine est donc le centre du rond d'attaque : mais celui-ci remonte à 3 mm., environ, au-dessus d'elle.

L'ensemble du puits de trépanation transspino-méatique est orienté différemment que celui de la trépanation classique. Tandis que celui-ci est creusé parallèlement à la paroi postérieure du conduit et en arrière d'elle, le puits transspino-méatique passe à travers la partie supéro-externe de cette paroi et son axe, partant de la spina, forme avec la paroi postérieure un angle de 125°, environ, ouvert vers le conduit, comme le montrent les lignes *B* et *D* de la figure XIV.

Supposons un antre de volume moyen (même figure). Dans ce cas, la trépanation *transspino-méatique* tombe au milieu de la face externe de l'antre. En suivant le procédé classique la trépanation serait parallèle au conduit et tomberait à la partie la plus externe de cette même face. Le but poursuivi serait donc, aussi, atteint avec le procédé classique, mais il faudrait creuser à une distance deux fois plus grande.

Supposons au contraire un antre tout petit, représentant un diverticule, de 3 ou 4 mm. seulement, situé à l'extrémité de l'aditus et dont le plancher ne descend pas plus bas que le plancher de ce dernier. Cette disposition se rencontre assez souvent dans les mastoïdes compactes. Si l'on suit le procédé classique, la trépanation *rétro-méatique* ne devant pas dépasser, en haut, un plan longeant le bord supérieur du méat, non seulement, le puits de trépanation devra être creusé à plus de 2 cm. de profondeur pour arriver au voisinage de l'antre, mais encore il passera au-dessous de lui et de l'aditus et le ciseau finira par arriver sur le facial.

Avec le procédé de trépanation transspino-méatique on ne court aucun de ces risques :

1° L'antre, si petit qu'il soit, ne doit pas échapper aux coups de ciseau, qui tracent la paroi supérieure du puits de trépanation, car ceux-ci



FIGURE XII

Cette figure représente la région mastoïdienne de la pièce, qui est représentée dans la fig. X, vue par sa face inférieure (face inférieure de la région mastoïdienne).—1, canal carotidien.—2, apophyse styloïde.—3, méat auditif externe.—4, trou stylo-mastoïdien.—5, sommet de l'apex.—6, rainure digastrique présentant de nombreux pertuis vasculaires.—7, crête digastrique.—8, sillon de l'artère occipitale, présentant aussi de nombreux pertuis vasculaires (l'ensemble de ces pertuis vasculaires, marqués dans les sillons 6 et 8, forme ce que j'appelle la zone criblée digastrique).—9, suture mastoïdo-occipitale inférieure.—10, orifices du canal de la veine mastoïdienne (11, de la figure X).—11, apophyse jugulaire de l'occipital.—12, trou déchiré postérieur (fosse jugulaire).

creusent l'os suivant un plan parallèle et sous-jacent au plancher de la cavité crânienne et situé à 3 mm. au-dessus de celui qui correspond à la paroi supérieure du conduit auditif, c'est-à-dire à 3 mm. au-dessus du plancher de la cavité adito-antrale. La partie supérieure du fond du puits de trépanation transspinale doit donc toujours rencontrer le fond de l'antre, même s'il est en position très élevée et cela avec un minimum de distance à parcourir.

2° On ne peut blesser le facial, parce que le puits de trépanation s'éloigne de la zone dangereuse, comme le montre la ligne *D* (fig. XIV). A moins d'un centimètre de profondeur on doit trouver l'antre. Si on ne le trouve pas, il n'y a qu'à agrandir le puits de trépanation en avant, aux dépens de l'angle postéro-supérieur du conduit : on se rapproche, ainsi, encore plus de l'aditus et toujours sans danger pour le facial, puisque l'orientation de la trépanation n'est pas parallèle à cette paroi. D'ailleurs resèquerait-on ainsi la paroi postérieure du conduit sur toute sa longueur, jusqu'au cadre tympanal, non compris cependant, on n'aurait rien à craindre, parce que c'est une erreur, répétée dans tous les livres, que de croire que le facial traverse la partie profonde de cette paroi. L'aqueduc de Fallope ne dépasse pas, en dehors, le cadre tympanal et reste presque toujours dans l'intérieur de la paroi postérieure de la caisse, ainsi que le montrent les coupes horizontales (fig. XVI). Plus la trépanation empiète sur le conduit, moins long est le chemin à parcourir pour arriver à la cavité adito-antrale.

La trépanation transspino-méatique réduit aussi au minimum les risques que l'on peut avoir de blesser le sinus latéral, même lorsqu'il est en procidence extrême et qu'il arrive au contact de la paroi postérieure du conduit. Dans cette disposition, en effet, le sinus procident se met en rapport surtout avec les $\frac{3}{4}$ inférieurs de la paroi postérieure du conduit et reste à distance suffisante de son angle supéro-postérieur, pour que la trépanation transspinale se fasse sans danger (figs. I, XIII, XV). De plus, bien que l'axe de cette trépanation ne soit pas parallèle à la paroi postérieure, l'obliquité en avant et en dedans, que je lui donne, l'éloigne suffisamment du sinus.

Tout récemment, j'ai eu l'occasion de vérifier ce fait. Mon assistant faisait, sous ma direction, la trépanation transspino-méatique sur une fillette de 12 ans, qui était atteinte d'otorrhée chronique. L'os était éburné, l'antre tout petit et le sinus latéral extrêmement procident. Après avoir trépané à 6 mm. de profondeur, je dis à mon aide de se donner du jour en évasant un peu la lèvre postérieure du champ de trépanation, et, pour cela, de donner un coup de gouge très oblique et presque tangentiel à la surface de l'os. Fort heureusement cette orientation fût même exagérée et le coup de gouge donné avec prudence, car à *2 millimètres* au-dessous de la corticale le sinus fût mis à nu. Nul doute que, dans ce cas, la trépanation classique, même faite avec la plus grande prudence, n'eût lésé le sinus, sur lequel le ciseau serait allé directement, le blessant fatalement, étant donnée sa situation extrêmement superficielle.



FIGURE XIII

Temporal provenant de l'autopsie d'un sujet sur lequel a été faite une large trépanation mastoïdienne, avec ouverture de la gouttière du sinus latéral, au niveau de son coude, et avec ouverture de la fosse cérébrale moyenne. Mastoïde petite, apex compact, antre petit, sinus procident.

1, racine horizontale de l'apophyse zygomatique.—2, racine transverse de cette apophyse.—3, branche descendante de la racine horizontale.—4, branche antérieure de l'arc tympanal : son extrémité supérieure est, sur cette pièce, bien détachée de la branche descendante 3, et limite avec elle une petite fossette.—5, paroi postérieure du conduit auditif externe. Les $\frac{3}{4}$ externes de cette paroi ont été enlevés par la trépanation, ce qui permet de bien voir la cavité antrale, qui est très petite.—6, cavité glénoïde.—7, apophyse styloïde.—8, saillie hémisphérique que le canal demi-circulaire externe forme sur la paroi interne de l'aditus. Celui-ci est mis assez largement à découvert par l'ablation de la plus grande partie de la paroi postérieure du conduit auditif externe.—9, paroi interne de l'antre. La saillie 8 faisant partie de l'aditus, on voit que cet antre est tout petit.—10, apex compact.—11, cellules.—12, large ouverture faite à la gouttière sinusienne au niveau du coude du sinus latéral. Elle permet de voir que la gouttière du sinus, procident, s'enfonce dans la base de la pyramide.—13, bord supérieur de la pyramide formant un bord saillant au-dessus du sinus, à la façon des briques au bord d'un toit.—14, pointillé représentant en projection le trajet du sinus latéral. Ce sinus est très procident et arrive très près de la paroi postérieure du conduit auditif externe, cantonnant l'antre, qui est tout petit, dans la région qui correspond à l'angle supéro-postérieur du méat auditif.—15, trépanation de l'os écailleux, conduisant dans la fosse cérébrale moyenne.—16, face supérieure de la pyramide pétreuse (paroi supérieure de l'antre).

La trépanation transspino-méatique me paraît n'avoir qu'un léger inconvénient : c'est qu'au niveau du bord supérieur du puits de trépanation on risque d'ouvrir la cavité crânienne, lorsque celle-ci est très procidente en bas et en dehors. Lorsque la position du plancher crânien est normale, on n'a rien à craindre, car, la limite supérieure de la trépanation ne remontant pas à plus de 3 mm. au-dessus du plancher de la cavité adito-antrale, on reste à distance suffisante de la cavité crânienne. Mais, si la voûte de l'antre est mince et si l'angle externe de la cavité crânienne est procident en bas et en dehors, on risque que cet angle ne se trouve sur le trajet même du tranchant de la gouge et que celle-ci n'ouvre alors la cavité crânienne. Cependant, même dans ce cas, cet accident a peu d'importance, parce que le tranchant de la gouge s'enfonce *tangentiellement* à la surface de la dure-mère et ne risque pas de la blesser, pour peu que l'on soit prudent. Or l'ouverture de la cavité crânienne, sans blessure des méninges, est sans importance, si l'on agit avec propreté, comme le doit faire tout chirurgien.

La **paroi inférieure** de l'antre est tournée vers l'apex, dont elle est séparée par une région riche en cellules, que j'appellerai, plus loin, *région sous-antrale*. Le niveau, auquel correspond cette paroi, varie suivant la forme et le volume de l'antre. Elle correspond, en général, à un plan horizontal passant par le point d'union du $\frac{1}{3}$ supérieur avec les $\frac{2}{3}$ inférieurs de la paroi postérieure du conduit auditif.

La **paroi antérieure** de l'antre est tournée vers la caisse et en est séparée par le bloc osseux, dans lequel descend la dernière portion de l'aqueduc de Fallope : pour cela, cette paroi est, au point de vue opératoire, une zone dangereuse. Elle est incomplète et n'arrive pas, en haut, jusqu'à sa paroi supérieure. Elle présente, là, une large brèche, qui fait communiquer l'antre avec la partie épitympanique de la caisse. Cette fenêtre, intermédiaire à l'antre et à la caisse, a été appelée par Sappey canal *pétro-mastoïdien*. Cette dénomination est impropre, car le canal est compris entre deux os, qui sont, embryologiquement, indépendants l'un de l'autre : l'os pétreux et l'os écailleux. Il vaudrait donc mieux l'appeler canal pétro-écailleux. Mais il est mieux connu des otologistes sous le nom *d'aditus ad antrum*, que nous lui conserverons.

Aditus ad Antrum.—L'aditus ad antrum est donc une ouverture intertympano-antrale, créée aux dépens de la partie supérieure de la paroi antérieure de l'antre et de la paroi postérieure de la caisse. La profondeur de cette ouverture représente la longueur de l'aditus et est égale à l'épaisseur même de la paroi intertympano-antrale, à ce niveau : elle mesure 4 à 5 mm. Chez l'adulte la hauteur de l'aditus est de 4 mm. Sa largeur est de 3 mm. au niveau de sa voûte et de 1 à 2 mm. au niveau du plancher. L'aditus a quatre parois. Sa paroi inférieure, ou plancher, correspond à un plan horizontal passant par le bord supérieur de la membrane du tympan. Elle est souvent creusée en forme de gouttière, à l'extrémité antérieure de laquelle se couche la courte branche de l'enclume. Le facial fait son deuxième coude sous le plancher

de l'aditus, dont il n'est séparé que par une mince lamelle osseuse. Cette paroi tombe à pic du côté de la caisse et forme, assez souvent, un rebord saillant, qui en surplombe la paroi postérieure. En arrière, le plancher de l'aditus se continue avec la paroi antérieure de l'antre, tantôt à angle droit, tantôt en plan incliné, parfois, lorsque l'antre est petit et haut placé, les parois inférieures de l'antre et de l'aditus se continuent l'une avec l'autre sur un même plan horizontal. — La paroi externe de l'aditus se trouve immédiatement au-dessus de l'angle supéro-postérieur du conduit auditif externe et de la spina-supra-méatum (fig. III). Dans les cas où l'ensemble de l'oreille moyenne est surbaissé, par suite d'un abaissement général de la cavité crânienne, la paroi externe de l'aditus correspond à un plan horizontal passant par l'angle supéro-postérieur lui-même et par la spina. — La paroi supérieure regarde la cavité crânienne, comme celle de l'antre et de la caisse. Rien ne marque la limite entre celles-ci et la paroi supérieure de l'aditus. Comme elles, cette paroi peut être mince, déhiscente, ou bien épaisse et souvent riche en cellules. Elle est très mince lorsque la cavité crânienne descend plus que normalement. Elle est un siège de prédilection pour les perforations pathologiques du tegmen-tympani, surtout dans le cas de cholestéatome. — La paroi interne correspond à la branche externe du canal semi-circulaire horizontal, qui, à ce niveau, traduit sa présence par une saillie de forme hémisphérique, lisse et polie comme de l'ivoire. Immédiatement au-dessous de cette saillie se trouve la partie terminale de la 2e portion (horizontale) de l'aqueduc de Fallope, c'est là que le facial fait son 2e coude en passant au-dessous du plancher de l'aditus, pour descendre dans l'épaisseur de la paroi intertympano-antrale.

Les parois de l'aditus présentent toujours de petits orifices, qui conduisent dans des cellules plus ou moins grandes et quelquefois à peine ébauchées. Les cellules manquent au niveau de la saillie hémisphérique, qui correspond au canal demi-circulaire horizontal. Celles qui sont au-dessus de cette saillie se dirigent vers la région intercanaliculaire. Les cellules du plancher entourent plus ou moins l'aqueduc de Fallope. Celles de la paroi externe existent toujours (cellules fœtales de la paroi externe) et sont souvent volumineuses : elles sont les premières à apparaître chez le fœtus au 7e ou 8e mois.

La suppuration des cellules de la paroi externe de l'aditus, comme celles du mur de la logette des osselets, dont elles ne sont, en somme, que le prolongement postérieur, provoquent l'ostéite et la périostite du mur de la logette (chute de la paroi postéro-supérieure du fond du conduit auditif externe), qui est un des signes importants et précoces des otites moyennes aiguës.

La suppuration des cellules de la voûte a la même importance que celles de la voûte de la caisse et de l'antre : propagation des lésions à la cavité crânienne.

La suppuration des cellules du plancher peut provoquer des troubles du nerf facial et, par l'aqueduc lui-même, se propager, soit en


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FIGURE XIV

Coupe horizontale d'un temporal passant un peu au-dessous du bord supérieur du méat auditif externe. A gauche : tranche supérieure ; à droite : tranche inférieure. La coupe a longé la paroi supérieure du conduit auditif externe, qui se trouve dans la tranche supérieure (gauche) ; elle est, aussi, passée à l'extrémité inférieure de la spina-supra-méatum, qui est restée tout entière dans la tranche supérieure, au niveau de l'angle supéro-postérieur du méat. La partie profonde, sus-tympanale, de la paroi supérieure du conduit a été touchée par la coupe, de sorte qu'on voit un segment du mur de la logette des osselets sur les deux coupes de la figure. Enfin, à la face postérieure du rocher, la coupe est passée par la partie supérieure de la fente unguéale et a, fort heureusement, ouvert l'aqueduc du vestibule, dans toute sa longueur. La ligne 22 représente l'axe antéro-postérieur de la cavité tympano-antrale. L'angle formé par les lignes 22 et 23 représente l'angle, que forme l'axe antéro-postérieur de la caisse et de l'antre avec la face postérieure du rocher. La ligne *A* représente la limite interne du bloc mastoïdien. La ligne *B* représente sa limite antérieure. La ligne *D* passe par la spina-supra-méatum, et tombe perpendiculairement sur l'axe antéro-postérieur de l'antre : elle représente le chemin de la *trépanation transspino-méatique* ; elle forme avec la ligne *B*, un angle ouvert en dedans et en avant, qui montre que dans la trépanation transspino-méatique, le ciseau s'éloigne de la paroi postérieure du conduit, à mesure qu'il s'enfonce dans l'os : c'est-à-dire qu'il fuit la région dangereuse du facial ; tandis que dans la trépanation classique, dans laquelle le ciseau s'enfonce parallèlement à cette paroi (flèche *F*), l'instrument s'approche, au contraire, de plus en plus de la région du facial, à mesure qu'il s'enfonce dans l'os. L'orientation de la ligne index 21, qui est sur le côté *droit* de la figure, est la même que celle de la ligne *D*, qui est sur le côté gauche : on voit que cette ligne, même prolongée à travers l'antre, ne peut atteindre le facial.

1, coude du sinus latéral.—2, cellules de la paroi interne de l'antre.—3, canal demi-circulaire postérieur.—4, canal demi-circulaire externe.—5 et 6, section de la fente unguéale.—7, aqueduc du vestibule.—8, vestibule.—9, cellule pétreuse postérieure (rétro-labyrinthique).—10, partie commune à la branche postérieure du canal demi-circulaire supérieur et au canal demi-circulaire postérieur. Cette partie a été coupée au niveau de son abouchement dans l'angle postéro-interne du vestibule, qui, dans la tranche droite de la coupe, est marquée d'une + blanche.—11, conduit auditif interne. La coupe n'a ouvert que la partie profonde de sa paroi supérieure au niveau du trou, qui sert au passage du nerf facial.—12, 1re portion de l'aqueduc de Fallope.—12′, orifice pour la sortie du grand pétreux superficiel par l'hiatus de Fallope.—13, 1er coude de l'aqueduc de Fallope.—14, 2e portion de l'aqueduc de Fallope.—14′, déhiscence de la paroi inférieure de l'aqueduc de Fallope au niveau du point où cet aqueduc passe au-dessus de la fenêtre ovale.—15, 2e coude de l'aqueduc. L'aqueduc a été ouvert par la coupe sur toute l'étendue de ses deux premières portions. Celles-ci ne se voient que sur la moitié droite de la coupe, parce que le trait de scie a seulement rasé la paroi supérieure du canal.—16, caisse du tympan.—17, petites cellules pétreuses antérieures, situées en avant du premier coude du facial, au-dessus de l'hiatus de Fallope.—18, mur de la logette des osselets.—19, paroi antérieure du conduit auditif externe.—20, sa paroi postérieure.—20′, spina-supra-méatum.—21, antre.—22, axe antéro-postérieur de la cavité épitympanique et de l'antre.—23, ligne longeant la face postérieure du rocher.—24, orifice du canal osseux que traverse, sous le bord supérieur du rocher, le sinus pétro-squameux pour arriver dans la gouttière du sinus latéral.

haut, vers la cavité crânienne, soit en bas, vers la région mastoïdienne inférieure, par le trou stylo-mastoïdien.

La suppuration des cellules de la paroi interne, labyrinthique, peut se propager aux canaux demi-circulaires. Les cellules de cette paroi entourent tout particulièrement le canal demi-circulaire externe. Leur suppuration peut provoquer une carie, avec ou sans fistule, de la branche externe du canal, et amener une labyrinthite suppurée. Mais il faut se garder de confondre une petite cellule suppurée péri-canaliculaire avec une véritable fistule de ce canal. Les cellules, qui sont au-dessus du canal demi-circulaire externe, peuvent se prolonger profondément au-dessus de lui, dans l'espace compris entre les trois canaux demi-circulaires (cellules intercanaliculaires) : leur suppuration peut aussi atteindre le labyrinthe en un point éloigné de l'aditus lui-même.

Paroi supérieure. — La paroi supérieure de l'antre se continue directement, en avant, avec la paroi supérieure de l'aditus et de la caisse. Projeté à la face supérieure du rocher, son axe antéro-postérieur (fig. II, 18) se dirige obliquement d'arrière en avant et de dehors en dedans, suivant une ligne, qui va de la gouttière du sinus latéral à l'angle que forment, en se rencontrant, les bords supérieurs des plans, qui limitent le bloc mastoïdien en avant (du côté du conduit et de la caisse), et en dedans (du côté de la pyramide) (fig. III, 8, 8′, 18 et fig. VII). Cette paroi, comme celle de la caisse et de l'aditus est parfois mince, lamelleuse, déhiscente (déhiscences en trou de mite (fig. XXI), déhiscences de la fissure pétro-squameuse interne) ; elle peut aussi être très riche en cellules et être très épaisse. Elle est en rapport avec le sinus pétro-squameux et la face inférieure de la 3e circonvolution temporale.

Paroi postérieure. — Cette paroi est tournée en arrière et en dedans vers la région du sinus latéral. La distance, qui sépare cette paroi du sinus, est très variable. Si le sinus est légèrement procident, il se rapproche de la paroi postérieure de l'antre et la paroi osseuse, qui les sépare, est en général mince. Si la procidence est très accentuée, non seulement le sinus semble refouler en avant la paroi postérieure de l'antre, qui est tout petit et en position fœtale, mais la paroi osseuse intersinuso-antrale est, aussi, très mince (fig. XIII) et peut même être déhiscente (cas de Brühl, figs. XIX et XX). A l'état normal (figs. VII, XIV), l'antre est séparé du sinus par une épaisseur d'os de 4 à 5 mm. Suivant la nature même de l'os, l'espace intersinuso-antral peut être formé soit par du tissu spongieux, soit par du tissu pneumatique. Lorsque l'antre est très grand, il peut s'étendre, en arrière, au delà de la zone qui correspond au sinus latéral (fig. XVIII).

Paroi interne. — La paroi interne de l'antre n'est pas directement tournée vers la ligne médiane. Elle est obliquement dirigée en dedans et en arrière. De même que sa paroi externe est tournée vers l'angle, que forment, en se réunissant, la face externe et la face antérieure du bloc mastoïdien (ligne *D*, fig. XIV), de même sa paroi interne est tournée vers l'angle, que forment en se rencontrant la face interne ou pétreuse

du bloc avec sa face postéro-interne et plus particulièrement avec la partie de celle-ci que j'ai appelée *zone pétreuse postérieure*. Elle regarde donc, tout à la fois, la région des canaux demi-circulaires et la face cérébelleuse de la pyramide. (Sur la figure XIV, l'angle, que fait la face interne du bloc avec sa face postéro-interne, est représenté par l'intersection de la ligne *A* avec la ligne 23.)

Nous pouvons donc lui distinguer deux parties : une antérieure, labyrinthique, et une postérieure, cérébelleuse.

La partie labyrinthique correspond à la grande courbure du canal demi-circulaire externe et à celle du canal vertical postérieur. Suivant que la cavité antrale est plus ou moins profonde : cette partie correspond directement à la grande courbure de ces deux canaux demi-circulaires (fig. XVIII) ou bien seulement à la courbure du canal demi-circulaire externe (fig. XIV). Dans ce dernier cas, la longueur de cette partie, mesurée à partir du versant postérieur de la saillie hémisphérique que forme le canal horizontal sur la paroi interne de l'aditus, est d'environ 3 mm. seulement. Dans l'autre, la partie labyrinthique s'étend jusqu'à la surface de la grande courbure du canal vertical postérieur et mesure 5 mm. Sa surface présente de petites dépressions cellulaires, qui, dans les cas de suppuration, peuvent être confondues, si l'on n'y fait bien attention, avec une fistule suppurée du canal demi-circulaire correspondant.

La partie postérieure ou cérébelleuse de la paroi interne de l'antre regarde la face postérieure de la pyramide et correspond à la zone pétreuse postérieure du bloc mastoïdien. La distance, qui sépare la cavité antrale de la fosse cérébelleuse, varie avec les sujets et surtout avec la constitution osseuse, plutôt qu'avec le volume même de la cavité antrale. Il semblerait, à priori, que plus celle-ci est profonde, plus la paroi osseuse doit être mince. Cela n'est pas exact. Dans la coupe (fig. XVIII), où l'antre est très grand, la paroi interantro-cérébelleuse est aussi épaisse que dans les coupes représentées dans les figures III, VII, XIV, XVII, où l'antre est beaucoup plus petit. Si cette paroi est pneumatique l'épaisseur peut être de 5 à 6 mm. Dans les mastoïdes compactes elle est souvent plus réduite, la paroi postérieure de l'antre peut être formée par une simple lamelle osseuse, ayant à peine 1 à 2 mm. d'épaisseur. Toutefois, même dans les mastoïdes compactes, l'épaisseur de la paroi interne de l'antre peut être aussi grande que dans les mastoïdes pneumatiques ; une lame épaisse et compacte sépare, alors, l'antre de la fosse cérébelleuse.

Si l'antre est très grand, dans le sens de la hauteur, et s'il descend presqu'au niveau d'un plan horizontal passant par la paroi inférieure du conduit auditif externe, la partie cérébelleuse de sa paroi interne est en rapport avec toute la hauteur de la zone pétreuse de la face postéro-interne du bloc. Lorsque l'antre est de volume moyen, elle ne correspond qu'à la partie supérieure de cette zone. S'il est surtout allongé dans le sens antéro-postérieur, la paroi interne peut se prolonger au delà de la région sinusienne : sa partie la plus reculée est, alors, en rapport avec le sinus latéral (fig. XVIII). Lorsque le sinus est procident à la

FIGURE XV

Coupe horizontale d'un temporal gauche, passant immédiatement au-dessous de la spina-supra-méatum. Le rocher est petit, le sinus est très procident, l'antre, tout petit et haut placé, est resté dans l'épaisseur de la tranche supérieure (côté droit), où j'ai représenté sa situation, ainsi que celle de l'aditus, par une ligne pointillée. L'apex mastoïdien était très petit, mais la rainure digastrique se prolongeait très haut sur la face externe de la mastoïde, ce qui fait qu'on la voit même sur cette coupe, qui passe près de la paroi supérieure du conduit auditif. La projection de l'antre en pointillé montre bien que, dans un cas semblable, la trépanation transspino-méatique peut seule permettre d'ouvrir l'antre sans ouvrir le sinus latéral.

1, rainure digastrique remontant très haut, ici, sur la face externe de la mastoïde. —2, spina-supra-méatum.—3, paroi supérieure du conduit auditif externe.—3′, conduit auditif.—4, mur de la logette des osselets.—5, gouttière du sinus latéral très procident.—6, antre petit et haut placé, contenu dans l'épaisseur de la tranche supérieure de la coupe, et représenté en pointillé à la surface de la section osseuse.—6′, cellule sous-jacente à l'extrémité postérieure de l'antre.—7, caisse du tympan (à droite, c'est plus particulièrement sa partie supérieure ou attique).—7′, marteau, dont la tête, coupée par le trait de scie, devrait se retrouver à droite dans l'attique, mais elle a disparu, ainsi que l'enclume, au cours des manipulations qu'a subies la pièce.—8, relief que fait l'aqueduc de Fallope en passant au-dessus de la fenêtre ovale. Entre lui et le vestibule (12) se trouve 8′, la fenêtre ovale. Cette fenêtre a été coupée au ras de son bord inférieur et ne se retrouve pas sur la moitié gauche (inférieure) de la coupe.—9, aqueduc de Fallope coupé au niveau de son 2^{e} coude.—10, hiatus de Fallope.—11, cavité où loge le muscle de l'étrier.—12, vestibule coupé au ras de son plancher : aussi dans la moitié gauche de la coupe, au lieu de voir le vestibule, on ne voit que la partie initiale du premier tour de spire du limaçon, partie dite cavité sous-vestibulaire (14′).—13, conduit auditif interne, ouvert par une fenêtre, qu'a faite la coupe en rasant sa paroi inférieure.—14, limaçon.—14′, cavité sous-vestibulaire ou partie initiale du premier tour de spire du limaçon : le relief qu'elle fait dans la caisse correspond au promontoire.—15, trou déchiré postérieur, en grande partie caché par le relief de la face postérieure du rocher.—16, trou condylien.—17, face postérieure du rocher.—18, gouttière du sinus pétreux inférieur.—19, trou occipital. —20, apophyse basilaire de l'occipital.


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surface du triangle rétro-pétreux du bloc mastoïdien la paroi interne de l'antre est en rapport direct avec ce vaisseau. Cette disposition est la règle chez le fœtus, le nouveau-né et le jeune enfant, car, à cet âge, la base de la pyramide pétreuse étant encore toute petite, les organes sont très rapprochés les uns des autres (Pl. VI, fig. F 1 et F 2 et Pl. XVI in *Annales des Mal. de l'Oreille*, 1913, loc. cit.).

Lorsque l'antre est tout petit, rudimentaire, sa paroi interne se trouve limitée à la partie labyrinthique.

La connaissance des rapports de la partie interne de l'antre est d'une grande importance pour le chirurgien, lorsqu'il doit intervenir sur elle-même. On peut, en effet, être amené à poursuivre les lésions dans la profondeur de cette paroi : soit du côté du labyrinthe, soit du côté de la fosse cérébelleuse. L'orientation qu'il convient de donner au bec de la curette ou au tranchant de la gouge, varie suivant la partie que l'on veut atteindre. Mais, avant tout, l'opérateur doit avoir bien présent à l'esprit, l'orientation des surfaces osseuses qu'il doit traverser :

1° La face cérébelleuse de la pyramide pétreuse n'est pas parallèle à la face interne de l'antre, elle a, au contraire, à peu près la même orientation que la paroi postérieure du conduit auditif externe et lui est presque parallèle (voir coupes horizontales).

2° La paroi externe et la paroi interne de l'antre sont orientées comme son grand axe lui-même, c'est-à-dire d'arrière en avant et de dehors en dedans, et chacune d'elles forme avec la face correspondante du bloc mastoïdien (l'interne avec la face postérieure du rocher, l'externe avec la paroi postérieure du conduit auditif), un angle de 45° environ (voir la fig. XIV). Mais tandis que l'angle, que forme la paroi externe de l'antre avec la paroi postérieure du conduit auditif, est ouvert en dehors et en arrière (angle formé par la rencontre de la ligne *B* avec la ligne 22), l'angle, que forme la paroi interne de l'antre avec la face postérieure de la pyramide (zone pétreuse de la face postéro-interne du bloc mastoïdien), est ouvert en dedans et en avant (angle formé par la rencontre des lignes 22 et 23).

3° La face interne de l'antre, considérée de l'extérieur, est, comme sa face externe, tournée, tout à la fois, vers la paroi postérieure du conduit auditif externe et vers la partie de la surface extérieure de la mastoïde, qui est en arrière du méat auditif (même fig. XIV).

4 La paroi profonde de l'antre ne peut être bien attaquée, que si elle est complètement mise à découvert : pour cela il faut abattre les parties extérieures qui la recouvrent, c'est-à-dire la *paroi postérieure du conduit et la zone mastoïdienne rétro-méatique*. La résection de la paroi postérieure du conduit, telle qu'on la fait pour l'évidement pétro-mastoïdien permet, seule, d'attaquer la paroi interne de l'antre bien directement et bien librement.

Pour aller au labyrinthe, à travers la paroi interne de l'antre, c'est la partie antérieure de cette paroi qu'il faut attaquer. On peut, par là, pénétrer soit dans le canal demi-circulaire postérieur, soit dans le canal demi-circulaire externe.

Pour ouvrir le canal demi-circulaire externe, le bec du ciseau doit

être appliqué sur la saillie hémisphérique, que forme la branche externe de ce canal sur la paroi externe de l'aditus. Mais il ne faut pas dépasser le bord inférieur de cette saillie, car, immédiatement au-dessus d'elle, se trouve la partie terminale de la 2e portion de l'aqueduc de Fallope. Ce voisinage immédiat du facial est démontré par les figures III et IV. La figure XIV est encore plus démonstrative. Cette coupe a été faite avec une scie de 1 mm. d'épaisseur, et cette épaisseur du trait de scie a été suffisante pour que la section passant au bord inférieur de la saillie hémisphérique ouvre, tout à la fois, le canal demi-circulaire externe (4) et le canal de Fallope (15). Sur la face supérieure de la coupe (côté gauche), on ne voit que le canal demi-circulaire ouvert ; sur la face inférieure de la coupe (côté droit), on ne voit que le canal de Fallope ouvert aussi. Cela prouve combien ces deux canaux sont tout prêts l'un de l'autre : la moindre échappée vers le bas porterait le tranchant du ciseau sur le facial. Il faut donc se tenir à la partie supérieure de la saillie hémisphérique et mieux encore attaquer celle-ci à sa partie postérieure. On a ainsi plus de chances de s'éloigner du nerf, qui descend sous le plancher de l'aditus au niveau de la partie moyenne du bord inférieur de cette saillie.

Pour ouvrir le canal demi-circulaire postérieur, il faut placer le tranchant du ciseau sur la limite postérieure de la saillie hémisphérique et, tout en le dirigeant obliquement *en avant et en dedans*, l'enfoncer dans la paroi interne de l'antre à 3 mm. de profondeur. Le ciseau, *tenu à plat*, pénètre alors dans la partie convexe du canal vertical postérieur. Si l'on a déjà ouvert le canal externe, on suit sa courbure en arrière et, quand on voit que cette courbure s'incline en avant (branche postérieure du canal horizontal), on l'abandonne pour continuer la trépanation directement en dedans : on dépasse la région du canal demi-circulaire externe et, à 1 ou 2 mm. plus loin, on trouve la partie moyenne de la courbure du canal vertical postérieur.

Pour pénétrer directement dans la loge cérébelleuse à travers l'antre, il faut attaquer sa paroi interne immédiatement en arrière des canaux demi-circulaires. L'espace est limité et une gouge de 7 à 8 mm. de large l'occupe entièrement lorsque l'antre a un volume ordinaire. Si l'antre est très grand et la paroi interne très étendue dans le sens antéro-postérieur, il faut, quand même, localiser la trépanation à une surface de 7 à 8 mm. en arrière de la région labyrinthique, car, au delà, la paroi interne de l'antre peut être en rapport avec le sinus latéral (fig. XVIII). Pour cette trépanation de la partie cérébelleuse de la paroi interne de l'antre, il faut aussi avoir, au préalable, réséqué largement la paroi postérieure du conduit auditif externe et donner à la gouge une direction *perpendiculaire à celle du conduit*. Mais dans ce procédé le tranchant de l'instrument tombe *perpendiculairement* sur la paroi postérieure de la pyramide pétreuse et la moindre échappée permet de blesser la dure-mère sous-jacente. A cause de cela l'ouverture directe de la fosse cérébelleuse à travers l'antre ne doit être faite que si les lésions de carie

osseuse ont déjà créé le chemin et s'il ne s'agit que de nettoyer ou agrandir une perforation déjà existante.

Si rien de cela n'existe, et qu'on veuille, délibérément, ouvrir la fosse cérébelleuse en abattant la paroi interne de l'antre, il vaut mieux prendre un chemin détourné, qui permettra d'attaquer la face postérieure du rocher, tangentiellement à sa direction et non perpendiculairement. Pour cela il faut d'abord ouvrir, en arrière de l'antre, la gouttière sinusienne. En élargissant, en arrière, le bord postérieur de la trépanation antrale, on découvre la région du sinus. La gouttière du sinus est plus superficiellement placée que la paroi interne de l'antre. On peut alors facilement l'attaquer par des coups de ciseau donnés tangentiellement à la surface osseuse, ce qui permet de ne pas blesser la dure-mère. La gouttière sinusienne ouverte, il est très facile d'agrandir la brèche osseuse avec une pince gouge et de réséquer, ainsi, toute la zone pétreuse de la face postéro-interne du bloc mastoïdien (zone cellulaire intersinuso-auditive), qui sépare l'antre de la fosse cérébelleuse. Ce procédé est bien préférable à la trépanation directe de la paroi interne de l'antre, parce qu'il permet de mettre à découvert, largement et facilement, l'espace rétro-pétreux de la fosse cérébelleuse, sans être obligé de travailler uniquement au fond d'un puits osseux trop étroit. Il est très commode à exécuter, lorsque le sinus latéral n'est pas procident. Il est un peu plus difficile, lorsque le sinus est procident ; mais la trépanation directe de la paroi interne de l'antre serait elle-même bien plus délicate aussi dans ce cas.

Le curettage et la résection de la paroi interne ou cérébelleuse de l'antre permet de traiter les suppurations des cellules de la *zone inter-sinuso-auditive*, les abcès extra-duraux de la région rétro-pétreuse, les abcès du sac endolymphatique, et les abcès cérébelleux par voie pré-sinusienne.

Cellules mastoïdiennes

L'étude du développement des cellules mastoïdiennes nous apprend qu'elles commencent à se former tout d'abord au pourtour direct de l'antre, et tout particulièrement sur sa paroi externe. De là elles rayonnent et s'étendent dans la profondeur de l'os, les unes dans le rocher proprement dit, les autres dans la mastoïde. Les premières s'insinuent entre la corticale périphérique et les organes que contient le rocher (oreille interne, conduit auditif interne) ; les autres vont jusqu'à la corticale périphérique de la mastoïde.

En principe, on peut admettre, et ces cas ne sont pas rares, que les cellules peuvent creuser totalement le bloc mastoïdien. C'est ce que l'on observe dans les mastoïdes très pneumatiques, qui sont les plus volumineuses, par suite de ce boursouflement intérieur. Mais il n'est pas rare qu'elles se développent seulement dans le voisinage de l'antre et que les parties plus éloignées en soient dépourvues. En principe, les cellules se multiplient en formant autour de l'antre une sorte de couronne cellulaire péri-antrale, dont l'épaisseur augmente peu à peu, pour s'étendre régulièrement partout, jusqu'au contact de la corticale périphérique. Mais cet

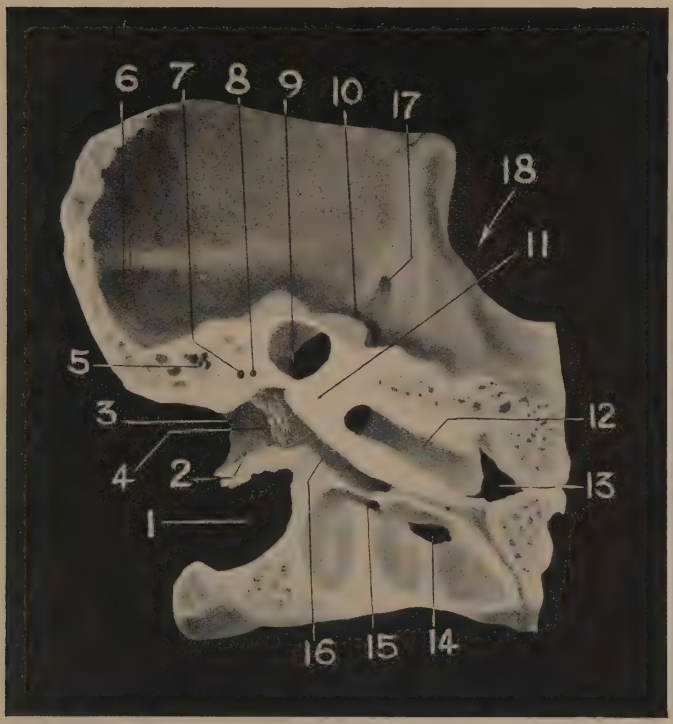


FIGURE XVI

Coupe horizontale du temporal droit, pris sur le même sujet que celui de la fig. XV. La coupe passe près de la paroi inférieure du conduit auditif externe, à travers la zone sous-antrale. Le bulbe de la jugulaire, étant procident sous la face inférieure du rocher, a été largement ouvert.

1, cavité glénoïde ouverte par la coupe.—2, paroi antérieure du conduit auditif externe.—3, sa paroi postérieure.—4, membrane du tympan (partie inférieure).—5, cellules sous-antrales.—6, dernière portion du sinus latéral, courant presque horizontalement, au-dessus de la face inférieure (région digastrique) de la mastoïde.—7, portion descendante de l'aqueduc de Fallope. Cette coupe montre que le facial ne dépasse pas, en général, les limites du cadre tympanal, dans lequel est enchâssée la membrane du tympan.—8, partie la plus inférieure de la cavité, qui loge le muscle de l'étrier.—9, bulbe de la jugulaire procident et ouvert par la coupe.—10, trou déchiré postérieur en partie caché par ce qui reste, dans cette coupe, de la face postérieure du rocher.—11, tissu éburné appartenant à la limite inférieure du labyrinthe.—12, portion horizontale du canal carotidien.—13, trou déchiré antérieur.—14, trou oval.—15, trou petit rond.—16, trompe d'Eustache osseuse.—17, trou condylien.—18, trou occipital.

accroissement cellulaire péri-antral n'est pas toujours régulier. Parfois, il se fait dans un sens et non dans l'autre : les cellules forment alors des groupes plus ou moins importants, que l'on désigne par des noms correspondant aux régions qu'ils occupent : *groupes supérieur, postérieur, inférieur, antérieur* ; ou bien on les désigne encore par des noms, qui précisent davantage leur situation à l'égard des organes avec lesquels elles sont en rapport : *Cellules de la paroi externe de l'antre, cellules périsinusiennes, cellules apicales, cellules limitrophes du conduit auditif externe, cellules intersinuso-faciales, cellules intersinuso-auditives*, etc. On a essayé d'établir quelle peut être la fréquence relative de ces divers groupes cellulaires. Rien n'est moins régulier. En opérant une mastoïdite l'opérateur doit agir comme si la mastoïde était tout entière creusée de cellules pneumatiques. Car il y a, aussi, des dispositions anatomiques, assez fréquentes, dans lesquelles les cellules mastoïdiennes peu nombreuses et petites ne forment pas de groupes, mais s'étendent isolément très loin dans la profondeur de l'os : les cellules sont alors diffuses et clairsemées dans la profondeur du bloc mastoïdien. Il faut bien connaître ces variabilités de disposition des cellules mastoïdiennes, mais rien ne peut les faire prévoir à l'avance. Une mastoïde très volumineuse est, en général, une mastoïde très pneumatique ; une mastoïde petite est souvent une mastoïde compacte ; mais cela n'est pas absolu et rien ne permet d'affirmer, de façon certaine, que telle région sera pneumatique et que les autres parties seront dépourvues de cellules. Même en trépanant une mastoïde, qui paraît être uniquement spongieuse ou compacte, on ne peut affirmer qu'il ne se trouve pas dans l'épaisseur du bloc mastoïdien des cellules clairsemées, suppurant peut-être, et dont la non-ouverture peut être la cause de conséquences graves, comme on en a mentionnées dans des cas de cellules ' aberrantes ' méconnues.

Ceci explique combien est fondée l'opinion des otologistes, qui veulent que la trépanation mastoïdienne ne soit pas simplement une trépanation antrale, mais qu'elle soit surtout un large évidement de tout le bloc mastoïdien.

Ouvrir parcimonieusement une mastoïde malade est un acte dangereux, non seulement parce qu'on risque de laisser des cloaques cellulaires pleins de pus et se vidant mal, mais, aussi, parce que le traumatisme opératoire, qui ouvre au pus la porte extérieure, ouvre malheureusement, aussi, des voies de passage dans la profondeur de l'os, en rendant béants de nombreux espaces intra-osseux, au niveau desquels les germes infectieux peuvent s'insinuer, et pénétrer plus facilement peut-être qu'avant l'acte opératoire, alors que la défense naturelle de l'organisme avait sans doute établi une barrière de résistance au pourtour direct des cavités suppurantes. Sans compter que le traumatisme opératoire, s'il est incomplètement et mal fait, peut augmenter encore la virulence des germes.

Dans la trépanation de la mastoïde, l'opération ne doit pas être parcimonieuse, mais doit, au contraire, être exécutée le plus largement possible : ne s'arrêter qu'en tissu sain et terminer l'évidement par une

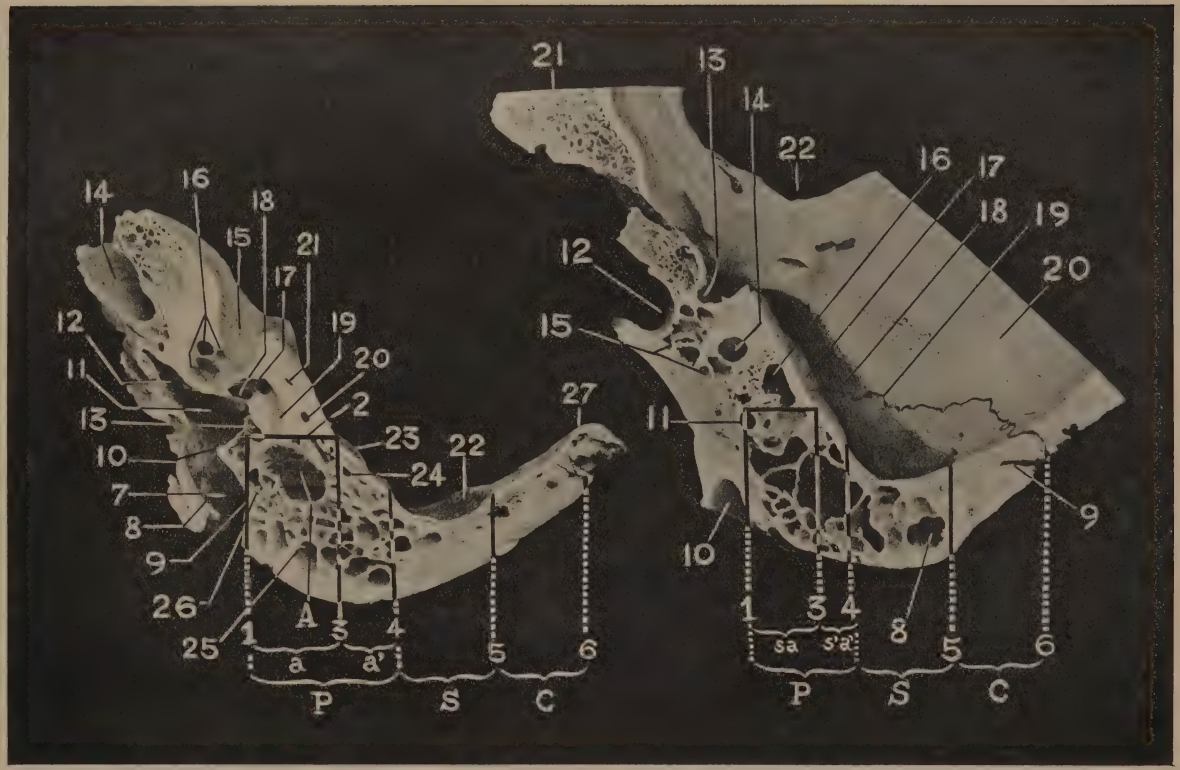
large cautérisation de la surface évidée avec un antiseptique susceptible de coaguler les albumines du sang au niveau des interstices osseux, qui ont été rendus béants par l'acte opératoire, et en fermer ainsi le passage. C'est de cette façon que doivent agir les badigeonnages faits, en fin d'opération, soit avec de la teinture d'iode, soit avec le chlorure de zinc au $\frac{1}{10}^{e}$.

Comme je viens de le dire, on a voulu désigner les groupes cellulaires de la mastoïde par des appellations basées soit sur la situation, que chacun occupe dans la mastoïde, soit sur les rapports que chacun affecte avec un organe important (sinus, nerf facial, etc.). Cette division ne manque pas d'intérêt et il convient de la connaître. Mais ce qui me paraît avoir peut-être plus d'importance pour le chirurgien, c'est de pouvoir diviser schématiquement la face extérieure, c'est-à-dire la surface opératoire de la mastoïde, en régions ou zones bien déterminées et de pouvoir, à l'avance, savoir à peu près exactement à quelles parties profondes de l'os ou de la cavité crânienne elles correspondent. Une telle connaissance des rapports des couches superficielles avec les parties profondes doit, en effet, permettre d'avoir des indications précises, non seulement sur l'interprétation des symptômes mastoïdiens au cours des otites, mais aussi sur l'orientation des opérations, qui doivent être faites, en donnant à l'opérateur le plus de sûreté dans la direction qu'il doit prendre, pour aller aussi profondément qu'il sera nécessaire.

Au point de vue descriptif, nous avons déjà pu diviser la surface extérieure de la mastoïde en région antérieure et en région postérieure, séparées l'une de l'autre par une zone rugueuse obliquement ascendante, sur laquelle s'insèrent les muscles mastoïdiens : Cette zone va du sommet de l'apex au voisinage de l'angle pariéto-occipito-mastoïdien. Nous avons dit que la partie située en avant d'elle représente plus particulièrement la région opératoire. Mais cette région, ainsi délimitée, ne donne pas d'indications sur les rapports de la surface extérieure de l'os avec les régions profondes. Il convient donc de remplacer cette division, purement descriptive, par une autre plus topographique et plus chirurgicale. Déjà Poirier et d'autres auteurs ont fait cette division, en décrivant à la surface mastoïdienne une région antérieure, antrale ; une région moyenne, sinusienne ; et une région postérieure, cérébelleuse. Je conserverai cette division en essayant de la préciser encore un peu plus. Bien entendu cette division sera établie pour une mastoïde dans laquelle toutes les parties ont des dimensions et des formes normales. A côté de cela il conviendra de faire les restrictions nécessaires, en prévision des anomalies, malheureusement assez fréquentes.

Considérons deux coupes horizontales de la mastoïde, telles que celles qui sont représentées dans les figures XVII[1] et XVII[2]. La figure XVII[1] représente une coupe passant immédiatement au-dessous de la paroi supérieure du conduit auditif externe et du plancher de l'aditus. La ligne 2 représente la limite interne (pétreuse) du bloc mastoïdien.

Les lignes 1, 3, 4 sont parallèles entre elles et perpendiculaires à l'axe médian et antéro-postérieur de la cavité crânienne. La ligne 1 longe la



FIGURES XVII[1] ET XVII[2]

Deux coupes horizontales d'un temporal servant à représenter les rapports des zones topographiques, que j'ai décrites sur la face externe de la mastoïde, avec les parties profondes de la région mastoïdienne.

Figure XVII[1]

P S C représentent l'étendue des zones pétreuses (*P*) antérieure (*a*), postérieure (*a'*), sinusienne (*S*) et cérébelleuse (*C*) à la surface externe de la mastoïde. Les n[os] 1, 3, 4, 5, 6 marquent les limites de chacune de ces zones. La ligne 1 marque la limite antérieure de la zone pétreuse antérieure du côté du conduit auditif externe. J'ai dit que pour *schématiser* les coupes, qui servent à délimiter le bloc mastoïdien, je fais une coupe verticale et transversale, perpendiculaire à l'axe antéro-postérieur du crâne : or, comme la paroi postérieure du conduit est un peu oblique de dehors en dedans et d'arrière en avant, il en résulte qu'à sa partie profonde (10) la paroi postérieure du conduit reste en avant de la ligne 1, c'est-à-dire en avant du bloc mastoïdien. Mais en réalité toute la paroi postérieure du conduit doit être comprise dans les limites du bloc mastoïdien. La ligne 2 marque la limite interne, pétreuse, du bloc mastoïdien. La zone pétreuse antérieure (*a*) correspond à la surface de section (2), qui sépare le bloc du restant de la pyramide pétreuse. La coupe, passant à la partie supérieure du conduit auditif externe, correspond à l'antre *A* ; c'est la zone antrale. La zone pétreuse *a'* est comprise entre la zone antrale et la zone sinusienne. Dans la profondeur, elle correspond à la partie de la face postérieure (23) du rocher, qui forme la zone rétro-pétreuse de la face postéro-interne de la région mastoïdienne. En dehors, celle-ci correspond donc à la zone pétreuse postérieure de la face externe de la mastoïde, qu'elle regarde obliquement ; mais comme la zone pétreuse de la face postéro-interne du bloc mastoïdien est, aussi, sensiblement parallèle à la face postérieure du conduit auditif externe, elle regarde aussi la région antrale.

7, paroi inférieure du conduit auditif externe.—8, sa paroi antérieure.—9, sa paroi postérieure.—10, extrémité profonde de la paroi postérieure (au niveau de l'anneau tympanal).—11, caisse.—12, trompe d'Eustache.—13, aqueduc de Fallope.—14, canal carotidien.—15, paroi inférieure du conduit auditif interne.—16, limaçon.—17, orifice vestibulaire de la branche inférieure du canal demi-circulaire postérieur.—18, partie initiale du premier tour de spire du limaçon ou cavité sous-vestibulaire.—19, branche interne du canal demi-circulaire horizontal.—20, coupe du canal demi-circulaire postérieur.—21, coupe de l'aqueduc du vestibule.—22, gouttière du sinus latéral.—23, face postérieure du rocher.—24, cellules de la paroi interne de l'antre.—25, cellules de la paroi externe de l'antre.—26, cellules limitrophes de la paroi postérieure du conduit auditif externe.—27, portion de l'occipital.

Figure XVII[2]

Les lignes de délimitation des diverses zones sont les mêmes que dans la figure précédente. Mais ici la coupe passe par la paroi inférieure du conduit auditif externe. La zone antrale est remplacée par la zone sous-antrale (*s–a*) dans laquelle on voit de très grandes cellules. La zone pétreuse postérieure est marquée *s'–a'*.—8, cellules de la région sinusienne.—9, canal de la veine mastoïdienne.—10, partie externe de la paroi inférieure du conduit auditif.—11, aqueduc de Fallope.—12, partie ascendante du canal carotidien.—13, encoche correspondant à la fossette pyramidale ; grâce à elle le trou déchiré postérieur n'est pas caché par le relief que forme la face postérieure du rocher au-dessus de lui.—14, brèche faite par la coupe à la paroi supérieure du golfe de la jugulaire.—15, cellules du plancher de la caisse.—16, cellule pétreuse postérieure sous-jacente à : 17, face postérieure du rocher.—18, portion terminale du sinus latéral.—19, suture mastoïdo-occipitale,—20, occipital.—21, apophyse basilaire de l'occipital.—22, trou occipital.

paroi postérieure du conduit auditif et tombe sur l'aqueduc de Fallope, à l'extrémité antérieure de la ligne 2. En avant de la ligne 1, la partie profonde de la paroi postérieure du conduit forme un bec saillant (10). Ceci tient à ce que la paroi postérieure du conduit, qui regarde en avant, n'est pas située dans un plan exactement transversal et perpendiculaire à l'axe médian et antéro-postérieur de la cavité crânienne, mais qu'elle est légèrement oblique de dehors en dedans et d'arrière en avant. Il est bien entendu que ce bec osseux fait aussi partie de la face antérieure du bloc mastoïdien, la coupe vertico-transversale, qui me sert à marquer la limite générale de la face antérieure du bloc, de même que la ligne 1 qui la représente sur cette figure, étant, en principe, un peu schématique.

La ligne 3 aboutit à l'extrémité postérieure de la ligne pétreuse 2, à l'intersection de la face postérieure du bloc mastoïdien, avec sa limite pétreuse.

La ligne 4 aboutit au bord antérieur de la gouttière du sinus latéral, c'est-à-dire à la limite externe de la face postérieure du rocher, ou limite entre la zone pétreuse et la zone sinusienne de la face postéro-interne du bloc mastoïdien.

L'espace compris entre 1 et 4 correspond à la base du rocher : cet espace marque, à la surface extérieure de la mastoïde, l'étendue des rapports que cette surface a avec le corps de la pyramide. Nous l'appellerons donc *zone pétreuse* (*P*). Si l'on projette les limites profondes du bloc mastoïdien à sa surface extérieure, on voit que la *zone pétreuse* peut être décomposée en deux zones secondaires, séparées par la ligne 3 : *zone pétreuse antérieure* (*a*) et *zone pétreuse postérieure* (*a'*).

La zone pétreuse antérieure (*a*) correspond à la face interne ou pétreuse du bloc mastoïdien. Elle contient l'antre et pour cela nous pouvons l'appeler aussi *zone antrale*.

La *zone pétreuse postérieure* (*a'*) correspond à la partie pétreuse de la face postéro-interne du bloc : elle est comprise entre la zone antrale et la zone sinusienne, pour cela on peut aussi l'appeler *zone intersinuso-antrale*.

La ligne 4 aboutit au bord antérieur de la gouttière sinusienne et la ligne 5 à son bord postérieur. Nous appellerons *zone sinusienne* l'espace compris entre ces deux lignes.

La ligne 6 aboutit à la suture mastoïdo-occipitale. L'espace compris entre cette ligne et la ligne 5 correspond à la portion de l'écaille mastoïdienne, qui est en rapport avec le cervelet, nous l'appellerons *zone cérébelleuse*.

La figure XVII² représente une coupe horizontale du même rocher, faite au-dessous de la précédente, et passant par la paroi inférieure du conduit auditif externe. La projection des parties profondes du bloc mastoïdien est la même. Toutefois la base de la pyramide est plus étroite à ce niveau et les zones pétreuses antérieure et postérieure y sont aussi plus étroites (*s a* et *s' a'*). Dans la zone pétreuse antérieure l'antre n'existe plus et ceci nous permettra, lorsque nous reporterons ces divisions

sur la face extérieure de la mastoïde, de distinguer une zone pétreuse antérieure et supérieure, ou *zone antrale* (*a*), et une zone pétreuse antérieure et inférieure, ou *zone sous-antrale* (*s a*).

En résumé, la projection des faces profondes du bloc mastoïdien sur la face extérieure nous permet donc de diviser celle-ci en quatre zones : zone pétreuse antérieure ; zone pétreuse postérieure ou intersinuso-antrale ; zone sinusienne et zone cérébelleuse. En traçant l'étendue de ces zones à la surface de la mastoïde, on obtient le schéma représenté dans la figure I et cela nous permet d'établir à la surface extérieure de l'os la topographie de ses parties profondes.

Il convient de remarquer que les coupes horizontales, qui passent au-dessous du conduit auditif externe, n'intéressent plus le rocher, mais seulement l'apex mastoïdien proprement dit. Nous devons donc dans cette topographie extérieure de la mastoïde établir une zone supplémentaire, que nous appellerons *zone apicale*.

Zone pétreuse antérieure. — La zone pétreuse antérieure correspond à la surface de coupe, qui limite le bloc mastoïdien du côté interne et le sépare de la pyramide. C'est pour cela que je l'appelle pétreuse ; je lui donne, en outre, le nom de *pétreuse antérieure*, parce qu'à la surface de l'os cette zone est placée en avant d'une autre que j'appellerai *pétreuse postérieure*.

La zone pétreuse antérieure est comprise entre deux lignes verticales (*a b–a′ d*) parallèles entre elles, dont l'une passe par le bord postérieur du méat auditif et dont l'autre est située à 1 cm., quelquefois à 2 cm. de la précédente. Cette différence de largeur est liée à l'épaisseur même de la pyramide. En bas, la ligne *d–b*, qui marque la base de l'apex, forme sa limite inférieure. En haut, la limite supérieure est formée par une ligne correspondant au plancher de la cavité crânienne. Cette limite est ordinairement indiquée par la racine horizontale de l'apophyse zygomatique, qui se prolonge en arrière vers l'angle de suture squamo-mastoïdo-pariétal, sous le nom de linéa temporalis. Il est de règle de considérer cette linéa comme étant la limite supérieure de trépanation mastoïdienne, au delà de laquelle on s'expose à pénétrer dans la cavité crânienne. Je me suis expliqué plus haut sur les rapports variables, qui peuvent exister entre le plancher de la cavité crânienne et la linéa temporalis. Je n'y reviendrai pas ici, me contentant de faire remarquer que sur la figure I le plancher est à 8 mm. au-dessus d'elle et que la limite supérieure de la zone pétreuse correspondant à la ligne *a′–a*. Toutefois, comme sur le vivant on ne peut savoir si la linéa-temporalis correspond, ou non, à la limite inférieure de la cavité crânienne, il faut, en cours d'opération, supposer que cette linéa marque la limite supérieure de la zone pétreuse et ne point porter la gouge au-dessus d'elle, tout au moins dans les premiers temps de la trépanation.

La zone pétreuse antérieure de la surface externe de la mastoïde peut être divisée en deux zones secondaires : *zone antrale* et *zone sous-antrale*, séparées l'une de l'autre par une ligne horizontale, passant par la

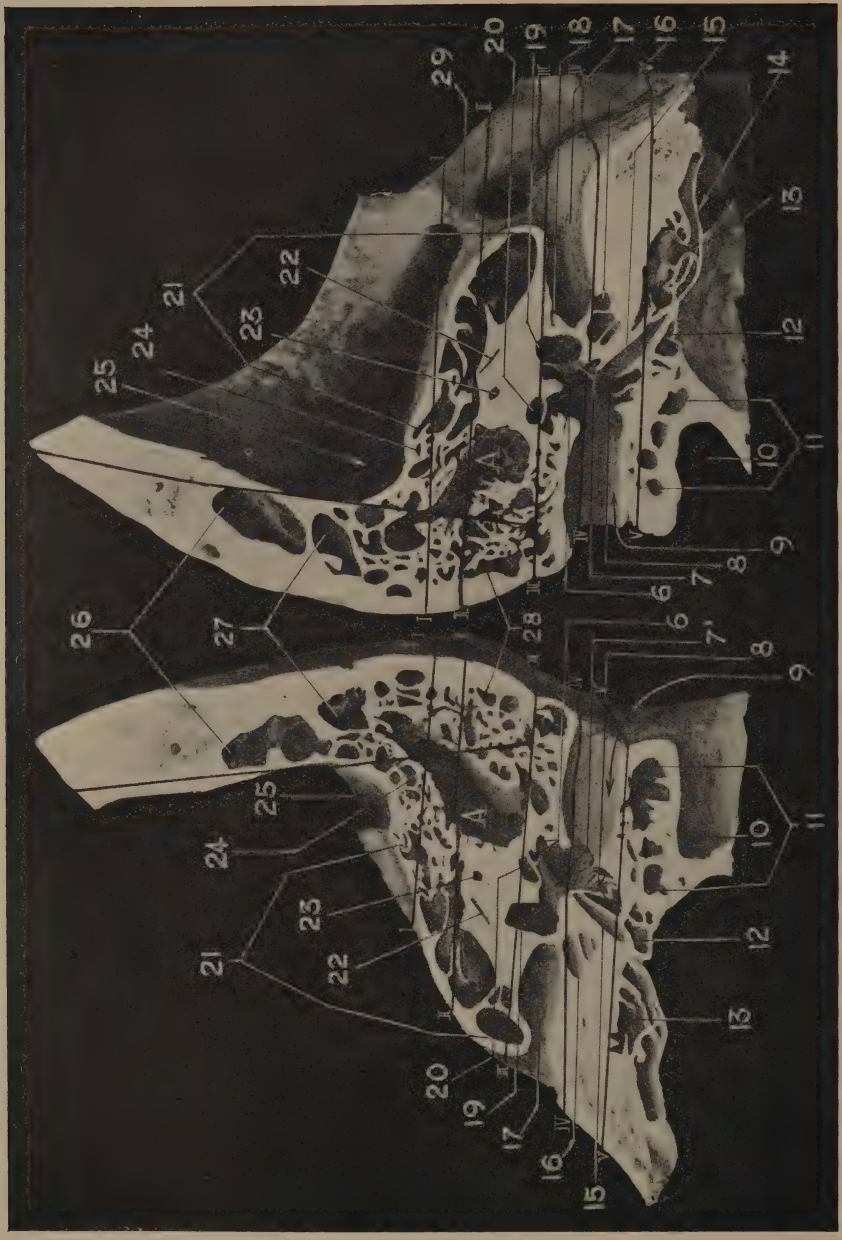

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FIGURE XVIII

Coupe horizontale d'un rocher très pneumatique, passant immédiatement au-dessous de la paroi supérieure du conduit auditif interne et du conduit auditif externe. Cette coupe est légèrement inclinée en avant. Elle est en réalité plus parallèle à la face supérieure du rocher que horizontale. Son orientation est indiquée par la ligne 6 dans la fig. IX. Cette pièce est intéressante par la grande pneumatisation du bloc mastoïdien et, aussi, par la grande richesse du rocher en cellules pneumatiques, lesquelles se prolongent, tout autour du labyrinthe, jusqu'au sommet de la pyramide pétreuse ; à gauche, se trouve la tranche supérieure de la coupe ; à droite, sa tranche inférieure.

A, antre très grand, dépassant, en arrière, la région du sinus latéral.—*C*, caisse du tympan.—I, II, III, IV, V indiquent les nombreuses sections, qui ont été faites sur cette pièce et que l'on retrouve dans le même ordre dans la fig. IX, qui la représente vue par sa face externe.—6, cellules de la paroi postérieure de la caisse.—7, bord inférieur de la fenêtre ovale.—7′, son bord supérieur.—8, cellules de la paroi antérieure de la caisse.—9, conduit auditif externe (à gauche, sa paroi supérieure ; à droite, sa paroi inférieure).—10, cavité glénoïde, ouverte par la coupe.—11, cellules situées dans la branche descendante de la racine horizontale de l'apophyse zygomatique, qui sépare la partie supérieure de la cavité glénoïde de la partie supérieure du conduit auditif externe (voir fig. IV).—12, cellules situées au-dessus de la trompe d'Eustache.—13, cellules pétreuses antérieures, situées au-dessus du canal carotidien.—14, partie horizontale du canal carotidien.—15, conduit du muscle du marteau.—16, limaçon.—17, conduit auditif interne.—18, conduit pour le nerf ampullaire postérieur.—19, vestibule.—20, aqueduc de Fallope.—21, cellules pétreuses postérieures (zone *intersinuso-auditive*).—22, aqueduc du vestibule. Il paraît très large parce qu'il est coupé près de la fente unguéale ; en réalité c'est plutôt le fond de cette fente que l'aqueduc lui-même.—23, canal demi-circulaire vertical postérieur.—24, cellules de la paroi interne de l'antre.—25, gouttière du sinus latéral.—26, cellules de la zone cérébelleuse de la région mastoïdienne.—27, cellules de la région sinusienne.—28, cellules de la région antrale (paroi externe de l'antre).

paroi postérieure du conduit, à l'union de son $\frac{1}{3}$ supérieur avec ses $\frac{2}{3}$ inférieurs.

Zone antrale. — La zone antrale forme en arrière du tiers supérieur du conduit auditif, au-dessous de la linéa temporalis, un carré d'environ $1\frac{1}{2}$ cm. de côté. Les dimensions peuvent varier, suivant que la pyramide est plus ou moins épaisse, que la linéa est plus ou moins élevée et que le sinus même est plus ou moins procident. Elle est étroite dans les mastoïdes spongieuses et compactes, plus large dans les mastoïdes très pneumatiques. Sa hauteur varie suivant que le plancher de la cavité crânienne descend plus ou moins bas. Dans la figure I elle remonte, au-dessus de la linéa temporalis, à près de 1 cm. au-dessus du bord supérieur du méat auditif. Chez d'autres sujets, au contraire, elle arrive à peine au niveau de ce bord. Dans ces cas la cavité crânienne est procidente en bas et en dehors, et la paroi supérieure du conduit auditif a, à peine, 1 mm. d'épaisseur. Cependant ces cas sont rares.

Quelquefois, et l'opérateur ne doit jamais l'oublier, la zone pétro-antrale peut être réduite à presque rien par suite d'une procidence extrême du sinus latéral en avant et en dehors. Le coude du sinus vient alors jusqu'à la limite antérieure de la zone pétreuse, au contact de la paroi postérieure du conduit auditif. Dans ces cas, surtout si la procidence du sinus est combinée avec la procidence de la cavité crânienne en bas et en dehors, la zone pétro-antrale peut être réduite à l'épaisseur de la paroi postérieure du conduit et *à une petite zone triangulaire située entre l'épine de Henle, la linéa temporalis et le bord supérieur du méat.* C'est dans ces cas, que j'ai rencontrés plusieurs fois en cours d'opération, qu'on a le plus à se louer de la trépanation antrale transspino-méatique. La procidence du sinus a été schématisée en une double ligne finement pointillée (16) sur la figure I.

Zone sous-antrale. — Cette zone est située en arrière des $\frac{2}{3}$ inférieurs du bord postérieur du méat auditif osseux, au-dessus de la ligne *d–b*, qui marque la base de l'apex, et au-dessous de la ligne *c–c'*, qui trace la limite inférieure de la zone antrale. La hauteur de la zone sous-antrale est d'environ 10 à 12 mm., sa largeur est semblable à celle de la zone antrale mais parfois un peu plus étroite à cause du sinus. Elle subit les mêmes variations qu'elle, lorsque le sinus est très procident, et peut même être réduite à zéro, lorsque le sinus arrive au contact de la paroi postérieure du conduit auditif externe. Elle correspond à la partie inférieure de la zone pétreuse de la face postéro-interne de la mastoïde (fig. XVII², *s a*). Lorsque la mastoïde est pneumatique, la zone sous-antrale est en général très cellulaire et les cellules y sont plus grandes, quelquefois même plus grandes que l'antre, pouvant alors faire croire au chirurgien non-averti qu'il s'agit de l'antre lui-même.

Dans la profondeur de la zone sous-antrale, au voisinage de l'angle, que forment, en se rencontrant, la limite antérieure et la limite interne du bloc mastoïdien, se trouve l'aqueduc de Fallope (fig. XVII², 11). En arrière de l'aqueduc, la zone sous-antrale se continue directement

avec les parties du rocher, qui sont sous-jacentes au labyrinthe. Sur la figure XVII², on voit que cette région est occupée par de grosses cellules (16) qui se prolongent, d'une part, vers le bulbe de la jugulaire (14) et, d'autre part, jusque sur la paroi postérieure de la pyramide pétreuse (zone cellulaire intersinuso-auditive, fig. VI). Dans des cas semblables, la région sous-antrale permettrait d'introduire profondément une curette au-dessous du labyrinthe et d'aller ouvrir les cellules suppurées, soit au-dessus et autour du bulbe, soit dans la fosse cérébelleuse. Mais il ne faut suivre cette voie qu'avec la plus extrême prudence : on passe très près du facial et l'on ne sait si l'on ne tomberait pas sur un bulbe procident (fig. II et fig. XVI).

Zone pétreuse postérieure. — J'appelle ainsi une surface triangulaire située en arrière de la zone pétreuse antérieure et de la zone sinusienne : pour cela on peut aussi l'appeler *zone intersinuso-antrale*. Le sommet, *d*, du triangle est dirigé en bas et correspond au point de rencontre des lignes *e–d* (bord antérieur du sinus) et *a'–d* (bord postérieur des zones antrale et sous-antrale) sur la ligne *b-b'* (limite de la base de l'apex). Sa base, *e–a'*, correspond au plancher de la cavité crânienne et est sur le prolongement de la limite supérieure de la zone antrale. Comme celle-ci, elle correspond, en principe, à la linéa temporalis ; mais elle peut, aussi, être soit au-dessus, soit au-dessous d'elle. Son bord postérieur, *e–d*, est oblique de haut en bas et d'arrière en avant, et correspond à la ligne *pariéto-apicale*, que j'ai déjà indiquée plus haut et qui s'étend de l'angle pariéto-squamo-mastoïdien au milieu du sommet de l'apex. Cette ligne, dans notre classification topographique de la surface extérieure de la mastoïde, s'arrête à la base de l'apex, au point *d* de la ligne *b-b'*. La ligne pariéto-apicale trace la limite entre la zone sinusienne et la zone pétreuse postérieure. Elle correspond aussi, en général, à la suture pétro-squameuse externe. Si l'on considère les rapports de la zone pétreuse postérieure de la face externe de la mastoïde avec les parties profondes (figs. XVII ¹ et XVII ²), on voit qu'elle correspond à la partie de la face postérieure du rocher, qui se trouve immédiatement en avant de la gouttière du sinus latéral et que j'ai appelée zone pétreuse de la face postéro-interne du bloc mastoïdien. Les deux zones correspondent exactement entre elles. L'épaisseur de cette région forme un prisme osseux (pneumatique, spongieux ou compact, suivant la variété de l'os), compris entre la zone sinusienne et les zones antrale et sous-antrale. Elle mesure 1 cm. au niveau de son bord supérieur, mais elle peut être réduite à presque rien, lorsque la pyramide pétreuse est très étroite. C'est de toutes les zones mastoïdiennes celle qui est la plus variable dans ses rapports soit avec l'antre, soit avec le sinus. Déjà, à propos de la suture pétro-squamo-externe, j'ai fait remarquer que, dans certains cas, cette suture est située en arrière de la ligne pariéto-apicale, qui correspond au bord postérieur du triangle, qui représente la zone pétreuse postérieure. De même l'antre, lorsqu'il est volumineux (fig. IX), empiète sur cette zone. Le sinus procident dépasse aussi sa limite postérieure et peut l'occuper toute

entière, surtout lorsque sa procidence est très grande (fig. I, 15, 16). La zone pétreuse postérieure ou zone intersinuso-antrale est donc très variable dans son étendue, dans sa constitution et dans ses rapports ; cela tient : 1° à l'épaisseur de la base de la pyramide, 2° à la position procidente ou non du sinus, 3° au volume de l'antre.

Zone sinusienne. — Cette zone se compose de deux parties : une supérieure et horizontale, l'autre descendante, deux fois plus étendue que la précédente. La partie supérieure (fig. I, 13, S_1) correspond à la portion terminale de la partie horizontale du sinus latéral. La portion descendante S_3 correspond à la moitié supérieure de la portion descendante du sinus latéral (portion descendante du sinus sigmoïde). La partie terminale du sinus latéral (qui forme la portion horizontale du sinus sigmoïde), ne peut être reproduite sur la face externe de la mastoïde, parce qu'elle abandonne tout rapport direct avec elle et qu'elle se dirige en dedans et un peu en avant, courant au-dessus de la face inférieure du bloc mastoïdien (région digastrique de la mastoïde) (fig. VIII). La partie horizontale de la région sinusienne occupe le tiers supérieur de la partie la plus reculée de la mastoïde, depuis la suture mastoïdo-occipitale jusqu'au niveau de l'angle de suture pariéto-squamo-mastoïdien. Sa longueur est de $1\frac{1}{2}$ à 2 cm. et sa hauteur de 1 cm. environ : la limite inférieure correspond à une ligne horizontale passant par le bord supérieur du méat auditif externe. La partie descendante s'étend derrière la ligne pariéto-apicale, jusqu'au milieu de la base de l'apex. La longueur de la portion descendante est de 3 à 4 cm. Sa largeur est aussi de 1 cm. environ. Longueur et largeur varient peu, mais suivent les variations de hauteur de la mastoïde et de volume du sinus latéral. L'angle que forment les deux portions sinusiennes correspond au coude du sinus latéral (S_2).

La figure, que donne la projection de la partie descendante du sinus à la surface externe de la mastoïde, est très variable suivant les individus. Cela tient : 1° à l'épaisseur de la base de la pyramide, 2° aux rapports que le sinus affecte envers elle.

Plus la base de la pyramide est épaisse, plus la région sinusienne est éloignée du conduit auditif et plus, aussi, est large la région pétreuse, c'est-à-dire la région opératoire. La distance, qui sépare la région sinusienne de l'épine de Henle, peut être de plus de 2 cm. Si le volume de la base de la pyramide est le même en haut et en bas, le sinus descend presque verticalement (figs. IX et X). Si, au contraire, la partie supérieure de la base de la pyramide est plus large que la partie inférieure, le sinus descend obliquement en bas et en avant, comme dans la figure I, S_3. Si la pyramide est petite et sa base étroite la région sinusienne est plus rapprochée du conduit, les zones pétreuses sont alors très étroites et peuvent n'avoir dans leur ensemble qu'un centimètre de largeur. Dans les mastoïdes d'enfant, les os étant plus petits que chez l'adulte, la région pétreuse est moins large, et la région sinusienne est moins éloignée du méat auditif. Le maximum d'éloignement entre le méat auditif et la

zone sinusienne se trouve dans les mastoïdes volumineuses et très pneumatiques de l'adulte.

A côté de ces différences de direction et de situation de la zone sinusienne descendante, qui tiennent au volume même de l'os, il y en a de plus importantes et de beaucoup plus imprévues, qui tiennent aux rapports intimes du sinus avec la pyramide.

La portion descendante du sinus latéral correspond en général à l'angle intra-crânien, que forme la face postérieure de la pyramide avec la portion écailleuse de la mastoïde. Le sinus peut subir deux genres de déplacement, qui, tous deux, le portent plus en avant. Dans l'un il se porte en avant et en dedans : c'est une sorte de procidence antéro-interne, dans laquelle le sinus se porte sur la face postérieure elle-même de la pyramide et se place sur la zone rétro-pétreuse de la face crânienne du bloc mastoïdien. Dans cette position le sinus correspond à la paroi profonde de l'antre. Ce genre de procidence n'offre d'intérêt que lorsqu'il s'agit de pénétrer dans la fosse cérébelleuse à travers la paroi interne de l'antre. Dans l'autre, le sinus se porte en avant et en dehors. Cette procidence est beaucoup plus importante à connaître, au point de vue de la trépanation mastoïdienne. Dans ce cas, le coude du sinus et toute sa partie descendante proéminent en avant et la gouttière sinusienne forme un large et très profond sillon, creusé à la base de la pyramide (fig. XIII). Les zones pétreuses sont excessivement réduites et cette disposition coïncide toujours avec une mastoïde petite et un rocher étroit. La couche osseuse, qui sépare la surface de la mastoïde du sinus procident est peu épaisse et peut même être réduite à une simple lamelle osseuse ayant seulement 2 ou 3 mm. d'épaisseur. Dans les formes peu accentuées le sinus s'interpose seulement entre la surface de l'os et la région triangulaire, que j'appelle zone pétreuse postérieure (fig. I, 15). Dans la forme plus procidente, le sinus occupe toute la zone pétreuse antérieure (antrale et sous-antrale) et arrive, dans les formes extrêmes, jusqu'au contact de la paroi postérieure du conduit (figs. I, 16 ; XIII, XIX). Le sinus ainsi incurvé en avant, occupe la place de l'antre, mais celui-ci ne disparaît pas pour cela : tout petit, il se cantonne, dans la région correspondant à la spina-supra-méatum, au niveau de l'angle supéro-postérieur du méat. Rien ne peut faire prévoir un tel bouleversement dans la disposition des zones mastoïdiennes. C'est une des principales raisons, pour lesquelles il y a grand avantage à établir la surface d'attaque de la trépanation antrale au niveau de l'épine de Henle, comme je l'ai dit plus haut.

Lorsque le sinus est procident, la mastoïde est peu ou même pas du tout pneumatique et la couche osseuse qui le sépare des téguments extérieurs est très mince. Lorsque le sinus est en position normale, on peut, au contraire, si la mastoïde est pneumatique, trouver dans la région sinusienne des cellules très nombreuses et souvent même très grandes (figs. V et VI), qui en cours de suppuration se vident mal du pus qu'elles contiennent. La corticale intra-crânienne de la région sinusienne est, en général, beaucoup plus mince que la corticale extra-crânienne. Ces deux

particularités réunies : cellules grandes, écoulant mal le pus qu'elles renferment, et corticale intra-crânienne mince sont deux conditions anatomiques, qui favorisent la rétention purulente et la nécrose osseuse. Aussi, dans les mastoïdes pneumatiques, la gouttière du sinus latéral est-elle, surtout au cours des mastoïdes aiguës, un siège fréquent de mortification osseuse.

Zone cérébelleuse. — La zone cérébelleuse occupe les $\frac{2}{3}$ inférieurs de la partie la plus reculée de la région mastoïdienne, au-dessous et en arrière de la région sinusienne, qui l'entoure de son coude (fig. I, *C*). La partie horizontale de celle-ci empêche cette zone de remonter, en haut, jusqu'à la suture mastoïdo-pariétale. Elle est limitée en arrière par la suture mastoïdo-occipitale postérieure (18). En bas elle est limitée par la ligne horizontale *b-b'*, qui marque la base de l'apex mastoïdien et qui, en arrière de celui-ci, se prolonge entre la zone cérébelleuse de la face externe de la mastoïde et le bord postéro-externe de la région digastrique (face inférieure de la mastoïde, 8). La zone cérébelleuse a 2 cm. de hauteur et 1 cm. de largeur. L'orifice du canal, que traverse la grande veine mastoïdienne pour aller au sinus latéral, occupe presque toujours cette zone et j'en ai, plus haut, indiqué la variabilité de situation.

Cette zone de la mastoïde est très souvent mince, écailleuse et pour cela certains auteurs la décrivent sous le nom d'écaille de la mastoïde. Cependant, il n'est pas exceptionnel de la trouver, aussi, creusée de grandes cellules (fig. V, 14), surtout lorsque la mastoïde appartient au type pneumatique.

Elle est séparée du méat auditif par les zones pétreuses et par la zone sinusienne. La distance qui l'en sépare varie avec la largeur même de celles-ci, c'est-à-dire avec l'épaisseur même du bloc mastoïdien. En général, pour marquer la place de cette zone sur le sujet recouvert de ses téguments, il suffit de placer l'index en arrière et au-dessus du bord postérieur de l'apex, qui est toujours assez facile à repérer à l'état normal. Mais, si les téguments de toute la région mastoïdienne sont tuméfiés et s'il n'est pas bien possible de reconnaître les bords de l'apex, on peut admettre que la région cérébelleuse de la face externe de la mastoïde est située à deux travers de doigt du conduit auditif externe. Il est utile de savoir bien repérer cette région, car, au cours d'une endomastoïdite, les douleurs provoquées par l'ostéite ou la périostite, peuvent, parfois, être localisées dans la zone cérébelleuse, alors que les régions antrale et apicale paraissent complètement indolores. Il faut savoir, en effet, que la douleur à la pression, que l'on recherche toujours avec soin dans l'endomastoïdite, comme signe de lésion ostéo-périostique antrale ou apicale, peut siéger en toute autre région que celles de l'antre et de l'apex. Si les cellules sont développées dans la zone cérébelleuse comme dans la figure V, la douleur spontanée ou provoquée par la pression digitale sur cette zone peut être la conséquence d'une rétention purulente dans l'une de ces cellules. Mais l'expérience montre aussi, j'en ai observé plusieurs cas, qu'au cours d'une endomastoïdite légère et sans mani-

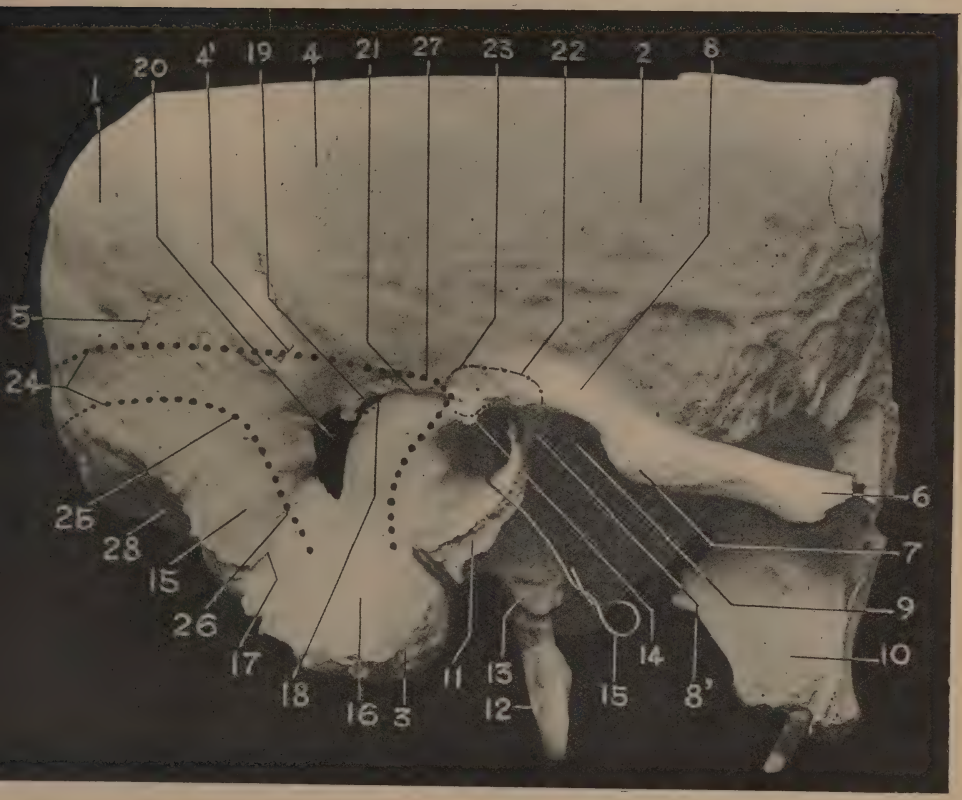


FIGURE XIX

1, pariétal.—2, écaille du temporal.—3, sommet de l'apex.—4, suture squamo-pariétale.—4′, angle squamo-mastoïdo-pariétal.—5, suture mastoïdo-pariétale.—6 apophyse zygomatique.—7, racine transverse de cette apophyse.—8, sa racine horizontale.—8′, branche descendante de la racine horizontale.—9, cavité glénoïde.—10, aile externe de l'apophyse ptérygoïde.—11, bord inférieur du méat auditif externe (os tympanal).—12, apophyse styloïde.—13, apophyse vaginale.—14, spina-supra-méatum.—15, fil de laiton passant dans la caisse et dans l'antre et ressortant sur la face postérieure du rocher (fig. XX, 14), à travers la déhiscence pétreuse.—16, surface de l'apex, dépourvue de l'aile squamo-mastoïdienne.—17, sillon.—18, lèvre pétreuse de la déhiscence.—19, lèvre squameuse de la déhiscence.—20, déhiscence pétro-squameuse.—21, sillon prolongeant la fente vers la spina-supra-méatum.—22, ligne représentant la projection de l'attique à la surface de l'os.—23, ligne représentant, de même, la projection de l'antre, qui est ouvert en arrière, au niveau du *bulbe supérieur* du sinus.—24, projection du sinus latéral.—25, son coude.—26, sa portion descendante.—27, son *bulbe supérieur* procident en avant.

festation douloureuse ni du côté de l'antre ni du côté de l'apex, les germes infectieux peuvent encore porter leur action malfaisante dans l'écaille de la mastoïde, même en l'absence de toute cavité pneumatique dans cette région, et y produire des points d'ostéite, dont le voisinage est grave pour l'endocrâne et pour la veine mastoïdienne. Il convient de bien connaître ces particularités des infections mastoïdiennes, qui doivent avertir le chirurgien que la douleur localisée à la zone cérébelleuse de la mastoïde a une valeur aussi importante, si ce n'est davantage, que celle des régions antrale et apicale. Il importe donc de savoir bien repérer la situation de cette zone. Les douleurs, qui siègent à deux travers de doigt du méat auditif externe, indiquent des lésions de la zone cérébelleuse de la mastoïde, lorsqu'elles sont au-dessus de la ligne horizontale *b–b'*, qui marque la base de l'apex. Celles qui, étant à la même distance du méat, siègent au-dessous de cette ligne, correspondent à des lésions de la face inférieure ou digastrique.

La surface rugueuse (fig. XI, 10), qui va du bord supérieur de l'écaille de la mastoïde, au sommet de l'apex et sur laquelle s'insèrent les muscles mastoïdiens, recouvre en grande partie la zone cérébelleuse (fig. I). On est obligé de ruginer ces insertions musculaires pour opérer dans cette région. La présence de ces insertions musculaires à la surface de la zone cérébelleuse donne aux périostites de cette région une allure particulière. Les lésions de la surface externe de cette zone donnent rarement lieu à des abcès sous-périostés, la périostite y prend plutôt une forme ' lardacée' et l'infection, qui sort de l'écaille de la mastoïde, a tendance à diffuser vers le cou, suivant les muscles mastoïdiens, donnant ainsi une variété de mastoïdite postérieure à évolution cervicale, dans le genre dit mastoïdite de Bezold.

Région de l'Apex. — La région de l'apex occupe la face externe de l'apophyse proprement dite, au-dessous de la ligne horizontale *b–b'*, qui passe par le bord inférieur du méat auditif et qui marque la limite inférieure des zones pétreuse et sinusienne. Cette zone n'a de rapports ni avec la cavité crânienne, ni avec la base de la pyramide, puisque l'apex se détache du bloc mastoïdien en formant une sorte de ' malléole ', qui protège la face inférieure de la mastoïde et dont la face interne fait partie de la région digastrique. Elle donne des insertions puissantes au sterno-cléïdo-mastoïdien, que la rugine a quelques difficultés à détacher. La suture pétro-squameuse externe s'arrête, en général, au-dessus de cette zone ; quelquefois cependant elle descend jusqu'à son sommet (figs. I, XI). L'apex peut être spongieux ou pneumatique. Dans ce dernier cas, les cellules qu'elle contient sont très souvent les plus grandes de toute la région mastoïdienne. Le grand volume de ces cellules, leur éloignement de l'antre et leur déclivité naturelle sont trois facteurs réunis, qui concourent à la stagnation du pus en cette région et favorisent la nécrose de l'apex. Mais celle-ci se fait rarement sur sa face externe. Presque toujours la perforation de l'apex par la suppuration se fait sur sa face profonde (face digastrique), car la corticale osseuse est très mince de ce

côté, tandis qu'elle est beaucoup plus épaisse en dehors. La nécrose de la face interne de l'apex fait sortir le pus endomastoïdien à la face inférieure de la mastoïde, dans la région digastrique, et prédispose aux abcès, qui diffusent dans le cou (abcès de Bezold).

Dans les suppurations endoapicales, l'ouverture large de l'apex s'impose, car ses grandes cellules forment de véritables puits, qui ne peuvent se vider librement. Dans les premiers temps de la chirurgie mastoïdienne, certains opérateurs ont espéré que la seule trépanation de cette partie de l'os pouvait garantir la guérison des mastoïdites, en assurant l'écoulement du pus par la partie la plus déclive de la région. C'est une erreur depuis longtemps reconnue.

Au cours d'une otite, les douleurs spontanées ou provoquées par la pression digitale sur l'apex sont un signe d'endomastoïdite, mais ce signe n'existe que quand l'apex est pneumatique ; son absence ne doit pas faire écarter le diagnostic d'infection endomastoïdienne, car, si l'apex n'est pas pneumatique, la suppuration l'envahit difficilement, mais celle-ci peut porter son action sur les autres parties de la surface extérieure, ou bien dans la profondeur de l'os.

L'ablation totale de l'apex est nécessaire lorsqu'on veut aborder largement la face inférieure de la mastoïde.

La division de la surface extérieure de la mastoïde en zones secondaires est utile pour bien déterminer le siège des lésions, que cette région peut présenter ; elle est surtout utile pour orienter le champ des interventions chirurgicales. Il convient donc que chacun la connaisse bien. Mais, ce qu'il faut savoir aussi, c'est que ce schéma ne s'applique qu'à la mastoïde normale, car les variations de volume, de structure et des rapports des zones mastoïdiennes avec les parties profondes sont tellement fréquentes que le chirurgien ne doit compter avec le schéma, que je viens de donner, que comme un moyen de se repérer facilement, lorsqu'il est en face de cas compliqués. L'opérateur doit toujours être prudent et agir comme s'il doit se trouver en présence d'un cas difficile. Au moment de trépaner la mastoïde, il faut toujours prendre les cinq points de repère suivants : 1° le bord postérieur du méat auditif, 2° son bord supérieur, 3° son angle postéro-supérieur, avec la spina-supra-méatum et la fossette criblée, qui sont le plus souvent à ce niveau, 4° la linéa temporalis, 5° la ligne oblique (sinusienne) pariéto-apicale.

Ces deux derniers sont trompeurs : la ligne sinusienne est des plus variables et la linéa temporalis est loin d'indiquer toujours la situation exacte du plancher de la cavité crânienne. Elles doivent seulement faire redoubler d'attention, lorsque, la trépanation étant déjà faite, on est obligé d'élargir le champ d'opération en haut ou en arrière. Mais le point délicat de la trépanation mastoïdienne n'est pas tant d'agrandir la brèche osseuse que de la commencer sûrement, sans léser le sinus, et d'aller directement et sûrement à l'antre par le plus court chemin. Le volume de cette *antichambre*, qui commande les *appartements* mastoïdiens, est très variable et est lié, le plus souvent, à la structure même de l'os,

ainsi qu'à la situation du sinus, que rien ne peut au préalable faire prévoir.

Or, des cinq points de repère que je viens de mentionner : le *bord supérieur*, le *bord postérieur* et l'*angle postéro-supérieur* du méat auditif sont les seuls qui donnent de la précision. Le milieu de la paroi externe d'un antre de volume moyen fait face à cet angle (fig. XIV). En faisant passer la trépanation par l'angle supéro-postérieur du méat auditif, on va à l'antre par le plus court chemin.

Le bord supérieur de la spina se trouve, en général, sur un même plan horizontal que le plancher de l'aditus : or, en comprenant dans le champ de trépanation un peu (3 mm.) de l'espace situé au-dessus de l'épine, on ne peut manquer d'arriver dans l'antre, si haut placé qu'il soit, car, même dans la position la plus élevée, son plancher n'est jamais plus haut placé que celui de l'aditus. Cependant comme la position et la forme de la spina peuvent varier légèrement d'un sujet à l'autre, il vaut encore mieux ne se repérer exactement que sur l'angle postéro-supérieur de méat auditif.

Modalités et Anomalies de la Région mastoïdienne

La forme générale de la région mastoïdienne est toujours la même, mais les différentes parties qui la constituent peuvent avoir une conformation extérieure et une structure intérieure très variables, suivant les individus. Chaque zone du bloc mastoïdien peut présenter des changements individuels, suivant que l'os est petit ou volumineux, qu'il est compact ou pneumatique, que les parties osseuses, qui le constituent, se sont développées régulièrement ou de façon anormale. Les rapports, que chaque zone présente avec les parties intérieures de l'os (antre, cellules) ou avec les parties intra-crâniennes (méninges, sinus) peuvent, par suite, subir des modifications assez importantes, que le chirurgien doit connaître. Sans vouloir entrer dans des détails, qui pour la plupart ont été déjà traités en leur place dans ce rapport, je résumerai seulement les parties essentielles.

Le bloc mastoïdien peut être volumineux ou petit. L'apex peut être très prononcé ou à peine ébauché.

La rainure digastrique peut n'être pas seulement marquée à la face inférieure de la région mastoïdienne, mais peut se prolonger aussi sur sa face externe : dans la pièce représentée dans la figure XV, elle remontait au-dessus d'une ligne horizontale passant par le bord supérieur du méat auditif, c'est pour cela que sur la coupe elle se présente sous forme d'une encoche très accentuée (1), bien que cette coupe horizontale ait été faite au niveau de la spina-supra-méatum.

La spina-supra-méatum peut manquer ou se présenter soit sous forme d'épine, soit sous forme de crête (fig. X), pouvant avoir plus de 1 cm. de longueur. Elle siège, le plus souvent, au niveau de l'angle postéro-supérieur du méat auditif. Elle peut aussi s'étendre, à la fois, sur la

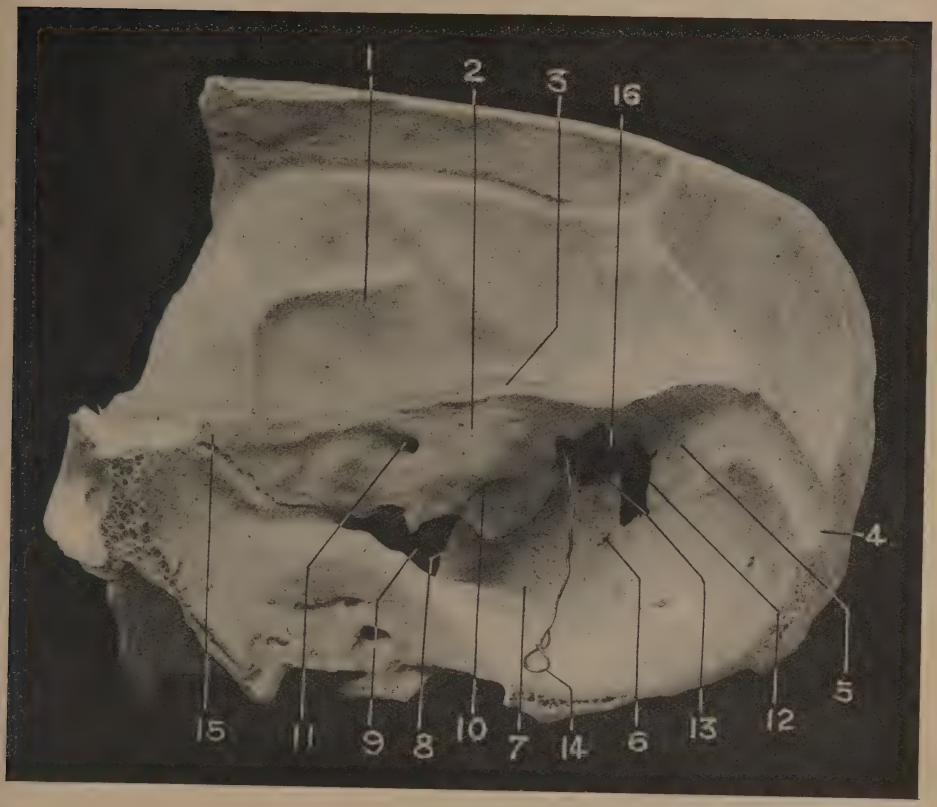


FIGURE XX

1, face interne de l'écaille du temporal.—2, face postérieure du rocher.—3, sa face supérieure, vue très obliquement et en raccourci.—4, partie horizontale de la gouttière du sinus latéral.—5, son coude.—6, sa partie descendante.—7, sa partie terminale.—8, crête s'élevant à l'extrémité terminale de cette gouttière, au point où le sinus arrive dans le trou déchiré postérieur.—9, trou déchiré postérieur.—10, fente unguéale.—11, trou auditif interne.—12, déhiscence quadrangulaire faisant communiquer la gouttière sinusienne avec la surface extérieure de la mastoïde.—13, déhiscence polygonale, sinuso-pétreuse, conduisant dans une large cavité, qui est creusée dans l'épaisseur de la portion pétreuse (base du rocher) du bloc mastoïdien. A sa partie profonde et antérieure cette cavité communique avec l'antre.—14, fil de laiton passant par la déhiscence polygonale, sinuso-pétreuse, traversant la cavité qui est creusée dans la base du rocher, traversant aussi l'antre, l'aditus, la caisse, le conduit auditif externe et ressortant à l'extérieur par le méat auditif (fig. XIX, 15).—15, sommet du rocher.—16, partie supérieure de la paroi externe de la cavité intra-pétreuse ; à ce niveau correspond la fente (18–19) qui prolonge l'angle supéro-antérieur de la déhiscence quadrangulaire, que l'on voit sur la face externe de la pièce (fig. XIX).

plus grande partie de son bord supérieur et de son bord postérieur, ou bien, encore, seulement sur l'un ou sur l'autre de ces bords. La fossette criblée, rétro-spinale, peut présenter des variations semblables.

La dépression de la zone antrale, sur la face extérieure de la mastoïde, peut être très accentuée et facilement reconnue, sur le vivant, elle peut aussi ne pas exister.

Le sillon de la suture pétro-squameuse externe offre des variétés plus grandes encore de situation, d'étendue et de profondeur.

Les orifices extérieurs des canaux, qui laissent passer les veines mastoïdiennes, varient de nombre et de situation : tantôt placés à la partie supérieure de la zone cérébelleuse, tantôt à sa partie inférieure.

La gouttière du sinus latéral peut être superficielle ou profondément creusée dans l'os, s'étaler sur la face postérieure du rocher ou s'enfoncer dans sa base.

Les faces pétreuses intra-crâniennes du bloc mastoïdien peuvent être aplaties, excavées ou convexes. Cela tient principalement au volume même de l'os et à sa structure intérieure.

Le volume du bloc mastoïdien est en rapport avec le volume général du squelette et avec la richesse de pneumatisation propre à chaque individu. Une mastoïde est, en général, d'autant plus volumineuse que le squelette est de grande taille, mais, toutes proportions gardées, ce volume est surtout en rapport avec la pneumatisation intérieure de l'os. Plus une mastoïde est riche en cellules, plus elle est volumineuse et semble être distendue par les cavités pneumatiques qu'elle contient. Une mastoïde très pneumatique présente une surface extérieure plus large et plus convexe que celle d'une mastoïde spongieuse ou compacte. La face postérieure du rocher (partie pétreuse de la face postéro-interne du bloc mastoïdien) est convexe lorsque l'os est pneumatique, plate ou excavée lorsque l'os est simplement spongieux ou compact.

Lorsque, chez l'adulte, on se trouve en présence d'une mastoïde volumineuse, on peut s'attendre à trouver un antre grand et des cellules nombreuses. Lorsqu'on se trouve, au contraire, en présence d'une mastoïde petite, on peut prévoir qu'on aura affaire à un os dur, compact et à un antre petit et haut placé, peut-être aussi à un sinus procident. Dans ces cas, le chirurgien doit rétrécir son champ d'opération, redoubler d'attention dans la recherche de l'antre et prendre toutes les précautions pour ne pas léser le sinus ou le facial.

Le plus souvent on n'a à compter qu'avec ces variations de volume et de rapports de la mastoïde, mais la région mastoïdienne peut aussi présenter des malformations, qui peuvent rendre très dangereuse toute intervention chirurgicale et qui peuvent aussi avoir une grande influence sur la marche des suppurations mastoïdiennes. Ces malformations tiennent à un développement anormal des parties osseuses, qui constituent la mastoïde, et qui, primitivement, chez le fœtus, sont indépendantes les unes des autres. La partie postérieure de l'os tympanal, qui forme le revêtement superficiel de la paroi postérieure du conduit (face antérieure

du bloc mastoïdien), l'aile squamo-mastoïdienne de l'os écailleux, qui forme la paroi externe de l'antre, la base du rocher, qui forme la partie essentielle du bloc mastoïdien, peuvent subir un arrêt de développement plus ou moins marqué, qui modifie plus ou moins, aussi, la partie correspondante de la région mastoïdienne.

La plus fréquente de ces anomalies est celle qui porte sur l'arc postérieur de l'os tympanal. Assez souvent cet arc n'est complet qu'au niveau même du cadre tympanal, dans lequel s'enchâsse la membrane du tympan : en dehors du cadre, sur toute la longueur de la paroi postérieure du conduit, l'arc tympanal peut ne s'élever qu'à quelques millimètres au-dessus de la paroi inférieure. Il en résulte que la moitié supérieure de la paroi postérieure du conduit est alors uniquement formée par l'os pétreux, non-doublé de l'arc tympanal : la corticale de la face antérieure du bloc mastoïdien se trouve amincie et les cellules, dites limitrophes du conduit, ne sont alors séparées du périoste que par une corticale excessivement mince. Cette disposition favorise beaucoup la formation des lésions de la paroi postérieure du conduit au cours des mastoïdites (périostites, fistules) ; en revanche, elle facilite la trépanation transspino-méatique, en rendant moins épaisses les parties osseuses que le ciseau doit traverser.

L'aile squamo-mastoïdienne, qui forme la paroi externe de l'antre, recouvre, en principe, toute la partie de la face externe de la mastoïde, qui est située en avant de la ligne pariéto-apicale ; elle peut même s'étendre un peu plus loin, en arrière ; mais assez souvent son développement est plus réduit et elle ne dépasse pas une ligne oblique, allant de l'angle squamo-mastoïdo-pariétal au bord inférieur du méat auditif. Cette variabilité d'étendue de l'aile squamo-mastoïdienne donne à la suture pétro-squameuse externe ou aux fissures, encoches, trous, qui en représentent les vestiges, des dispositions assez différentes suivant les sujets. Mais cela ne paraît pas avoir grande importance, tant que l'aile squamo-mastoïdienne recouvre bien la région antrale.

Il en est tout autrement, si l'aile squamo-mastoïdienne reste rudimentaire. Dans ces cas la conformation extérieure de la mastoïde peut être profondément troublée ; sa surface extérieure est aplatie et semble évidée, comme à la suite d'une large trépanation, dans laquelle on aurait enlevé les plans superficiels de l'os.

La partie de l'os pétreux, qui prend part à la constitution de la région mastoïdienne, peut, elle aussi, présenter des anomalies, qui sont dues à des troubles de l'ossification au cours du développement de la région mastoïdienne.

Les déhiscences, que j'ai décrites sous le nom de ' déhiscences en trou de mite ' peuvent se présenter sur toutes les parties de la corticale périphérique de l'os temporal. Mais on les trouve plus particulièrement sur la corticale endocrânienne, soit dans la gouttière du sinus latéral, soit, plus souvent encore, à la face supérieure du rocher, le long de la suture pétro-squameuse interne et le long du toit de la trompe d'Eustache,

de la caisse, de l'aditus et de l'antre. La pièce n° XXI est un curieux spécimen de cet ordre. Toutefois ces troubles d'ossification sont superficiels et n'intéressent pas la profondeur même du rocher.

Mais, à côté de ces troubles d'ossification, qui ne portent que sur la corticale périphérique de l'os, il y a des anomalies plus importantes, dans lesquelles l'os a subi un arrêt de développement tellement grand que ses parties centrales, elles-mêmes, manquent et permettent à la cavité crânienne de communiquer soit avec les cavités accessoires (cellules, antre, caisse), soit avec l'extérieur. Ces déhiscences osseuses ne sont, en réalité, en communication directe avec ces parties que sur le squelette sec, car, sur le sujet recouvert des parties molles, la muqueuse des cavités aériennes d'une part, les téguments extérieurs (périoste, peau) d'autre part, forment un mur de séparation entre les deux. Néanmoins, ce mur de séparation est bien peu de chose. Aussi dans des cas semblables, les complications intra-crâniennes sont bien à redouter, qu'elles soient la conséquence d'un état pathologique des cavités pneumatiques, ou qu'elles soient le résultat d'une intervention chirurgicale.

La pièce, qui est représentée dans les figures XIX et XX, est un spécimen remarquable des anomalies de ce genre. Elle appartient à la collection du professeur Brühl, de Berlin, qui a bien voulu me la confier pour en prendre la photographie.

Cette pièce présente une série d'anomalies très complexes : 1° La surface extérieure de la mastoïde est comme évidée ; 2° elle présente une large perforation, qui fait communiquer la gouttière du sinus latéral avec l'extérieur ; 3° une autre perforation occupe la paroi postérieure du rocher et tout particulièrement la partie de cette paroi, que j'ai décrite, comme faisant partie du bloc mastoïdien, sous le nom de zone pétreuse de la face postéro-interne de la région mastoïdienne ; 4° l'antre est tout petit et haut placé ; 5° le sinus latéral était procident en deux régions distinctes : (*a*) en haut, au niveau du coude, (*b*) en bas, au niveau de son abouchement dans le bulbe de la jugulaire. Je décrirai d'abord chacune de ces anomalies et j'essaierai ensuite d'en donner l'explication.

Face extérieure (fig. XIX). — Au lieu d'être, comme d'habitude, légèrement convexe, la surface extérieure de la région mastoïdienne est creuse et comme évidée. A un centimètre en arrière du méat auditif externe se trouve une grande perforation osseuse (20), qui fait communiquer la surface de l'os avec la cavité crânienne, au niveau de la partie descendante de la gouttière du sinus latéral, un peu au-dessous de son coude. Cette perforation a la forme générale d'un quadrilatère. On peut lui distinguer quatre bords et quatre angles. Elle a 11 mm. de haut et 5 mm. de large. Au niveau de son angle supéro-antérieur, elle se prolonge en haut et en avant, en forme de fente (19), sur une longueur de 7 mm. Celle-ci va en se rétrécissant et se continue avec un sillon, très accentué à la surface de l'os, qui prolonge la fente jusqu'à la spina-supra-méatum. La partie quadrilatère de la perforation se voit sur la face endocrânienne

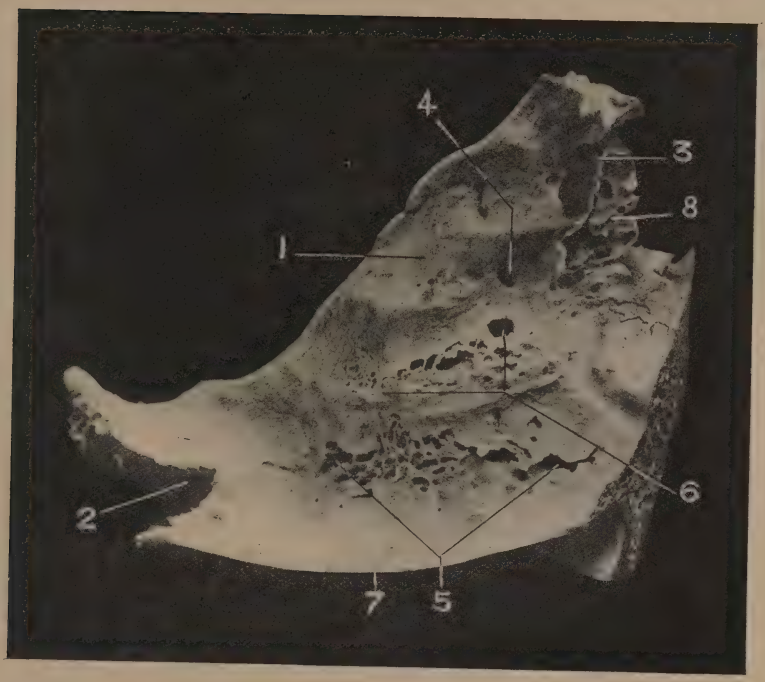


FIGURE XXI

Cette pièce appartient à la collection du professeur Brühl, de Berlin. Le rocher est vu par sa face supérieure, qui présente de nombreuses déhiscences de la corticale, dans la région voisine de l'angle intra-crânien, squamo-pétreux, le long de la ligne de suture pétro-squameuse interne, et aussi le long du toit de la caisse et de l'antre. Ces déhiscences sont dues à une insuffisance de l'ossification de la corticale osseuse intra-crânienne et appartiennent au genre des déhiscences que j'ai appelées en 'trou de mite' (voir 'Déhiscences intra-crâniennes des cavités de l'oreille et déhiscences de l'aqueduc de Fallope,' *Bulletins et Mémoires de la Société française d'oto-rhino-laryngologie*, 1910, et *Revue hebdomadaire d'oto-rhino-laryngologie*, 1910).

1, face supérieure du rocher.—2, angle squamo-mastoïdo-pariétal.—3, canal carotidien.—4, hiatus de Fallope.—5, déhiscences échelonnées le long de l'angle pétro-squameux de la face supérieure du rocher.—6, déhiscences du toit de la caisse, de l'aditus et de l'antre.—7, écaille du temporal en partie réséquée et vue en raccourci.—8, cellules pétreuses péri-carotidiennes.

(fig. XX, 12). La fente (19) fait communiquer la surface extérieure de l'os avec une cavité, qui est creusée dans l'épaisseur de la base de la pyramide pétreuse et qui est elle-même largement déhiscente dans la cavité crânienne (fig. XX, 16). Les bords de la perforation sont très minces, mais cette minceur est extrême le long du bord antérieur : toute la partie osseuse, qui est comprise entre ce bord et le méat auditif, est comme papyracée.

La surface osseuse, qui est au-dessus et en arrière de la déhiscence, paraît d'aspect normal, bien que l'ensemble de l'os soit aussi très mince. De l'angle postéro-inférieur de la perforation part un sillon superficiel (17), qui descend obliquement vers le bord postérieur de la mastoïde. Ce sillon, le bord postérieur et le bord supérieur de la perforation (20), le bord supérieur de la fente (19) et du sillon (21) marquent les limites antérieure et inférieure des parties, qui paraissent avoir un aspect normal. C'est toute la partie de la région, qui est au-dessous et en avant de ces limites, qui semble évidée. Toute cette surface osseuse, qui n'est pas occupée par la perforation, présente un aspect lisse et uni. La grande perforation (20) correspond au sinus latéral. La fente (19) et le sillon (21) correspondent à la zone antrale de la mastoïde, et au premier examen de la pièce, on se demande si la cavité intra-pétreuse, qui se trouve en dedans d'elles, n'est pas l'antre lui-même. Mais, à un examen plus attentif, on voit qu'il n'en est rien, ainsi que je le dirai un peu plus loin.

Face endocrânienne (fig. XX). — La gouttière du sinus latéral est plus large que d'habitude. A sa partie terminale, cette gouttière s'élève en forme de crête (8), qui obligeait la partie terminale du sinus à se relever, en formant un coude procident vers le haut et soulevant la face inférieure du rocher, au niveau du plancher de la caisse. Un peu au-dessous de coude (5) du sinus, la gouttière sinusienne présente une perforation de forme quadrilatère, qui est la même que celle déjà vue sur la face extérieure de l'os (20). Au niveau de son angle supéro-antérieur, cette perforation se continue avec une autre perforation (13), qui intéresse la corticale de la face postérieure du rocher et occupe presque toute la surface, qui correspond à ce que j'ai désigné sous le nom de zone pétreuse de la face postéro-interne du bloc mastoïdien. Elle est de forme polygonale, un peu plus longue que haute. Sa longueur = 14 mm., sa hauteur = 10 mm. Son bord supérieur n'atteint pas le bord supérieur du rocher, mais monte, cependant, un peu plus haut que le bord correspondant de la perforation (12), qui occupe la gouttière du sinus latéral. Son bord inférieur descend au niveau du milieu du bord antérieur de celle-ci. Cette perforation conduit dans une grande cavité, qui est creusée dans l'épaisseur même de la base du rocher, à l'endroit où est habituellement l'antre. Mais ce n'est pas l'antre qu'elle représente, car celui-ci est tout petit et cantonné très haut et très en avant, au voisinage immédiat de l'aditus. En dedans, cette cavité n'a d'autre paroi que les minces bords, qui entourent la perforation (13) ; en dehors, elle correspond à la fente (19) et au sillon (21), déjà décrits sur la face extérieure de l'os et à la mince lame

osseuse (18), qui se trouve en arrière du méat auditif. En haut, elle correspond à la paroi supérieure de la base du rocher, qui lui forme un toit excessivement mince. En avant, la cavité s'étend jusqu'au voisinage de la paroi postérieure de la caisse et du conduit. A la partie supérieure de la paroi antérieure de cette cavité se trouve un orifice, qui conduit dans un autre espace, lequel est l'antre lui-même. Un fil de laiton (14), introduit dans la cavité intra-pétreuse, passe à travers l'antre et la caisse et ressort, sur la face extérieure, par le conduit auditif externe (15).

Sinus latéral. — Je n'ai pu voir que le squelette de cette pièce, sans le sinus lui-même ; mais l'étude des rapports de ses diverses parties avec la gouttière osseuse, qui correspond au sinus, corrobore la description donnée par Brühl. La portion descendante du sinus était, au niveau de la perforation quadrilatère, en contact direct avec les téguments extérieurs de la région mastoïdienne. Immédiatement au-dessous de son coude, le sinus se dilatait en avant et s'enfonçait dans l'intérieur même du rocher, en passant à travers la perforation polygonale. Il formait, là, une sorte de *bulbe supérieur* (Brühl, *Berlin. Otol. Gesellschaft*, Dezember 1911), qui n'était séparé de la face supérieure du rocher que par une lame osseuse très mince et qui s'avançait au voisinage direct de la paroi postérieure du conduit auditif externe. Le point culminant de ce *bulbe supérieur* faisait saillie dans l'intérieur même de la cavité antrale, dont il n'était séparé que par la muqueuse de revêtement, au niveau de la déhiscence qui, sur le squelette sec, fait communiquer l'antre avec la cavité intra-pétreuse. J'ai essayé de représenter sur la figure XX la projection du sinus à la surface extérieure de la mastoïde. On voit, ainsi, que le coude vient très loin en avant, et correspond à la large perforation de la face externe, ainsi qu'à la fente et au sillon, qui continue celle-ci. Ce sinus procident et dilaté occupe non seulement la zone sinusienne, mais aussi les zones que j'ai appelées zone antrale et zone sous-antrale.

Au niveau de son abouchement dans le golfe de la jugulaire, la partie terminale du sinus formait un coude pour passer au-dessus de la crête (8), qui s'élève entre le trou déchiré postérieur et la gouttière du sinus. Ce coude terminal du sinus soulevait le plancher de la caisse jusqu'au niveau du bord supérieur de la fenêtre ronde.

D'après Brühl, Hartmann a aussi décrit une pièce de la collection de Politzer, dans laquelle la base de la pyramide était atrophiée et permettait au sinus latéral d'arriver au contact de la paroi postérieure du conduit auditif ; Zuckerkandl aurait aussi décrit une disposition semblable.

Antre. — L'antre est tout petit, haut placé. Il représente un antre fœtal, correspondant aux premiers stades de sa formation chez le fœtus du 5e mois. Sur un sujet semblable, la trépanation antrale ordinaire, rétroméatique, aurait exposé à de graves lésions du sinus. Ces lésions auraient pu se produire dès le premier temps de l'opération, au moment du décollement du périoste du bord postérieur de l'incision des parties molles,

Figure XXII

Temporal d'un enfant de 2 mois coupé en 6 tranches dans le sens vertical et antéro-postérieur. La coupe *a* est la plus externe ; la coupe *g*, la plus interne. A cet âge, le conduit auditif externe et la caisse du tympan n'ont pas encore acquis la position qu'ils ont chez l'adulte : la membrane du tympan forme avec l'horizon un angle très accentué, ouvert en dehors et en avant. La cavité tympanique paraît couchée au-dessus du conduit auditif externe et le déborde en dehors. Cette disposition est nettement démontrée par ces diverses coupes.

a.—La coupe passe à travers la paroi externe de l'antre.—1, sinus latéral.—2, cellules de la paroi externe de l'antre, entourées d'un tissu osseux, très dense.—3, partie de ce tissu compact séparant les cellules du tissu spongieux : corticale péricellulaire.

b.—La coupe passe en plein antre, mais n'intéresse pas encore le conduit auditif externe.—1, sinus latéral.—2, sinus pétreux supérieur.—3, antre.—4, tissu compact formant la paroi inférieure et externe de l'antre au voisinage de la paroi supérieure du conduit, lequel sera intéressé dans la coupe suivante.—5, tissu compact périantral : corticale centrale, périantrale.—6, cellules de la voûte de l'antre.

c.—La coupe passe par l'antre, l'aditus et le conduit auditif externe.—1, sinus latéral.—2, antre : cellules dans sa paroi postérieure.—3, cavité épitympanique.—4, courte apophyse de l'enclume.—5, paroi supérieure de l'antre.—6, paroi inférieure (fibreuse) du conduit auditif externe.—7, lumière du conduit auditif externe.—8, corticale périantrale.—9, paroi supérieure, osseuse, du conduit auditif externe (mur de la logette).

d.—Cette coupe intéresse, à la fois, les canaux demi-circulaires, la caisse et le conduit auditif externe.—1, sinus latéral.—2, loge dure-mérienne de la vésicule endolymphatique.—3, canal demi-circulaire postérieur.—4, canal pétro-mastoïdien (canalis subarcuatus).—5, canal demi-circulaire supérieur.—6, canal demi-circulaire externe.—7, section de la longue apophyse de l'enclume.—8, section du marteau.—9, paroi inférieure du conduit auditif externe, formée ici par l'os tympanal.—10, membrane du tympan.—11, canal de Fallope.—12, loge du muscle de l'étrier.—13, lumière du conduit auditif externe.—14, caisse.—15, cavité épitympanique.

e.—Cette coupe intéresse, à la fois, le vestibule, la caisse et le cul-de-sac inférieur, prétympanal, du conduit auditif externe.—1, sinus latéral.—2, fenêtre ronde.—3, loge de la vésicule endolymphatique, au niveau de la fente unguéale.—4, aqueduc du vestibule ouvert dans toute sa longueur.—5, fossa subarcuata.—6, vestibule (empreinte de l'utricule et du saccule).—7, canal de Fallope.—8, hiatus de Fallope.—9, cavité sous-vestibulaire (partie initiale du premier tour de spire du limaçon).—10, conduit du muscle du marteau.—11, orifice de la trompe d'Eustache à la partie antérieure du plancher de la caisse. Celui-ci, étant donnée l'orientation de la caisse et du conduit chez le très jeune enfant, semble, sur cette coupe, former la paroi interne de la cavité tympanique, de même que la membrane du tympan (paroi externe) semble en former la paroi inférieure.—12, membrane du tympan, coupée au voisinage de son pôle inférieur.—13, golfe de la jugulaire.—14, paroi inférieure du conduit auditif externe : en ce point, cette paroi est plus mince que dans la coupe précédente ; cela tient à ce que l'os tympanal présente ici une déhiscence comblée par du tissu fibreux.

f.—Cette coupe intéresse le fond du conduit auditif interne, le limaçon et l'orifice de la trompe d'Eustache au niveau de la paroi antérieure de la caisse.—1, conduit auditif interne, coupé tout près de son extrémité profonde au voisinage de la fossette destinée au nerf facial, en haut, 2.—3, encoche du bord supérieur du rocher au voisinage de la fossa subarcuata.—4, limaçon.—5, fissure pétro-tympanique.—6, conduit du muscle du marteau.—7, fosse jugulaire.—8, canal carotidien.—9, cellules de la partie antérieure du plancher de la caisse.—10, trompe d'Eustache.

g.—La coupe ouvre largement la fosse jugulaire, la partie ascéndante du canal carotidien et la trompe d'Eustache cartilagineuse.—1, conduit auditif interne.—2, limaçon.—3, fosse jugulaire.—4, canal carotidien.—5, trompe d'Eustache.—6, fissure pétro-tympanique (voir 'Déhiscences intra-crâniennes des cavités de l'oreille,' *Bulletins et mémoires de la Soc. fr. d'O.-R.-L., Congrès de* 1910).


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car la rugine aurait certainement blessé le sinus, qui était si peu protégé par l'os. Mais, abstraction faite de cela, la trépanation exécutée suivant le procédé classique, en arrière du méat auditif, n'aurait même pu être tentée, sans occasionner des graves blessures du sinus. Seule la trépanation transspino-méatique aurait permis d'ouvrir un antre aussi petit et si haut placé. Les rapports de ce sinus avec l'antre et avec la paroi postérieure du conduit auditif, abstraction faite des déhiscences osseuses, sont les mêmes que ceux que j'ai mentionnés dans l'observation, dont j'ai parlé au cours de ce travail, dans laquelle le sinus fût trouvé au contact de la paroi postérieure du conduit, sous une corticale de 2 mm. d'épaisseur, à la limite postérieure du trou de la trépanation transspino-méatique.

La conformation très anormale de cette pièce ne peut s'expliquer que par un arrêt de développement de la base du rocher et de la portion de l'os écailleux, qui forment la mastoïde.

L'aile squamo-mastoïdienne de l'os écailleux s'est arrêtée au cours de sa formation. Au lieu de s'étaler sur la face externe de la base du rocher et de recouvrir toute la partie pétreuse proprement dite (zone antrale et zone sous-antrale), jusqu'à l'apex, la partie squameuse de la mastoïde n'a pas dépassé une ligne horizontale passant par l'angle supéro-postérieur du méat auditif : c'est le bord inférieur de l'aile squamo-mastoïdienne, restée rudimentaire, qui forme le bord supérieur de la perforation, ainsi que la lèvre supérieure du sillon qui prolonge la fente vers la spina-supra-méatum. Elle laisse ainsi à découvert toute la partie antérieure de la mastoïde, en arrière du conduit auditif. C'est pour cela que toute cette région manque de relief et apparaît, au contraire, comme une surface évidée : il lui manque la couche squameuse, qui la recouvre habituellement.

La partie pétreuse de la mastoïde, c'est-à-dire la base de la pyramide pétreuse a subi, elle-même, des troubles profonds au moment de son développement. L'ossification s'est mal faite en dehors et en avant du sinus, au voisinage de son coude. En dehors, le manque d'ossification a permis au sinus d'être en rapport direct avec les téguments extérieurs. En avant, la base du rocher a englobé dans son intérieur le coude du sinus latéral, au cours de son développement. Au lieu de former, entre celui-ci et l'antre, une paroi épaisse, dont l'élargissement progressif a pour résultat de refouler le sinus en arrière, l'ossification est restée superficielle et rudimentaire et a permis au sinus d'être en contact direct avec la cavité de l'antre. Le coude du sinus est ainsi resté en situation très antérieure et s'est allongé en forme de bulbe (bulbe supérieur), pendant que le crâne augmentait de volume : la base du rocher s'est ébauchée seulement tout autour du genou procident, en l'englobant dans son intérieur. Mais l'ossification a été encore plus incomplète en dehors, au niveau de la fente (19), où l'arrêt de développement de l'aile squamo-mastoïdienne a permis au genou d'être, lui aussi, en rapport avec les téguments extérieurs comme l'est sa portion descendante.

En résumé, il s'agit bien ici d'une anomalie très importante, qui a frappé tout à la fois la base du rocher et l'aile squamo-mastoïdienne de l'os écailleux.

Quelle est la cause qui préside à la formation de pareilles anomalies ? Nous ne pouvons rien dire à ce sujet, il convient simplement de constater les faits. Rien, en général, ne peut les faire prévoir. Elles sont très rares, fort heureusement, pour les malades et pour les chirurgiens. Mais il faut savoir qu'elles peuvent exister.

SECTION XVI

OTOLOGY

DISCUSSION No. 2

THE PATHOLOGY OF ACQUIRED DEAFMUTISM

REPORT BY PROFESSOR DR. HOLGER MYGIND, COPENHAGEN

THE term 'acquired deafmutism' will be used here as applying to deafmutism caused by pathological changes of the hearing organs occurring after birth.

My esteemed colleague, Professor Denker, who introduces the subject of the pathology of congenital deafness before this section, has proposed [1] to use the name 'im Leben erworbene Taubstummheit'; but as we also speak about fœtal *life*, I think the term 'after birth', used already by me in my monograph,[2] is to be preferred.

The object of this paper is to put before the section a short synopsis of our present knowledge of the pathology, i.e. the anatomy and pathogenesis, of deafmutism acquired after birth.

This general view will principally consist in the presentation of facts; but it cannot be otherwise than that the explanation of these, the significance here given to them, and the deductions drawn from them are open to discussion. The paper will therefore, I hope, also fulfil its purpose of serving as basis for a discussion on this important subject so happily chosen by the committee of the section.

The material used here is purely literary. It consists principally of reports of the examination published in literature of petrous bones of deafmutes with acquired deafness.

It has been necessary to exclude a series of these cases, as I intend to use only such cases (1) which have been thoroughly investigated and fully described, or where at least the description given justifies the supposition that important anatomical changes have not been overlooked; (2) where the history of the case states the deafness to have been acquired after birth; and (3) where the pathological changes described do not distinctly speak against the history of the case.

It seems a matter of course that only cases chosen with these restrictions should be used. It is, however, a fact that several authors have classified their cases regardless of these reservations. I shall especially point out that cases published have been classified according to hypotheses as to the anatomical alterations found post mortem. Several mistakes are, in my opinion, made in this way. These have principally

[1] *Anat. d. Taubst.*, Lief. vii, p. 53.

[2] *Taubstummheit*, 1894, p. 10.

arisen from the want of regard to the fact, which I stated twenty-three years ago,[1] and which has later on been corroborated by Siebenmann,[2] Goercke,[3] and Alexander,[4] that it is very difficult, and in fact frequently impossible, to determine by anatomical examination alone whether an individual case of deafmutism is congenital or acquired. If further proofs are wanted, this paper will furnish them.

The subdivision of acquired deafmutism into several groups or types seems natural when one intends to give a survey of the various anomalies found in the hearing organ. Uchermann,[5] and later on Siebenmann,[6] have divided the cases according to their ætiology (meningitis, scarlet fever, measles, &c.). There is one objection to this classification, viz. that the history of the case is in many instances uncertain. It is, however, a classification which gives least room for theories and hypotheses, which, according to my opinion, play a too prominent part in the classification proposed by other authors, of which Denker's must be considered the best.[7] Against Denker (who subdivides acquired deafmutism into three groups, (1) cases caused by meningitis, (2) cases of 'tympanal' origin, and (3) those caused by primary labyrinthine changes [8]) the following objections may be made. First, the existence of primary otitis intima is not proved as yet. Secondly, it is, as mentioned before, difficult and often impossible to determine—as Denker has done—by anatomical examination only when a case is congenital or when it is acquired, and the discrimination between meningitis deafness and other forms of acquired deafness is, as I shall show later on, most difficult. Thirdly, the division is based partly upon ætiology (meningitis) and partly upon pathogenesis (spreading from the middle ear).

I consider a subdivision of acquired deafmutism into groups or types is not indispensable for a better understanding of the pathological changes causing it. For, as will be shown later on, whether we use the one classification or the other, we shall find that the principal anatomical alterations do not vary as to their nature in the different groups. We have the same processes in all of them, only varying as to the intensity and extent of them.

It seems therefore to be a better plan to describe the pathological changes found in deafmutes with acquired deafness according to the topographical seat of the lesions found.

Before carrying out this purpose I will present a list of the post-mortem examinations which form the material for this paper.

[1] *Arch. f. Ohrenheilk.*, vol. xxx, p. 113.
[2] *Anatomie und Pathogenese d. Taubstummheit*, 1904.
[3] *Pathologie d. Taubstummheit*, 1908.
[4] *Arch. f. Ohrenheilk.*, vol. xlviii, 1904, p. 112.
[5] *Les Sourd-Muets de Norvège*, 1901. [6] loc. cit.
[7] *Anat. d. Taubst.*, Lief. vii, p. 53.
[8] Amongst the cases of primary labyrinthitis Denker mentions that of Citelli (*Arch. Ital. d. Otol.*, vol. xvii). It concerns, however, a *living* deafmute, whose deafness was caused by purpura hæmorrhagica.

I. Cause: Meningitis or Acute Disease with Brain Symptoms

1. Ibsen and Mackeprang (1832).—Prep. 46 and 47, *Z. f. O.*, vol. xxiv, 1893, p. 116.
2. —— (1833).—Prep. 48 and 49, ibid., p. 117.
3. Bochdalek (1834).—*Med. Jahrb. d. Oest. Staat.*, vol. xxxviii, 1840, p. 8.
4. —— (1838).—Ibid., p. 202.
5. Schwartze, H. (1851).—*A. f. O.*, vol. v, 1867, p. 296.
6. Moos and Steinbügge.—*Z. f. O.*, vol. xii, 1883, p. 96.
7. Politzer, A.—*Lehrb. d. Ohrenheilk.*, 1882, p. 809.
8. —— ibid., p. 822.
9. Schultze, Fr.—*Virchow's Arch.*, vol. xix, 1885, p. 1.
10. Larsen, P., and Mygind, H. (1889).—*A. f. O.*, vol. xxx, 1890, p. 188.
11. Habermann, I.—*Z. f. Heilk.*, vol. xii, 1890, p. 133.
12. Steinbrügge, H.—*Orth's Lehrb. d. path. Anat.*, vol. vi, 1891, p. 96.
13. Scheibe, A.—*Z. f. O.*, vol. xxvii, 1895, p. 100.
14. Nager, F. (1903).—Ibid., vol. liv, 1907, p. 217.
15. Schwabach.—Ibid., vol. xlviii, 1904, p. 301.
16. Stein, W. (1904) —*Anat. d. Taubst.*, Lief. iii, 1906, p. 20.
17. Denker, A.—Ibid., Lief. v, 1908, p. 20.

II. Cause: Scarlet Fever

18. Field (1875).—*Diseases of the Ear*, 1879, p. 82.
19. Uchermann, V. (1883).—*Z. f. O.*, vol. xxiii, 1892, p. 70.
20. Donaldson, Bryant, and Sears.—*Am. Journ. of Psych.*, vols. iii and iv, 1890–1.
21. Mygind, H.—*Z. f. O.*, vol. xxiii, 1892, p. 217.

III. Cause: Measles

22. Ibsen and Mackeprang (1831).—Prep. 32 and 33, *Z. f. O.*, vol. xxiv, 1893, p. 114.
23. —— (1832).—Prep. 44 and 45, ibid., p. 116.
24. Mygind, H.—*Z. f. O.*, vol. xxii, 1892, p. 196.
25. Schwabach.—Ibid., vol. xlviii, 1904, p. 296.
26. Alexander.—*A. f. O.*, Bd. lxxiv, 1907, p. 112.

IV. Cause: Injury of the Head

27. Bochdalek (1839).—*Med. Jahrb. d. Oest. Staat.*, vol. xl, 1842, p. 132.
28. Nager, F. (1904).—*Z. f. O.*, vol. liv, 1907, p. 229.

V. Cause: Uncertain

29. Mürer.—*De causis cophoseos*, 1825.
30. Ibsen and Mackeprang (1827).—Prep. 16 and 17, *Z. f. O.*, vol. xxiv, 1893, p. 110.
31. Bochdalek (1831).—*Med. Jahrb. d. Oest. Staat.*, vol. xxx, 1840, p. 9.
32. Ibsen and Mackeprang (1831).—Prep. 34 and 35, *Z. f. O.*, vol. xxiv, 1893, p. 114.
33. —— (1832).—Prep. 38 and 39, ibid., p. 115.
34. —— (1832).—Prep. 42 and 43, ibid., p. 116.
35. Bochdalek.—*Med. Jahrb. d. Oest. Staat.*, vol. xl, 1840, p. 269.
36. Moos.—*Arch. f. Aug. u. Ohrenheilk.*, vol. ii, 1871, p. 98.
37. Mygind, H.—*Taubstummheit*, 1893, p. 255.
38. Schwabach.—*Z. f. O.*, vol. xlviii, p. 294.

These 38 cases represent 75 petrous bones, as only the one side was examined in Mürer's case.

Before entering into an analysis of these 75 petrous bones, it should be stated that in a small series of cases published in literature and not examined microscopically, no changes were found by macroscopical examination of the labyrinth. These cases, which principally belong to an older period, when no microscopical examination was made, are not included in the list, p. 90.

It should further be stated that *there is no case on record in which the microscopical examination of the membranaceous labyrinth has yielded a negative result.*

Another fact is that *in none of all the cases of acquired deafmutism were abnormal ties found in the cerebral part of the hearing organ which could be considered as cause of the deafness.*

The pathological changes found in the cases mentioned above, p. 89, may be divided into the following groups: Abnormalities of (1) the middle ear, (2) the labyrinth, and (3) the acoustic nerve. A table of the principal changes described is appended:

TABLE OF PRINCIPAL PATHOLOGICAL CHANGES IN 75 PETROUS BONES

	Pathological Changes.	*Meningitis*	*Scarlatina*	*Morbilli*	*Trauma*	*Cause unknown*	*Total*
Auris media.	Otitis med. supp. chron.	4	5	2	0	3	14
	Otitidis med. supp. sequelæ	5	2	6	0	7	20
Fenestra rotunda.	Total osseous obliteration	12	8	4	2	9	35
	Deposits of osseous or fibrous tissue	6	2	2	0	0	10
Fenestra ovalis.	Total osseous obliteration	3	3	0	0	3	9
	Deposits of osseous or fibrous tissue	8	0	0	2	2	12
	Anchylosis of the stapes	6	1	0	2	1	10
	Stapes or basis of the stapes wanting	2	4	0	0	3	9
Vestibulum.	Total or almost total osseous obliteration	3	1	0	0	2	6
	Partial osseous or fibrous obliteration	12	4	0	2	5	23
	Slight endosteal changes	5	0	0	0	0	5
Aquæductus vestibuli.	Total osseous obliteration	8	1	0	0	3	12
	Partial osseous or fibrous obliteration	4	2	0	2	0	8
Canales semicirculares.	Total or almost total osseous obliteration	12	4	2	0	6	24
	Partial osseous or fibrous obliteration	12	4	0	2	5	23
Cochlea.	Total or almost total osseous obliteration	12	4	2	0	6	24
	Partial osseous or fibrous obliteration. The cochlear duct totally or partially wanting	2	1	2	0	5	10
	Slight endosteal changes. The cochlear duct principally preserved	13	0	0	2	0	15
	No endosteal changes. The cochlear duct preserved	3	0	4	0	0	7
Aquæductus cochleæ.	Total osseous obliteration	6	1	0	0	0	7
	Partial osseous or fibrous obliteration.	9	0	0	2	0	11
Nerv. cochlear. and its ramifications.	The fibres in the spiral lamina wanting or degenerated	17	0	2	2	2	23
	The spiral ganglion wanting or degenerated	17	0	2	2	2	23
	The cochlear nerve degenerated	9	0	0	2	0	11
Nerv. acusticus.	Atrophy and degeneration (microscopical)	6	0	0	0	0	6

I. PATHOLOGICAL CHANGES OF THE MIDDLE EAR

The middle ear is frequently the seat of pathological changes, as 34 petrous bones out of 75 exhibited abnormalities. In these 34 cases are not included a few cases where there was acute catarrhal or suppurative otitis media.

The predominant pathological condition in the middle ear of deafmutes with acquired deafness is *chronic suppuration*, often combined with destruction of the osseous content or the osseous walls of the tympanum (14 petrous bones).

In cases where there is no chronic suppuration, the middle ear contains not infrequently *residua of an inflammatory process* in the shape of adhesive fibrous tissue, new formation of bony substance, total or partial absence of the stapes, &c.

In no case could the changes in the middle ear be considered as the immediate cause of the deafness, as considerable abnormalities were found in the cochlea in all these cases, except that of Donaldson (case No. 20), and, according to the author himself, the one petrous bone examined by Uchermann (case No. 19), in which two cases, however, no microscopical examination of the labyrinth was performed. Further investigations must show whether we are allowed to generalize this fact as concerning all cases of acquired deafmutism.

The importance of the anatomical alterations of the middle ear must be sought in the circumstance that their existence points, in individual cases, towards the middle ear as the primary seat of an inflammation which has spread to the internal ear.

The presence of chronic inflammation of the middle ear or of inflammatory products does not, however, prove with certainty that this part of the hearing organ has been the primary seat of disease, which has resulted in deafmutism. It is, especially, a fact that cerebro-spinal meningitis, one of the most frequent causes of deafmutism, may simultaneously produce inflammation of the labyrinth and the middle ear. We also find that 9 out of 34 petrous bones belonging to the meningitis group exhibited inflammation of the middle ear or residua thereof. Besides, the coexistence of inflammation in the middle and the inner ear may be accidental.

Furthermore, the absence of inflammatory residua in the middle ear does not absolutely prove that the primary inflammation has not commenced there, as it is a fact that an acute suppuration of the middle ear need not leave any trace behind.

Looking at our material, we shall, however, see that in all our cases the primary suppuration of the middle ear caused by scarlet fever and measles having resulted in labyrinthine changes, has not only left traces behind in the middle ear, but that these also are very marked, consisting in considerable destruction or its residua. The one petrous bone in Uchermann's case (No. 19) seems an exception, according both to

Uchermann himself and to Siebenmann,[1] who both mention the middle ear as normal on the right side. I cannot admit this, as the membrane of the round window is reported to form ' an ossified plate ', which very likely has been caused by a suppuration of the tympanum having destroyed the membrane.

Altogether Uchermann's case cannot be used to prove the existence of deafmutism caused by scarlatina-meningitis, as the left tympanum contained caseous matter, which also filled the small osseous cavities of the temporal bone, and therefore cannot be considered as having been normal as suggested by Siebenmann, who also mentions my case (No. 21), as being probably meningeal, although the tympanic cavity in both specimens was filled with pus, and did not contain any ossicula.

The only case which proves the existence of deafmutism caused by meningitis during acute exanthematic diseases is Nager's (No. 14), where both the history of the case and the anatomical examination, under which the middle ears only exhibited signs of a slight catarrhal process, distinctly show that the deafmutism was caused by meningitis developed in connexion with measles.

In most cases without history it is difficult to decide whether the anatomical changes of the labyrinth are of tympanic or intracranial origin. As far as the material used here allows one to draw a conclusion in this question, I should say that *deafmutism of tympanic origin is characterized by the presence of highly destructive processes in the middle ear or their residua together with labyrinthine new formation of osseous or fibrous tissue of comparatively moderate degree and extent.* Deafmutism caused by *meningitis* or other intracranial diseases with brain symptoms is, on the other hand, characterized *by more considerable new formation in the internal ear, combined with a normal tympanic cavity, or with a tympanic cavity with moderate inflammatory alterations.*

The destructive character of the middle-ear inflammation in cases of deafmutism of tympanic origin is also shown by the fact that the *stapes was entirely absent* in 4 petrous bones of the 18 belonging to the two groups where the cause of the deafness was scarlet fever and measles, while amongst the 34 petrous bones corresponding to the meningitis group, the base only of this ossiculum was wanting in 2 petrous bones. Finally, 2 deafmutes of the two groups first mentioned had *retro-auricular fistulæ*, which existed on both sides in one case (Field, No. 18), and only on one side in the other (Mygind, No. 21).

It is not accidental that the middle-ear suppuration was so destructive in the two deafmutes just mentioned, as they both had acquired their deafness from *scarlet fever, which is more deleterious to the middle ear than measles. The labyrinthine changes in deafmutism from measles are also the result of a less destructive process than they are in cases of scarlatinal deafmutism,* and in two cases (Schwabach, No. 25, and Alexander, No. 26) they were only demonstrated by microscopical examination.

[1] loc. cit., p. 39.

In cases of deafmutism of tympanic origin it is often difficult to find the way through which the inflammation has spread from the middle to the internal ear. This cannot surprise one considering that a long space of time has always elapsed, when the petrous bone is examined, since the acute stage.

While we often find in clinical cases a fistula from the middle to the internal ear as the way of propagation of the inflammation, such *labyrinthine fistulæ has never been found in deafmutes.* This fact is, I think, best explained by the circumstance that *in deafmutism we nearly always have to do with an acute suppuration of the middle ear*, while fistulæ generally are the result of chronic suppuration. The possibility of the existence of labyrinthine fistulæ is, however, proved by Manasse,[1] who observed the initial state of such a fistula in a child, who became completely deaf through a scarlatinous suppuration of the middle ear. In this case the breaking down of the osseous wall of the horizontal semicircular canal commenced from the labyrinthine side.

We must therefore look upon the two fenestræ as the gates through which middle-ear suppuration causes changes in the internal ear leading to deafmutism.

The two Fenestræ. In the cases of deafmutism caused by scarlet fever and measles, where a tympanic origin of the labyrinthine disease is predominant, we find the two fenestræ as seat of pathological changes in 14 petrous bones out of 18.

The significance of this considerable frequency with which the two windows show abnormalities in the two groups mentioned is considerably diminished by the fact that the two fenestræ are also the seat of changes with considerable, though less, frequency in deafmutes belonging to the meningitis group, viz. in 22 petrous bones out of 34. In both groups the abnormalities found could in all cases be explained as results of an inflammation.

These facts show that the two windows and their content can be attacked both from the tympanic side and also—though with a little less frequency—from the labyrinthine side.

An examination of the character of the anomalies of the two fenestræ and of the frequency with which each window is abnormal in the two different groups, viz. the meningitis group, and the scarlatina and morbilli group, gives the following results.

In the meningitis group the most frequent anomaly was the *complete closing of the fenestra rotunda* with bony substance (12 petrous bones), while in 6 specimens it was only *partially closed* with osseous or fibrous tissue. *The oval window* was closed with bony substance in 3 petrous bones.

In all the cases of the same group where the round window was completely closed with a bony mass, the entire cavity of the cochlea was filled up with osseous tissue, which, as I have observed myself in all the

[1] *A. f. O.*, vol. lxxxix, 1912, p. 155.

preparations in question of the collection of Mackeprang and Ibsen, and in the case described by Larsen and myself, forms the bottom of the fossula fenestræ cochleæ.

The osseous closure of the round window can, then, be formed from the labyrinthine side, and need not be produced by any inflammation of the tympanum. There was also amongst the specimens mentioned only one which exhibited signs of inflammation of the middle ear (Schwartze, case No. 5) in the shape of a chronic suppuration.

In the two groups of deafmutism of tympanic origin the round window was still more frequently closed by osseous tissue (12 specimens), which seems to point towards this locality as one which plays an important part in the propagation of the suppuration from the middle ear to the labyrinth. But also in this group this anomaly was frequently present together with solid osseous obliteration of the cochlea (4 petrous bones). This circumstance diminishes, of course, the significance of the frequency stated above.

On the other hand, inflammatory processes may spread through the membrana secundaria without producing any loss of substance,[1] and need not, therefore, leave any new formation of tissue behind.

It will be seen that our material does not allow of any definite estimate of the frequency with which the tympanic cases of deafmutism are caused by the suppuration in the middle ear going through the round window.

A comparison of the frequency with which the round window is abnormal with the frequency of alterations of the oval window, does not give us any certain answer to this question. For while there was found inflammatory products in and about the oval window in 17 specimens out of 34 belonging to the meningitis group, this was the case in 3 out of 18 of the group where scarlet fever and measles were the cause of the deafmutism.

Neither does a closer investigation of the individual cases of the two latter groups give any hint, as only in one case (Mygind, No. 21) was the oval window open on one side admitting a communication of the cholesteatomatous masses in the middle and the internal ear. On the other ear the oval window was closed by osseous tissue, but here the vestibule was entirely obliterated by an osseous mass. In Donaldson's case (No. 20), the oval window on both sides was also closed, while the state of the vestibule is not described; on both sides the stapes was missing, and this was also the case in the deafmute whose petrous bones were examined by me.

Siebenmann puts great stress upon the absence of the stapes as a sign that the oval window has formed the road of communication; but it must be remembered that the acute suppurations of the middle ear caused by scarlet fever or measles, and which lead to deafmutism, are exceedingly destructive in their character. According to Manasse [2] they attack all bony tissue in the middle ear very quickly, the inflam-

[1] Grünberg, *Z. f. O.*, vol. lxiv, p. 164. [2] loc. cit.

mation having a decidedly explosive character. Manasse's investigation also points decidedly in the direction that both windows get attacked simultaneously, and the post-mortem examinations of deafmutes do not speak against but rather in favour of this.

Lastly, I will mention that the pathological changes of the oval window in cases of meningitis deafmutism consist principally of anchylosis of the stapes and the formation of fibrous tissue on the labyrinthine side of the window.

II. PATHOLOGICAL CHANGES OF THE LABYRINTH

Several cases are recorded where no changes were found in the labyrinth, but they are all cases where microscopical examination of the labyrinth was not performed.

In most cases of acquired deafness the changes found are macroscopical; in a few cases they are only microscopical, and *in no case has a microscopical examination yielded a negative result.*

A. Pathological Changes of the Vestibulum

In about half of the number of cases of acquired deafness leading to deafmutism the vestibulum was found to be of normal appearance by macroscopical examination.

Amongst the cases microscopically examined, there were 3 petrous bones in which no abnormality was found (Schwabach, No. 25, both sides, Stein, No. 16, on one side). On the other ear in Stein's case the alterations were very slight, consisting in degeneration of the neuro-epithelium and of slight degeneration of the terminal ramifications of the vestibular nerve. Similar abnormalities were found by Alexander in the utriculus, while the sacculus and the canalis reuniens were destroyed (case No. 26, both sides).

Altogether, the vestibule is the part of the labyrinth which is least affected. This speaks against the opinion that the fenestra ovalis is most frequently the road by which suppuration of the middle ear attacks the labyrinth, and against the vestibular aqueduct being a frequent channel for the propagation of meningitis. There are, however, two circumstances emphasized by Siebenmann [1] which might explain the fact mentioned. The one is the wideness of the vestibule compared with that of the cochlea and the semicircular canals; the other, the double supply of vessels stated by Siebenmann.[2]

The vestibule was *totally obliterated by bony substance* in 6 petrous bones out of 75, a comparatively small number considering that the cochlea was totally filled up with osseous tissue in 20 petrous bones. Amongst the other macroscopical changes should be mentioned *partial filling up of the cavity with osseous or fibrous tissue*, cholesteatomatous

[1] loc. cit., p. 22. [2] *Die Blutgefässe d. menschl. Labyrinths.*

masses and other products of inflammation. Amongst these latter must be mentioned the peculiar pathological tissue found on one side by Schwartze (No. 5), and on both sides by Schwabach (No. 15). It was formed by a *mixture of nerves and connective tissue*, which Schwabach has described as resembling neuromata after amputation. Both cases belong to the meningitis group.

In most cases the membranaceous content of the vestibule was totally destroyed. In other cases it had at least partially resisted the destructive processes. The microscopical examination revealed then the presence of destruction and degeneration of the neuro-epithelium of the maculæ in connexion with slight osseous or fibrous endosteal deposits and fibrous adhesions in the perilymphatic space. In the few cases mentioned above the membranaceous content was normal or nearly normal.

In all cases, except one (*Nager, No.* 28), *the pathological changes were much more marked in the sacculus than in the utriculus*, the latter being present in some cases, while the sacculus was totally destroyed. In Nager's case it was the reverse.

Aquæductus Vestibuli. The vestibular aqueduct is frequently the seat of pathological changes in acquired deafmutism.

As seen from the table, p. 90, this canal was *entirely obliterated by osseous tissue* altogether 12 times in the 75 petrous bones, and much more frequently altered in this way in the meningitis group than in the scarlatina-morbilli group. There is reason to believe that this pathological alteration is still more frequent than it appears from the reports, as there are still 4 petrous bones, amongst which 3 belong to the meningitis group, and 1 belongs to the group of cases caused by an uncertain disease, in which the vestibule was entirely filled up with osseous matter, which is likely to have obliterated also the vestibular aqueduct, although it is not mentioned.

Partial obliteration of the aqueduct with osseous or fibrous tissue comes next in frequency, and here also the meningitis group is predominant.

As the vestibular aqueduct may be one of the paths through which the meningitis propagates itself from the cranial cavity to the labyrinth, the facts mentioned above point towards the aqueduct as a frequent channel of propagation. It must, however, be remembered that this canal is very narrow, and that inflammatory products, therefore, easily cause obliteration just as in the semicircular canals. Besides, it is most frequently combined with considerable osseous deposits in the vestibule, from whence the obliteration may begin. We find also in Nager's case (No. 28), where the cause of the deafmutism was of traumatic origin, the aqueduct partially closed by osseous-fibrous deposits on the one side.

In this case of Nager, the aqueduct was dilated on the other side. This anomaly is noted also on one side in one of Mackeprang and Ibsen's

cases (No. 34), and on both sides in Alexander's case (No. 26). There is, I think, every reason to believe that this anomaly is congenital. Amongst the congenital alterations, Alexander also mentions the transformation of the sacculus endolymphaticus into small cavities lined with a cubic epithelium (No. 26), an anomaly also reported in Nager's case (No. 28). The same may be the case with another abnormality, viz. that the aqueduct has an abnormal direction towards the internal auditory meatus (Alexander, No. 26).

B. Pathological Changes of the Semicircular Canals

Macroscopical alterations of the semicircular canals are found with considerable more frequency than they are found in the vestibule, and with about the same frequency as in the cochlea.

The frequency with which the semicircular canals are abnormal is principally due to *total or partial obliteration of their interior by a bony mass.* This mass is often of exactly the same structure as the labyrinthine capsule, the contours of which can disappear entirely. The canals have, therefore, often been described as wanting, or as reduced to small cavities. In a few cases, the obliterating mass is calcareous or fibrous. In some cases only one of the semicircular canals is altered in this way, in others two of them are obliterated, and in not a few cases they are all closed in this way.

The osseous obliteration of the semicircular canals is found with about the same frequency in the meningitis group as in the scarlatina-morbilli group.

The frequency of these alterations can be best explained by the narrowness of the canals which favours the occurrence of inflammatory synechiæ.

Besides bony or fibrous deposits in the canals, granulations and cholesteatomatous masses have also been found in a few cases.

On the other hand, *microscopical alterations* of the semicircular canals and their membranaceous content are less frequent than the similar changes in the vestibule. They do not differ much in character from those described as found in the vestibule, but are, on the whole, of less intensity. In 6 petrous bones the microscopical examination revealed very doubtful abnormalities or none at all (Nager, No. 14, Stein, No. 16, and Schwabach, No. 25).

As the *microscopical alterations* of the labyrinth are most marked in the cochlea, it will be seen that *they generally diminish from the cochlea through the sacculus, and the utriculus to the semicircular canals.* This may also be expressed in this way, that *the superior part of the membranaceous labyrinth generally is less affected than the inferior part.*

C. Pathological Changes of the Cochlea

The cochlea is the part of the labyrinth where pathological changes are most frequently present. There is, in fact, no case reported where an examination has yielded negative results, at least with the assistance of the microscope.

Macroscopical Alterations. These occur with about the same frequency as in the semicircular canals, and are of the same nature.

They consist principally in the *partial or total obliteration of the normal cavity of the cochlea by osseous or fibrous tissue.*

As in the semicircular canals, *osseous deposits fill the cavity of the cochlea so completely,* and assume the appearance of the capsula and the bone surrounding this so thoroughly, that the normal conditions of the cochlea disappear entirely. Cases of this kind are frequently described as absence of the cochlea.

In some cases *a part only of the cochlea is obliterated by bony deposits.* This partial obliteration is generally confined to the basal part of the cochlea, which is mostly—macroscopically and microscopically—more affected than the apex. Besides, the alterations are in several instances more pronounced in the scala tympani, and in the meningitis cases more in the axial part of the scalæ than in the external.

A few times the apex of the cochlea is converted into a cavity without any trace of modiolus or of helicotrema—an abnormality which Siebenmann has termed 'pseudokongenitale Kloake' (Mygind, No. 21, on one side, and 37 on both sides), as it also appears in congenital cases.

Besides the partial obliteration of the cochlea with osseous substance, *fibrous tissue* may appear. In Field's case (No. 18), inflammatory products were found, and in Mygind's (No. 21, on one side), cholesteatomatous masses.

Finally, *slight endosteal alterations* appeared in some specimens. In most of these they were only revealed by microscopical examination. They were present in all cases belonging to the meningitis group where a microscopical examination was performed, except in Politzer's case (No. 8) on both sides, and on the one side of Stein's (No. 16), but not in any of the deafmutes who belonged to the scarlatina-morbilli group.

While complete or almost complete obliteration of the cochlea by bony substance, and consequently *complete destruction of the membranaceous content of the cochlea,* appear with the same frequency in the meningitis group as in the scarlatina-morbilli group, there is a marked difference between the cases caused by scarlatina and those by morbilli. For while this abnormality was present in 4 out of 8 cases of the scarlatina group, it is only reported in 2 out of 10 of the morbilli group.

Partial obliteration of the cochlea with osseous or fibrous tissue, combined with complete destruction of the membranaceous content appears in all three groups. The figures are, however, too small to draw any conclusion from them, especially as the material has other defects as to the significance of these facts. I shall only refer here to the circum-

stance, to be mentioned later on, that the microscopical examination of the cochlea in the cases belonging to the morbilli group comparatively frequently only revealed slight pathological alterations.

Microscopical Alterations. Microscopical examination has been performed in 26 petrous bones of the 75 which form the material of this paper.

We meet here the difficulty that many details described as pathological changes may only be caused by post-mortem or artificial circumstances. Brühl especially [1] is very reserved, and does not consider it advisable to rely upon such changes as slighter degeneration of the organ of Corti, detachment and dislocation of the membrana tectoria, absence of the membrane of Reissner, folding, distension, and collapse of the same membrane in connexion with normal insertion, slitting up of the spiral ligament, the appearance of 'ring cells', coagulation of the endolymphs, &c.

I have therefore, considering our present imperfect knowledge, not thought it suitable to go deeply into those details, which might be dubious.

The membrana basillaris. In several cases the destruction of the cochlea was so intense that the basilar membrane was completely or almost completely wanting. In several other cases the basilar membrane was wanting in the lower part of the cochlea, together with the other parts of the cochlear duct, being substituted by the obliterating osseous and fibrous tissue mentioned before.

In some cases, where the membrana basillaris was present, the vestibular surface was covered only by quite flattened homogeneous cells. In these cases the organ of Corti must be said to have been wanting entirely.

From this condition there is a gradual transition to a more and more normal appearance of a papilla basillaris. One sees first the papilla as a small lump of higher cells, which are still not differentiated. By degrees rod-cells, hair-cells, and the other elements emerge, though in a degenerated shape, and in a few cases the tunnel of Corti is slightly marked.

The membrana tectoria is wanting completely in several cases; in other cases it may just be traced as a cord, which often is the subject of hyaline degeneration. Hyaline degeneration of the membrane appears also in other cases. Shrivelling up and dislocation of the membrane also occur, but these changes are perhaps artificial. In Alexander's case (No. 26) it adhered totally to the basilar membrane. In Nager's two cases (No. 14 and 28) the membrane is described as being enveloped in a layer of cells—an abnormality which otherwise has been considered as congenital. This is another fact which points out that it is hazardous to judge from an anatomical examination only whether a doubtful case has to be ranged amongst the congenital or the acquired form of deafmutism.

The membrane of Reissner is also wanting in several cases. Where

[1] *Passow's Beitr.*, vol. v, p. 437.

it is present it is generally described as distended or collapsed or folded, the result being either a dilatation or a flattening of the cochlear duct. The dubious character of these changes is strengthened by the fact that there may, in the same individual, be distension on one side and collapse on the other, or the one alteration in some part of the cochlea and another in a different part.

In Nager's case of traumatic deafness (No. 28) there seems, however, to be a true pathological distension of the membrane of Reissner, as the membrane was pressed up against the ceiling of the scala vestibuli, which was completely obliterated thereby. Besides, the cochlear duct was further dilated on account of the lamina spiralis sloping downwards, the lamina spiralis membranacea having also a low insertion.

The other parts of the membranaceous content of the cochlea: the sulcus spiralis internus, crista spiralis, and ligamentum spirale do not exhibit, in cases where they are present, any special interest. The stria vascularis is generally atrophic, the subject of hyaline degeneration, and has few vessels according to the predominant obliterating tendency of the pathological process.

The Nerves of the Modiolus. The nervous fibres of the cochlear nerve itself is degenerated in about half of the petrous bones examined microscopically. The spiral ganglion is more frequently degenerated, and has often fewer cells than normal; in several cases it is, however, well preserved, especially in the apex. The degenerative processes are most pronounced in the spiral nerves, which often are wanting entirely, being often substituted by fibrous tissue. In one case (Alexander, No. 26) the nerves were normal all through the cochlea.

The alterations of the cochlear nerve and its ramifications seem often comparatively less pronounced than the other changes in the cochlea, also in cases of meningitis deafmutism.

This latter circumstance does not speak in favour of the supposition that intracranial inflammation frequently spreads to the labyrinth following the perineural and perivascular lymphatic spaces.

We see, therefore, that *the examination of petrous bones of deafmutes does not on this point either give us any decided answer of the question through which channels the labyrinth is generally secondarily affected.* Even if pathological changes were predominant in the modiolus in cases of deafmutism of intracranial origin, this would not positively prove the importance of the modiolus as medium in such cases. For, as K. Grünberg has reported,[1] a fresh case of labyrinthitis of undoubted tympanic origin may cause a severe inflammation along the nerve-canals of the modiolus with partial destruction of the spiral ganglion, and with a propagation almost to the porus acusticus internus.

The fact mentioned, p. 98, that the osseous and fibrous deposits are more pronounced towards the modiolus in cases of meningitis deafmutism does not either prove the theory of the spreading of intracranial

[1] *Z. f. O.*, vol. lxiv, p. 158.

inflammation along the nerve-fibres and vessels of the modiolus, as the larger surface of this part of the cochlea and the numerous vessels it contains can explain the larger new formation of tissue around it.

The Aquæductus Cochleæ. The most frequent anomaly of this canal is the *total obliteration* or want of it. The figures mentioned in the table, p. 90, are certainly too low, as there are several cases where this abnormality is not mentioned, although the cochlea was filled up entirely with bony substance.

Besides, in some cases the aqueduct was *partially obliterated* by osseous or fibrous tissue.

As the changes mentioned were only present in one petrous bone belonging to the scarlatina-morbilli groups, but rather frequent in the meningitis group, this circumstance speaks in favour of the cochlear aqueduct being a frequent channel through which inflammation spreads from the cranial cavity to the labyrinth.

The canal is, however, narrow, and as bony and fibrous deposits are more frequently met with in cases caused by meningitis, this circumstance may, to some extent, give an explanation.

III. PATHOLOGICAL CHANGES OF THE ACOUSTIC NERVE

The acoustic nerve is in several cases found abnormal. Amongst 32 specimens in which there are notes about the state of the acoustic nerve during its course in the internal meatus, it was found *abnormal by macroscopical examination only* 8 *times*. It is in these cases most frequently described as thinner than usual, or of a more fibrous character. Once the cochlear and vestibular branches are described as ending blindly in the bottom of the meatus, there being in this case obliteration of the cochlea.

A *microscopical examination* was performed in 18 specimens, but only 6 *times were changes found*. In Schwabach's case (No. 15), slight degenerative alterations were present on both sides. In Larsen and Mygind's deafmute (No. 10) the vestibular branch was replaced by a fibrous thread, and the corresponding portion of the acoustic nerve only contained a few nerve-fibres, but a great deal of fibrous tissue. In Schultze's case (No. 9) half of the bundles were atrophic.

All the cases where the acoustic nerve was found abnormal by macroscopical or microscopical examination belonged to the meningitis group, except in the case of Bochdalek (No. 35), in which the cause was unknown.

The circumstance that *the acoustic nerve only in exceptional cases shows signs of atrophy or degeneration*, speaks in favour of the opinion expressed by me more than twenty years ago,[1] that *the acoustic nerve is not much inclined to atrophy or to degenerate as a result of inactivity*. Perhaps such a form of atrophy or degeneration does not occur at all,

[1] *A. f. O.*, vol. xxx, p. 115.

as these pathological changes might very well be the product of the original inflammation.

A retrospective view gives me occasion for the following remarks.

Just as the principal pathological changes found in *the middle ear* of deafmutes, with deafness acquired after birth, are destructions caused by or products of inflammatory processes, the changes in *the labyrinth* must be regarded in the same way. This applies particularly to the labyrinthine destruction and to the new formation of osseous or fibrous tissue in the cavities of the internal ear. The degenerative alterations of the nerve epithelium and the nerves found in several cases are also most naturally explained in this way, as they have most frequently been found together with other undoubtedly inflammatory products in the labyrinth. The traumatic cases do not form an exception.

Two cases only (Schwabach, No. 25, and Alexander, No. 26), both caused by measles, form apparently an exception in this regard. In Schwabach's case there were no other inflammatory products in the labyrinth, and S. considers it therefore as congenital. In Alexander's case there were only deposits of pigment and adhesion of the tectorial membrane to the membrana basillaris. In both cases there was, however, exactly the same degeneration of the neuro-epithelium as found in several other indubitable specimens from deafmutes with *acquired* deafmutism. Stein's case (No. 16) proves also that these alterations can be present on one side, together with bony and fibrous new formations, while there are no other inflammatory processes present on the other side, and in Politzer's case (No. 8) the new formation of fibrous tissue was confined to the vestibule.

We find, altogether, as will be seen from the foregoing pages, *all transitions in acquired deafmutism from total destruction of the labyrinth to a case like Schwabach's, where there is only degeneration of the neuro-epithelium of the cochlear duct. On the other hand, there is often,* as in Stein's case, *a considerable difference between the two sides in the same individual.*

Nor is there a marked line between the alterations found in acquired deafmutism and those described in some congenital cases, in which the changes found also are most naturally explained as being the results of fœtal inflammatory processes, as first stated by me in 1890.[1] Goercke is therefore somewhat entitled to group such fœtal inflammations together with those acquired after birth.[2]

Further, we have seen that it is frequently impossible to distinguish between the labyrinthine changes of tympanal and those of endocranial origin, and also, within each of these two groups, to find in individual cases the route through which the inflammation has spread itself to the labyrinth.

[1] *A. f. O.*, vol. xxx, p. 113.

[2] *Pathologie d. Taubstummheit*, 1908.

This difficulty does not, perhaps, arise alone from the difficulty of tracing the alterations, but may also be due to the circumstance that the labyrinthine inflammation in cases of meningitis is—as suggested by Siebenmann—an integral part of a systemic disease. There is also the possibility, I think, of the inflammation of the membrane of the brain spreading at the same time through all ways simultaneously, just as I have suggested speaking about the spreading of the inflammation of the middle ear to the labyrinth.

It is therefore difficult to classify cases of acquired deafmutism of unknown origin, although it is, of course, possible in a series of cases as Denker has done it. When I have abstained from adding to my material such cases of unknown origin, which seem to be acquired after birth, the reason has principally been that I wanted to make the foundation of this paper as solid as possible. Besides, an additional number of cases would not alter much the results arrived at here.

There are still several points of the pathology of acquired deafmutism which want clearing up.

Some of these questions will certainly be solved by a continuation of the excellent work—*Die Anatomie der Taubstummheit*—edited by Denker on behalf of the German Otological Society.

Other questions, and especially those belonging to the pathogenesis of acquired deafmutism, will, I feel convinced, only be solved by further anatomical examinations of cases of fresh otitis intima. For, as I have proved all through this paper, *the pathology of deafmutism acquired after birth is really the pathology of infantile labyrinthitis, which has come to its final stage, and caused lasting deafness.*

SECTION XVI

OTOLOGY

DISCUSSION No. 6

CLIMATIC AND OCCUPATIONAL INFLUENCES IN DISEASES OF THE EAR

Report by CLARENCE JOHN BLAKE, M.D., Professor of Otology, Harvard University

Both climatic and occupational conditions are liable to affect the organ of hearing prejudicially in various ways.

Differences in barometric pressure, in the temperature and in the humidity of the air, which constitute the main physical factors in a consideration of climate, may, one or all, produce variations from the normal in the acoustic balance represented by the sound-transmitting apparatus of the middle ear, and can be influencing elements even in the internal ear.

Occupational influences are, however, more liable to evoke a greater variety of deleterious changes, in a greater degree, and more extensive in their application, because the occupations themselves include in their whole range not only variations in atmospheric pressure, in temperature, and in humidity of the immediately environing atmosphere, artificial climatic conditions, but subject the deeper-seated sound-transmitting and perceptive apparatus of the ear to the influx of a variety of sound waves beyond the normal accommodative limit of the acoustic ear; these occupations, many of them, being moreover accompanied by a degree of mechanical disturbance which has, in some instances, a prejudicial effect upon that portion of the internal ear which serves as a peripheral organ of equilibration.

Variations in barometric pressure at any given level usually fall within the normal limit of compensation, but those incident to changes in elevation may be productive of a temporary tension anomaly which, by frequent repetition or undue prolongation, may produce deleterious effects, examples of this being found in the United States of America in transcontinental travel by railway lines crossing the Sierras, the Rocky Mountains, and the Cascade Range, and, in Canada, the Canadian Rocky Mountains, the reverse of this condition, but with similar tension anomaly production, being found, at the other end of the scale, under artificial, occupational, climatic conditions in deep mining and caisson working under degrees of atmospheric pressure incident to the mechanical necessity of the work done.

In both the climatic and occupational changes from the basic average in atmospheric pressure, either upward or downward, the prejudicial results are less when the change is gradually effected, for, precisely as an immediate change in atmospheric pressure may result in gross structural solutions of continuity, rapid change may induce hyperæmia of the tympanic mucosa or of the intra-labyrinthine structures, or even congestion and effusion, examples being found in the fluid accumulations in the middle ear and the serous, and sometimes the hæmorrhagic, bullæ upon the upper portion of the drumhead in caisson workers and divers, together with the sensory manifestations of intra-aural circulatory changes in mountain climbers and in aviators.

As would be expected, extremes of temperature and of humidity, unless excessive and in combination, have a less effect upon the ear than the more mechanically operative variations in atmospheric pressure, climatically considered, while, occupationally, caloric and hygrometric influences are more apt to be exerted upon the external than the middle or internal ear.

The contrasting effects of extreme heat and extreme cold upon the auricle, as exhibited in drawers, puddlers, pourers, casters, beaters, wire-drawers, and other hot-metal workers, firemen, stokers, and braziers, on the one hand, and persons subjected to the rigours of winter or to continuous exposure in refrigerating plants, on the other, are unified in the character of the incident lesion, an inflammatory process marked by destruction of tissue, in usually limited areas, or a more extensive distortion of the auricle as the result of the contracture following hæmorrhagic effusion and the binding effect of inflexible cicatrices, a condition comparable to that of an untreated othematoma incident to a blow upon the auricle, a form of distortion familiar not only clinically but in the busts of ancient gladiators.

The influence of natural climatic conditions upon diseases of the ear is much less in effect than the influence exerted by artificial occupational conditions, except in so far as a continuous predisposing tendency is concerned, including the local prevalence of diseases in which the ear may become secondarily affected, as, for example, the influenza endemic in the Caroline Islands, the postnasal mucorrhœas of subtropical countries, and the atrophic disorders of the mucosa in persistently dry air. Change of climate, as a part of the treatment of the great majority of the diseases of the ear, is to be regarded of value more on account of its general systemic and hygienic influence than because of any directly local bearing upon the aural condition ; changes in elevation, in temperature, and in humidity may seem variously advisable in diseases, especially of the middle ear, but their prepondérating avenue of approach is through their influence, primarily, upon the general economy.

The literature of climatic influence in diseases of the ear is both meagre and unprecise so far as other than purely clinical observations are concerned, and deals principally with the purely secondary influence

exerted upon the middle ear, through the medium of its relationship to the nasal and postnasal mucosa, and by predispositions, incident to systemic conditions, in which climate has to be considered; but the sum of their effect is far less than that pertaining in the more intensive artificial climatic conditions of the trades, including overheated and moistened air, sometimes carrying irritating vapours, or poisonous and infective dust, and variations from normal in the atmospheric pressure, all of which are mainly prejudicial in their influence upon the middle ear and the sound-transmitting apparatus, and, beyond these, the deeper effect produced by the continuous subjection of the sensory acoustic organ to the inroad of the sounds, mainly those of high pitch, incident to the mechanical operation of the trades themselves and to the economic surroundings of daily life.

In addition to the occupational implications of the auricle, trades which are productive of dust and grime contribute not only to the cases of impaired hearing as a consequence of impaction in the external canal, and to the dermatitis incident to local irritation, arsenical dust for example, and to lay attempts at its removal, but also to the secondary non-suppurative affections of the middle ear consequent upon the irritation and inflammation of the nasal mucous membrane, an influence pertaining in white-lead workers, japanners, tinners, bronze-stampers in wall-paper factories, and workers in the so-called chemical trades, where the liberation of acrid fumes is a part of the process of manufacture.

The welfare work of the trades growing in vigour, and in value, with the increase in public appreciation of its importance, is doing much to abolish prejudicial conditions, among them those which come into the consideration of the particular subject of this report, but there is one class of prejudicial occupational outputs, one by-product of mechanical industry, the most injurious of all, especially affecting the organ of hearing and sometimes, secondarily, the organ of equilibration, which has been almost the last to receive welfare consideration, that is, the deleterious effect produced by unnecessary noise.

In mining operations, in addition to the exposure to superheated air saturated with moisture and often laden with foul gases, the incident dust, in addition to its effect upon the mucous membrane of the nose and throat, and incidentally upon that of the middle ear, causes obstruction of the external auditory canal and more or less irritation according to the mechanical structure of the dust or the operation of its chemical constituents, while, in addition to these disturbances, the miners are exposed to the consequences of detonation, occurring in circumscribed spaces, without possibility of diffusion, with resultant occasional injury to the sound-transmitting apparatus, and furthermore the effects produced by shock to the internal ear.

So far as the consequences of dust, in free air, are concerned, the trades which include both woodwork and metal construction, and the founders and core-makers, together with polishers, glass-cutters, grinders,

and metal-finishers, are liable to the same disturbances in the middle ear and external auditory canal, and to the effects of the influx of loud, high-pitched continuous sounds incident to the mechanical operations of their work, it being clinically observable that high grades of progressive impairment of hearing, presumably due to degenerative processes of the labyrinth, are more common among those metal-workers whose day-labour confines them to subjection to intense sounds made up largely of overtones of high pitch, than among the workers in noises of lower pitch, beamers in cotton-mills, for example, and water-tenders in paper-mills being less subject to progressive impairment of hearing than are blacksmiths, boiler-makers, button-makers, metal-stampers, and weavers.

Gottstein and Kayser found marked impairment of hearing in 50 per cent. of smiths and machinists examined by them. Holt found marked impairment of hearing in 35 per cent. of coppersmiths, and Barr found normal hearing in a little over 9 per cent. only of boiler-makers, while Habermann reports that out of thirty-one boiler-makers there was not one with normal hearing.

The consensus of opinion is to the effect that the degree of impairment of hearing in all these cases had a definite relationship to the duration and the intensive character of the operative sound. Both ears were usually tested, but more particularly that which was habitually directed toward the sound source; subjective noises were an almost unvarying accompaniment of the impairment of hearing, but dizziness or vertigo was present only in the more advanced cases, where the upper tone limit in hearing was notably decreased. Dizziness is also more likely to be an accompanying symptom, in mill and factory operatives, when to the noise of the machinery there is added a sensible vibration.

The majority of authorities dealing with this subject regard the affection (of the hearing incident to exposure to loud sounds of high pitch) as one of the internal ear solely, but Holt expresses the opinion that changes in the middle ear and sound-transmitting apparatus are mainly the cause of the impairment of hearing, and Gradenigo makes three differentiations: labyrinthitis without middle-ear implication, chronic catarrhal middle-ear disease with labyrinthitis, and chronic advanced middle-ear inflammation with implication of the labyrinth and vertigo as an accompanying symptom, while Röpke is of the opinion that the majority of the cases of impaired hearing consequent upon exposure to loud noise are a combination of a disturbance of the labyrinth and of a catarrhal process in the middle ear; and Urbantschitsch reports a case in which the concussion of the labyrinth was accompanied by contraction of the intrinsic muscles of the middle ear.

A review of the clinical literature of this subject as distributed throughout special and general medical journals, and a reference to such compilations as those of Friedrich and Röpke shows that the majority of the occupational implications of the ear come mainly through the participa-

tion of the middle ear in incidental conditions of the nasal and naso-pharyngeal mucous membrane, with exception of those immediately resultant in changes in the labyrinth, as the effect of the influx, to an undue extent, of sound waves. These occupational sounds are divisible into two classes, sounds of sudden access and considerable amplitude, detonations, for example, and sounds of prolonged duration, intensive, of high pitch, and replete with metallic overtones.

The detonation sounds are usually of comparatively low pitch, expending their concussive effects both upon the middle ear and the labyrinth, and, when accompanied by a mechanical concussive effect, resulting, sometimes, in a solution of continuity of the sound-transmitting apparatus with a minimum degree of effect upon the labyrinth, the cases in which the labyrinth suffers the major injury, in this form of aerial concussion, being those in which the sound-transmitting apparatus remains intact.

The literature of this particular part of the subject includes the resultant effects of detonation, both underground and in the open air, the two principal contributions to the latter being the comparison of injuries produced by the discharges of artillery on land and at sea, the detonation effect having been found to be more serious in the navy than in the army, because of the circumscribed spaces and the opportunities afforded for an augmentation of the detonation by reflection and condensation, with a consequently greater tendency to transmission of the resultant concussive effects through the medium of the body, as well as through the natural channel for sound transmission.

In all the clinical observations in regard to the effect of sound waves of considerable amplitude aerially conveyed, as a part of the occupational inroad upon the organ of hearing, the evidence is mainly in favour of the supposition that the principal channel through which the internal ear is subjected to injury from this source is that through the medium of the sound-transmitting apparatus of the middle ear, and not through other bodily channels ; this view being further supported by the fact that it is not the tones of medium low pitch, those most readily transmitted by bone conduction, which are the operative factors in the causation of the progressive impairment of hearing in the majority of the cases of occupational origin, but those of comparatively high pitch which find their access to the internal ear through the sound-transmitting apparatus only because of the obstacle presented to the passage of their short sound-waves through the soft tissues of the body, in which their impulse is almost immediately lost.

The mass of reported clinical observations upon this subject has found its more intimate support in physiological experimentation only within a few years, and that by a series of investigations made by individual observers, but often in collaborative relationship, their purpose being the exact determination of the effects produced in the acoustic labyrinth as the result of subjection of animals to the continued influx of loud noises of different kinds.

The majority of these experiments included the use of very high-pitched tones produced by whistles, organ-pipes, high-pitched metallic bodies of various kinds, and metallic sounds of lower pitch under resonant conditions simulating those of the trades as exhibited in factory operatives, machinists, and boiler-makers, the general results of these observations being that the exposure of one or both ears continuously, for a period of several days or weeks, to a pure tone of high pitch, or to a mixed tone of medium low pitch with correlated high-pitched overtones, was followed by a degenerative process in the organ of Corti, beginning in the nucleated ciliated cells, progressing to the neuron, and then, secondarily, attacking the vibrating mechanism and extending to the contiguous membranous labyrinth.

The degenerative process was found to bear a measure of relationship to the intensity and duration of application of the invasive tone, and to be situated in corresponding relationship in the upper part of the first and the beginning of the second whorl of the cochlea.

Where one ear was closed to the extent of preventing the entrance of sound-waves through the normal external passage the cochlea exposed to the sound-waves was alone found to have undergone a degenerative process. Von Eichen, for example, stopped the left ear air-tight in one experiment, and, subjecting the animal to a whistle tone of C5, found only the right labyrinth affected by the characteristic degenerative process, the left labyrinth remaining normal. Care was taken, in the majority of the experiments, to transmit these experiment tones aerially only, and not through the medium of the cage or box in which the animal was confined and consequently through the body.

Gruenberg exposed the right ear of a pigeon from seven to fourteen days to the continuous tones of a Bezold-Edelmann whistle A3, A4, with resultant evidences of degenerative processes in the papilla acoustica up to one-quarter the length of the ductus cochlearis, the left ear remaining normal.

Hoessli used, as the container for his animals, a suspended section of an iron water-pipe, struck by automatic hammers to reproduce the conditions to which boiler-makers are subjected, one ear being closed by cotton and collodion, and, in some instances, both ears, in order to determine the effect of the sound corporeally conveyed. Where one ear was closed the degenerative process was exhibited in the ear remaining open; where both ears were closed the degenerative process was not exhibited, the observer concluding that air-conduction was the only medium through which the acoustic effect was produced and that the ossicular chain is a conductive and not a protective mechanism.

In concurrence with the determinations of other observers, Hoessli found that the labyrinth lesion occurred in relatively the same area in the cochlea in all the animals subjected to the influence of a continuous tone of high pitch, and that the higher the tone the lower was the area of lesion in the cochlear tube, while Habermann, in the observations

made upon boiler-makers and blacksmiths, found that the degenerative alterations in the organ of Corti, in the nerve, and in the related ganglion cells were more extensive in the basal whorl, the upper portion of the cochlea remaining normal.

Wittmarck found that with intense sounds of high pitch and even of short duration the corresponding area in the cochlea was affected in contradistinction to other observers, but placed especial stress upon the influence exerted by influx of sound corporeally conveyed.

Marx is of the opinion, from a corresponding series of investigations, that the local degenerative process in the organ of Corti is, in proportion to the intensity of the high pitch employed, not only more pronounced but more extensive in area; the high tones of a continuously blown whistle, A3 and A4, producing the recognized degenerative effects in the organ of Corti; corresponding tones of lower pitch, A2 and E2, being less intense, did not result in degeneration.

In the detonation experiments of Hoessli only the upper portion of the first whorl and the lower portion of the second whorl were affected, a result consonant with the results of Siebenmann and others, and with the clinical observations of Friedrich and Jaehne, while Yoshii reports, as the result of detonation, with middle-ear injury, rupture of the membrane of the round window in but one case, the membranes of the oval and round windows remaining intact notwithstanding the presence of petechiæ and hæmorrhage in the vicinity of the windows, there being corresponding changes in all parts of the organ of Corti, the outer and inner ciliated cells being swollen, distorted, and sometimes separated from their base, the rods distorted, and the intermediate space filled with cellular detritus.

Clinical experience has shown that with continued exposure of the human subject to intense sounds of high pitch there is evoked, in addition to a sense of fullness, subjective noise and malaise, a greater or less degree of dizziness, and the laboratory evidences of the location of degenerative changes in the lower portion of the cochlea suggest the possibility, in view of the vestibular contiguity, of a possible secondary excitation of the equilibrating end-organ.

While the demonstration of labyrinthine changes has been sufficiently precise and sufficiently extensive to emphasize the importance of prophylactic measures in reference to the noises of the trades, there is another question, still a matter of debate and open to clinical observation for its determination, and that is the possible secondary implication of the sound-transmitting apparatus of the middle ear, a change progressively decreasing the conductivity in the channel through which the causative factor in the cochlear degeneration finds access to the internal ear.

Hensen, Bockendahl, and Pollak have reported observations of apparent reflex contraction of the tensor tympani muscle in response

to high tones, and it is a not uncommon clinical experience to find in boiler-makers, machinists, locomotive engineers and firemen, in addition to a decrease in hearing for tones of medium and high pitch, both by bone conduction and aerially conveyed, objective changes in the sound-transmitting apparatus of the middle ear evidencing a thickening of the tympanic mucosa and a contraction of the tensor tympani muscle, conditions referable in many instances to such other occupational influences as sudden changes in temperature and exposure to dust and other causes inducing irritation of the nasal and naso-pharyngeal mucous membrane.

In default of these other causative conditions in cases of continued exposure to loud sound, the drumhead is often found to be more concave because of the increased contraction of the tensor tympani muscle, and more or less generally opaque because of the thickening of the mucous membrane forming the inner coat of the drumhead; it being permissible to regard both of these changes as the result of an effort for protection of the deeper-seated and more delicate sound-transmitting apparatus of the middle ear; the contraction of the tensor tympani muscle hindering the movement of the drumhead as a whole and the thickening of the mucous coat decreasing the segmental vibration.

In the earlier stages of impairment of hearing as the result of exposure to loud, continuous noise, clinical observation shows a depression of the membrana tympani consequent upon retraction of the tensor tympani muscle, this objective evidence appearing in advance of the thickening process in the middle ear.

Further clinical observations in these cases have shown that the correction of a measure of this contraction, by means of the Politzer process, of air-tight stoppage of the external auditory canal for periods of several hours daily, or two or three times in the week, preferably at night, has resulted in an improvement in the hearing for tones of medium high pitch, notwithstanding the fact of a continuation of exposure to the occupational loud noises during the daytime.

Further observations in the cases of workers in loud noises serve to substantiate the idea that the muscular contracture and consequent fixation of the sound-transmitting apparatus, supplemented by the thickening of the tympanic mucosa, serve as an obstacle to the inroad of those loud and high-pitched sounds which have been shown, by animal experimentation, to find their access to the labyrinth, with deleterious effect, especially through the natural channel of audition.

Similar changes, more especially the muscular contracture, have been found in telephone operators, and are more probably the result of the sudden and unexpected movements of the telephone armature in response to current interruptions than of the normal, ordinary use of the instrument in listening to the human voice, measurements of the intensity of the voice at the receiving instrument, as compared with that at the

transmitting end of the line, having shown so considerable a loss as to make the volume of voice received a very inconsiderable percentage of that immediately transmitted, while the concentration of the voice directly upon the ear of the operator, as is the case in the majority of the forms of apparatus including close adjustment of the telephone receiver, makes the determination of even a very moderate tone a matter of custom without effort except in so far as accommodation to the qualitative over-tones of the consonant sounds of nearest resemblance is concerned.

Repeated observations, made at intervals, in the cases of telephone operators with normal or approximately normal hearing, have shown that very little change in the hearing, even in years of service, results from the occupation, but that with changes in the sound-transmitting apparatus, with appreciable diminution of hearing upon entering the service, a further diminution is noticeable after prolonged occupation in telephone work.

The demand upon the normal hearing power in telephone operation would seem, therefore, to be so little in excess of the usual conversational demand as to cause no resultant defect.

The sum of the recorded statistics of observations in this rapidly increasing branch of public utility service is too small to make other than very broad and very general conclusions possible, and the subject is one which might advisedly receive more attention on the part of the medical profession.

Among the most complete are the conclusions arrived at by Blegvad as the result of a series of precisely conducted observations, embodied in an extended record, beyond the capacity of a brief review, but well repaying careful reading, to the following effect: In 26·4 per cent. of the 371 telephone operators, with normal hearing, who were made the subject of examination, there was a retraction of the drumhead in the ear to which the receiving instrument was usually applied, the other ear evidencing none, or only a slight retraction. The continued use of the telephone caused no depreciation of the hearing in operators with normal ears, nor was there, on the other hand, any appreciable increase in the capacity for the hearing of tones of high pitch and slight intensity, as is sometimes claimed by the operators, this claim being supported rather by the increase in the accommodative power for tones of this class and by the gradually acquired habit of eliminating mentally the coincident and the extraneous noises. The aural lesions and traumatic neuroses incident to the accidental introduction upon the telephone line of strong currents are the result of the exhibition of a loud and sudden sound, and are to be guarded against by proper protective construction in the telephone lines.

In this brief report of influences affecting the organ of hearing it is desirable to emphasize both in professional and in public estimation the injurious effects of unnecessary noise; these effects are now not

merely in the field of conjecture and of clinical experience, but, within the compass of little more than a decade, have been abundantly demonstrated by scientific research. As a part of preventive medicine, as a part of the obligation of the medical profession to the public, the furtherance of such welfare work as shall decrease the unnecessary noise of traffic and of manufacture is a distinct duty, not only for the conservation of a special sense but because of the generally exerted injury to the nervous system.

SECTION XVI

OTOLOGY

DISCUSSION No. 6

DE L'INFLUENCE DU CLIMAT SUR LES MALADIES DE L'OREILLE

Rapport par M. le Prof. G. GRADENIGO, de Turin

Sur l'invitation du comité d'organisation de ce Congrès, je me bornerai à étudier *l'influence du climat sur les affections auriculaires*, tandis que mon distingué corapporteur, le Dr Blake, s'occupera des *influences professionnelles*.

Nous entendons par l'expression *climat* l'ensemble des conditions cosmo-telluriques pouvant influencer la santé de l'homme, c'est-à-dire les variations atmosphériques et aussi celles du sol, mais en considérant ces dernières uniquement au point de vue des modifications qu'elles font subir à l'atmosphère. Par le mot *climat*, nous désignons donc la manière d'être habituelle de l'atmosphère d'un pays, sa *formule météorologique*.

Avant tout, nous ferons observer que la littérature se rapportant à l'influence du climat sur les maladies de l'oreille est très incomplète et contradictoire sur bien des points, ce qui s'explique par la complexité des phénomènes qui rendent incertaine l'interprétation des faits.

Par lui-même, le climat est la résultante de facteurs multiples qui s'influencent mutuellement. Les facteurs principaux du climat sont la température de l'air, son humidité absolue et relative, la pression atmosphérique, l'abondance des pluies et des neiges, la direction et la force des vents, la luminosité du ciel, la présence et l'absence de nuages et de brouillards, l'existence de l'ozone, de l'électricité dans l'air, etc. Il convient donc de distinguer, pour une région déterminée, les variations météorologiques de la moyenne annuelle, des saisons, des mois et jours, ainsi que les modifications météorologiques horaires, etc.

En dehors de l'ensemble des conditions climatiques, la santé de l'homme qui vit sur un point déterminé du globe, subit l'influence de quantité d'autres conditions inhérentes au pays et qui n'ont qu'une relation indirecte avec le climat.

Ainsi, les particularités de la race, les habitudes d'hygiène et de propreté de la population, la fréquence et la gravité des maladies infectieuses, aiguës ou chroniques (*tuberculose, syphilis*), la présence dans la région de certaines industries, les particularités de culture du sol, du drainage des eaux, et enfin les voies de communication plus ou moins

rapides entre la campagne et les centres urbains exercent une influence réelle sur les maladies de l'oreille, attendu qu'en ce dernier cas il advient que certaines affections ne peuvent être traitées en temps voulu par des médecins compétents. On comprend aisément combien on éprouve de difficulté à établir la part qui revient au climat dans l'éclosion des maladies de l'oreille dans une région déterminée, en présence de la complexité des facteurs que nous venons d'énumérer.

En cas de maladie auriculaire, il faut considérer un point important, c'est l'incapacité des médecins n'ayant fait aucune étude spéciale pour dépister des affections d'oreilles mal dessinées. Il est évident que les résultats des statistiques doivent être faussés pour cette dernière raison ; si l'on se basait exclusivement sur les travaux des auristes, la pauvreté des chiffres et la diversité des modes de classification adoptés par certains auteurs entretiendait un degré d'incertitude réel, quant aux résultats terminaux.

La plupart du temps, l'action du climat sur les oreilles se vérifie par la voie de la trompe d'Eustache dont les conditions de fonctionnement plus ou moins favorables sont intimement liées à l'état de la muqueuse rhino-pharyngienne, nasale et pharyngo-buccale. Une étude sur les affections auriculaires de nature climatique doit comprendre nécessairement des considérations sur l'état morbide des premières voies aériennes et plus spécialement des maladies susceptibles d'attaquer la perméabilité de la trompe, telles que les *végétations adénoïdes* chez les enfants, le *catarrhe rhino-pharyngien* et les *hypertrophies de la muqueuse des cornets* des adultes ; dans les cas d'affections aiguës, on envisagera aussi le banal refroidissement (coryza).

Il résulte de ce qui précède, qu'en l'état actuel de nos connaissances, nous ne possédons aucune indication rigoureusement scientifique quant à l'influence du climat sur les affections auriculaires, nous devons nous borner à des propositions qui une fois adoptées, pourraient résoudre dans un avenir plus ou moins éloigné la question de l'influence du climat sur les maladies de l'organe auditif.

Pour étudier les influences du climat sur l'audition à l'état normal et pathologique, il existe deux voies principales :

(I) Surveiller les modifications apportées dans l'acuité auditive ou dans les phénomènes morbides auriculaires chez un sujet suivant les modifications atmosphériques horaires ou journalières, où à la suite d'un changement de résidence.

(II) Étudier la nature et la fréquence des maladies de l'oreille dans des climats différents.

I

On n'a jamais envisagé d'une manière très précise l'action exercée sur l'acuité auditive normale par les variations atmosphériques à différentes heures du jour ou de la nuit ou au cours de plusieurs journées consécutives.

Masini s'est livré à des recherches sur ce sujet et j'ai eu également l'occasion d'en faire en me servant d'un acoumètre fort exact, celui de Stefanini.

Ces recherches mettent en lumière l'existence de variations assez sensibles du *seuil physiologique* auditif à différentes heures du jour, mais elles ne permettent pas de tirer des conclusions précises quant aux influences atmosphériques, puisque l'on a négligé d'exclure rigoureusement d'autres causes, telles que l'influence des repas, la fatigue physique et mentale, les rumeurs ambiantes, etc.

Nous possédons des détails plus étendus mais également vagues sur l'influence exercée par les variations atmosphériques horaires ou journalières sur les affections auriculaires et en particulier sur le degré d'audition et l'intensité des bruits subjectifs au cours des otites moyennes catarrhales.

Tous les auristes savent que la plupart de leurs malades éprouvent un sentiment de bien-être relatif lorsque le temps est sec, tiède ou serein ; tandis qu'ils se plaignent davantage de leur surdité et de bruits subjectifs par les jours humides et froids, où lorsque soufflent certains vents. Il est plus rare que les malades se trouvent bien par un temps humide et froid. Il est avéré que le degré de perméabilité de la trompe joue un certain rôle dans la détermination des variations brusques de l'audition ; ce serait une étude attrayante mais que l'on n'a pas encore envisagée sérieusement.

On n'a pas encore institué de recherches méthodiques sur l'effet d'un changement de séjour sur les symptômes auriculaires. On a remarqué que certains malades affectés d'otites catarrhales chroniques ou de sclérose auriculaire observent l'atténuation et la cessation des bruits subjectifs habituels, chaque fois qu'ils résident dans un pays sec et chaud, tandis que les troubles reparaissent aussitôt qu'ils vont dans une localité humide et froide, même sans avoir apporté nulle modification à leur genre de vie.

La clinique nous enseigne l'heureuse influence exercée parfois par le climat d'altitude sur l'évolution de certaines labyrinthites d'origine syphilitique congénitale ou par l'air marin sur des enfants lymphatiques ou scrofuleux atteints d'otorrhées. Il reste toujours à déterminer le mécanisme d'action des divers climats pour en formuler avec précision les indications thérapeutiques. Nous aurons plus loin l'occasion de voir que les avis sont très partagés en ce qui regarde l'influence du climat maritime.

Non seulement la température et l'humidité de l'air, mais encore la pression atmosphérique peuvent exercer une grande influence sur les manifestations morbides auriculaires, ainsi qu'on l'observe chez certains malades souffrant d'une légère insuffisance tubaire avec dysacousie, qui voient régulièrement leur surdité s'accroître dans de fortes proportions chaque fois qu'ils font des excursions de montagne qui les transportent en quelques heures ou en peu de jours à des hauteurs où la

pression atmosphérique diffère notablement de la pression des pays où ils résident.

Les effets désastreux de la pluie et du froid et de variations brusques de température au cours d'excursions de montagnes ou de longues promenades en plaine sont manifestes chez certains individus qui attribuent surtout à ces facteurs l'aggravation subite et parfois durable de leur surdité.

II

Nature et fréquence des maladies de l'oreille dans diverses localités

Plusieurs auteurs de publications ayant trait à l'influence du climat sur la nature et la fréquence des affections auriculaires dans diverses régions, ont émis des hypothèses reposant sur leurs appréciations personnelles, plutôt que sur des statistiques exactes. Lorsqu'il s'agit de régions du globe très éloignées les unes des autres, on devra s'inquiéter, en dehors du climat, de la diversité des races ; c'est surtout aux États-Unis où la race blanche et les nègres vivent sous un même climat que ces observations sont faciles à faire.

Burnett[1], E. T. Murrell[2], de Roaldès[3] ont consacré des études à la fréquence des maladies de l'oreille dans la race blanche et la race noire en Amérique. Sauf certaines divergences de détails, ces auteurs sont d'accord pour reconnaître que chez le nègre, la cavité rhino-pharyngienne, le nez, le conduit auditif externe et peut-être aussi la cavité tympanique sont plus vastes que chez le blanc, aussi les manifestations de sténose sont-elles beaucoup plus rares chez les nègres. En général, ces derniers sont moins fréquemment affectés que les blancs d'otites catarrhales et suppurées chroniques et de scléroses auriculaires.

Pour la race indienne, nous possédons un travail de J. Cursetji[4] établissant que dans l'Inde les affections auriculaires sont à peu près aussi répandues qu'en Angleterre.

Quelques publications traitent de l'influence du climat marin. Il convient de distinguer le climat de la mer du climat côtier et l'action de ce dernier vis-à-vis de l'influence des bains de mer. Les auteurs qui ont écrit sur ce sujet n'ont pas toujours tenu un compte suffisant de ces dictinctions.

Sexton[5] et Knapp[6] ont étudié l'action du climat côtier sur les maladies d'oreilles. Cozzolino[7] a insisté, dans une leçon, sur l'influence nocive de ce climat ; Moure[8] affirme que l'air marin, humide, chargé

[1] *Archives of Otol.*, vol. xvi, 1887, p. 294.
[2] Onzième Congrès international de Médecine, tenu à Washington.
[3] *Revue de Laryngologie*, vol. xxiv, p. 1169, 1895.
[4] *Ann. des Mal. de l'Oreille et du Larynx*, tom. i, 1900, p. 144.
[5] *New York Med. Record*, n° 18, mai 1878.
[6] *Zeitschrift f. Ohrenh.*, vol. viii, p. 34, 1879.
[7] Leçon faite à Naples en 1885.
[8] *Revue de Laryngol.*, vol. x, 1889, p. 145 ; Quatrième Congrès intern. d'Otologie, tenu à Bruxelles en 1888.

de sels, sujet à de brusques variations de température, aggrave les sténoses tubaires, les otites catarrhales accompagnées d'exsudation et n'améliore aucunement les otites suppurées, bien que l'auteur ait observé des cas isolés où l'air marin joua un rôle bienfaisant. Les assertions de Moure exprimées au IV[e] Congrès international d'Otologie, tenu en 1888 à Bruxelles, donnèrent lieu à une vive discussion au cours de laquelle on émit des opinions contradictoires. Kenny [1] affirma qu'en Australie, où le climat est très chaud et la plupart des villes sises sur les côtes, on prend jusqu'à trois bains de mer par jour et qu'au cours de la saison, qui dure d'octobre à juin, les affections auriculaires sont bien plus répandues, surtout par suite de l'action exercée par l'eau salée sur les téguments du conduit auditif et la pression de l'eau.

A Kœrner [2] revient le mérite d'avoir institué durant plusieurs années consécutives des recherches sur des enfants des deux sexes, âgés de 4 à 15 ans, vivant dans un hospice sur les côtes de la mer Baltique. Il a distingué diverses catégories : (*a*) les enfants originaires de la zone côtière jusqu'à 15 kilomètres de la mer ; (*b*) les enfants de la région intermédiaire (de 15 à 100 kilomètres de la mer), et finalement (*c*) les enfants provenant de l'intérieur du pays. Régulièrement les végétations adénoïdes étaient plus répandues chez les enfants des côtes (36 pour cent en moyenne), tandis qu'on les rencontrait bien moins fréquemment parmi les autres enfants (26 à 28 pour cent). Si l'on envisage l'influence des végétations adénoïdes sur la production des lésions de l'oreille moyenne chez l'enfant, on comprendra que les observations de Kœrner permettent de conclure à l'action défavorable du climat marin sur les affections auriculaires. Kœrner toutefois a éprouvé une vive surprise en constatant, à la suite des bains de mer, la guérison de plusieurs formes d'otorrhée et parfois la cicatrisation de la perforation du tympan.

Certains cas de perforation sèche se maintiennent sec en dépit des bains de mer froids.

Sur une série d'enfants habitant l'île de Héligoland, Kœrner a remarqué l'absence d'affections auriculaires graves, mais il attribue ce fait moins au climat marin qu'aux excellentes conditions hygiéniques des insulaires parmi lesquels on n'observe que rarement la tuberculose et presque jamais de maladies exanthématiques. Kœrner n'a pas eu l'occasion de contrôler la fréquence des végétations adénoïdes parmi les enfants héligolandais, mais il a relevé fréquemment de légères lésions d'oreille qui en dérivent. Moure considère que l'atmosphère marine joue un rôle délétère au point de vue auriculaire.

Contrairement à Kœrner et à Moure, Bobone, de San Remo, dans une communication lue en 1899 au Congrès international d'Otologie de Londres, soutient que l'air de la Riviera ligurienne est plus utile que nocif pour les

[1] *Comptes rendus du Congrès international d'Otologie, tenu à Bruxelles en* 1888, p. 193.

[2] *Zeitschrift f. Ohrenheilkunde*, vol. xxxiv, 1899, p. 21 ; vol. xxxvi, p. 224 ; vol. xxxviii, 1901, p. 319.

oreilles, et il proclame la rareté exceptionnelle des adénoïdiens dans la région ; de plus, il déclare que les enfants porteurs de végétations adénoïdes et originaires d'autres pays voient le tissu adénoïdien régresser en totalité à la suite d'un séjour sur la Riviera italienne.

Pour expliquer la contradiction existant entre ses résultats et ceux de Moure, Bobone prend soin de signaler les divergences des côtes maritimes. Par exemple, l'air chaud et humide des régions maritimes françaises baignées par le gulf-stream rend les muqueuses atoniques et l'organisme moins résistant envers la maladie. Le climat de la Riviera agit favorablement sur les muqueuses des premières voies aériennes, étant donné sa sécheresse et sa luminosité.

Morpurgo [1], qui s'est livré à une étude sur les enfants recueillis sur la côte aux environs de Trieste, au point de vue de la fréquence des maladies de l'oreille et de l'influence exercée sur elles par les bains de mer, a noté leur action nettement favorable sur les formes scrofuleuses.

Il résulte de ce qui précède que l'action du climat maritime sur les affections des premières voies aériennes de l'oreille n'est pas encore parfaitement élucidée.

Aussi faudrait-il dresser de nouvelles statistiques d'après une classification unifiée en tenant compte des conditions météorologiques de l'air dans les différentes localités côtières. Très vraisemblablement, l'action du climat serait variable selon qu'il s'agirait des côtes de la mer du Nord et de l'Atlantique ou des rives de la Méditerranée et de l'Adriatique.

Roques [2] se rallie également à cette opinion.

Koch [3], au contraire, a étudié l'action des bains de mer sur vingt-neuf sujets accoutumés aux immersions, et il a découvert des oreilles tolérantes et d'autres particulièrement sensibles. Il est rare que la plongée à 4 mètres n'entraîne pas certains troubles qui se manifestent parfois dès que l'immersion atteint 2 mètres ; ils consistent en sensations de pression douloureuse et en douleurs lancinantes dans les oreilles s'associant parfois à des bruits subjectifs. En sortant de l'eau, le malade éprouve une sensation de pression et une certaine atténuation de l'audition pouvant persister durant plusieurs heures et même plusieurs jours. On observe également des altérations objectives de la membrane du tympan.

Pour compléter la revue des appréciations d'auteurs divers sur l'influence exercée par le climat sur les affections auriculaires, nous citerons une publication de J. Allen Patterson [4], relative au climat du Colorado. L'air de ces montagnes aurait une action bienfaisante dans les formes de catarrhes des premières voies aériennes et dans les cas de phtisie laryngée ; les otites moyennes suppurées s'en trouveraient bien aussi, tandis que les otites catarrhales s'y aggraveraient plutôt. Ch. Nelson

[1] *Archivio italiano di Otologia*, vol. v, 1897.
[2] *Revue hebd. de Laryngol.*, 1904, partie ii, p. 188.
[3] *Festschrift des Friedrich Wilhelms-Instituts*, Berlin, 1895.
[4] *American Laryngol. Association*, 1908, p. 194.

Spratt [1] a consacré au climat du nord et du nord-est de l'Amérique, une étude dans laquelle il déclare que l'influence du climat sur la production des maladies d'oreille a été exagérée et que si dans les pays froids on observe, principalement en hiver, les inflammations aiguës et chroniques des premières voies aériennes, ce fait est dû surtout aux inconvénients du chauffage des appartements et de la température artificielle qu'on y entretient. L'auteur conclut à la fréquence des affections auriculaires aux États-Unis.

D'après R. Müller [2] il existe une maladie d'oreille propre aux régions tropicales, consistant en une tuméfaction diffuse de parois du conduit auditif externe, qui, à un certain moment, s'accompagne de violentes douleurs imputables sans doute à la propagation au périoste du processus morbide. Il se produit une surdité prononcée par suite de l'occlusion du conduit et par conséquent d'origine mécanique. Cette affection, le plus souvent bilatérale, finit par déterminer une hypérostose du conduit, toujours de la part de l'os tympanique, elle n'est pas l'apanage de la race blanche, non plus que de la race noire, mais elle est véritablement liée au climat ; on l'observe assez fréquemment dans l'Afrique orientale.

La malaria engendrerait aussi certaines variétés d'otites sur lesquelles Weber-Liel, Voltolini et F. C. Hotz [3] ont attiré l'attention. Le plus souvent on est en présence d'une affection présentant les symptômes d'une otite moyenne suppurée aiguë qui résiste et s'aggrave même par les procédés habituels de traitement et qui au contraire cède promptement sous l'action de la quinine. Longhi a décrit une forme de maladie offrant de l'analogie avec le syndrome de Menière qui serait engendrée par la malaria, aussi en raison de cette étiologie, préconisa-t-il, en cas de vertige auriculaire, l'emploi de la quinine longtemps avant que Charcot ne l'eut recommandé en se basant sur d'autres conceptions pathogéniques contre les vertiges. Plus récemment, Ferreri [4] s'est livré à l'étude de l'influence de la malaria sur les lésions auriculaires, et il a reconnu que 1 pour cent seulement des maladies d'oreilles soignées à Rome étaient imputables à la malaria, mais il ajoute que très probablement cette proportion est encore exagérée.

Nos études nous permettent de conclure que les auteurs que nous venons de citer ont apprécié l'action du climat sur les affections auriculaires, seulement d'après leur pratique personnelle et non sur des statistiques précises.

Il vaudrait mieux évaluer la totalité des maladies d'oreilles observées dans diverses localités et essayer de différencier l'action du climat d'autres phénomènes étiologiques probables, mais actuellement cette délimitation rencontre de sérieux obstacles : en limitant cette étude au nombre d'affections auriculaires observées dans plusieurs endroits, en faisant abstraction des facteurs étiologiques, nous manquons de base

[1] Ibid., p. 197.
[2] *Zeitschrift f. Ohrenh.*, 42, 1903, p. 11.
[3] Ibid., ix, 1890, p. 356.
[4] *Archivio italiano di Otol.*, vol. viii, 1899, p. 290.

fondamentale. En effet, les nombreux comptes rendus des cliniques et des consultations otologiques ne peuvent nous fournir aucune indication précise, vu que les malades sont originaires de localités plus ou moins éloignées, qu'ils sont attirés par la renommée d'un spécialiste et qu'il est par conséquent impossible d'établir un pourcentage parmi les consultants originaires d'un endroit déterminé. On tiendra compte également que dans une même ville la clientèle qui fréquente les diverses consultations otologiques est très variable ; ainsi c'est dans les cliniques hospitalières que l'on rencontre les formes les plus graves de maladies d'oreilles, celles qui relèvent de la chirurgie ; tandis que dans les petites consultations, on observe surtout les affections bénignes non-justiciables d'un traitement opératoire. Ainsi les statistiques des différentes cliniques d'une même ville fournissent des chiffres très divergents quant à la fréquence des diverses maladies.

Nous ne sommes guère plus édifiés par la statistique des complications auriculaires mortelles et le registre des autopsies des grands hôpitaux. On sait que dans ces derniers, surtout en l'absence de spécialistes, beaucoup de décès dus à des complications d'affections auriculaires sont imputés à d'autres maladies et inscrits sous les rubriques de *fièvre typhoïde*, *piémie*, *méningite généralisée*, etc.

Il est assez rare qu'au cours d'une autopsie on examine l'oreille moyenne. Étant donné que l'élaboration soigneuse des statistiques recueillies fournit des résultats très divers, on peut conclure que d'après ces documents, les maladies d'oreilles seraient beaucoup moins répandues qu'elles ne le sont réellement. Dans une intéressante étude publiée en 1894, et sur laquelle nous reviendrons ultérieurement, d'Aguanno énonce des chiffres très démonstratifs au sujet de la pratique hospitalière, pour l'Italie.

En parcourant la statistique des malades traités au cours de l'année 1887 dans les hôpitaux du royaume d'Italie, on observe une proportion assez élevée pour la Lombardie (170 cas), le Piémont (120 cas), la Toscane (96 cas), l'Émilie (58 cas), le Latium (43 cas), la Sicile (28 cas), la Campanie (24 cas), la Ligurie (22 cas), les Marches (19 cas), l'Ombrie (17 cas), les Pouilles (17 cas) fournissent des chiffres très inférieurs et concernant uniquement des lésions de l'oreille externe ! Les hôpitaux des Calabres (11 cas, *tous externes*), de la Sardaigne (5 cas), des Abruzzes (2 cas) sont à peu près nuls sur le chapitre des maladies d'oreilles, que l'on ne signale même pas dans ceux de la Basilicate.

Ainsi, durant l'année 1887, les hôpitaux d'un tiers du royaume d'Italie ne reçurent pas ou très peu de malades atteints d'affections auriculaires ! Bien qu'au cours de ces dernières années la connaissance de l'otiatrie se soit diffusée, le nombre des malades figurant dans les statistiques hospitalières varie notablement suivant la présence ou l'absence de spécialistes attachés à ces établissements.

Les chiffres publiés par certaines grandes administrations au sujet de leur personnel n'offrent pas non plus une garantie suffisante au point

de vue des maladies d'oreilles, attendu que très souvent ils n'ont pas eu recours à l'examen de médecins compétents. A ce propos, il convient de signaler l'exemple cité dans un rapport de Della Vedova [1] concernant seize administrations italiennes de chemins de fer secondaires qui sollicitées de déclarer les maladies d'oreilles existant parmi leurs employés affirmèrent qu'on n'en connaissait aucun cas. Dans la publication de Della Vedova il n'est pas indiqué s'il s'agissait dans ce cas de quelques centaines ou de quelques milliers d'employés !

Quelques Conclusions du Rapport du Prof. Gradenigo de Turin ' Sur l'influence du climat sur les maladies de l'oreille '

Nos connaissances actuelles ne nous permettent pas d'émettre une opinion exacte quant à l'influence du climat sur les maladies de l'oreille.

Les statistiques devraient pour l'avenir être faites par des médecins fournis de connaissances otologiques suffisantes : on devrait uniformer pour tous les pays les procédés d'enquête et les *critériums* de classification.

Les statistiques récentes traitant de la fréquence des maladies de l'oreille et des premières voies aériennes chez les écoliers, outre qu'elles sont basées d'ordinaire sur un nombre relativement restreint de sujets, sont fondées sur des critériums si différents, que la comparaison de résultats au point de vue de l'influence éventuelle du climat n'offre aucune garantie d'exactitude. Nous n'aurions qu'à faire un choix parmi les nombreuses statistiques publiées pour confirmer nos assertions, et il nous suffira de remémorer que, dans un résumé de fraîche date, Kobrak [2] démontre que certains auteurs ont totalement négligé les végétations adénoïdes. En ce qui concerne ces dernières, qui constituent le facteur primordial des affections auriculaires de l'enfance, l'incertitude des statistiques provient toujours de la diversité des méthodes d'examen et de classification adoptées selon les cas, au point que les résultats sont très divergents, même pour les statistiques dressées dans les cliniques otologiques, où les observations devraient être mieux prises que celles des médecins inspecteurs des écoles non-spécialistes. Il résulte d'une étude à laquelle je me suis livré [3] que le nombre des enfants affectés d'hypertrophie de l'amygdale pharyngienne relativement au total des malades soignés dans les consultations otiatriques oscille selon les pays et les auteurs, d'un minimum de 0,2 à 0,5 pour cent à un maximum de 40 et même 50 pour cent.

Évidemment certains auteurs qui ont porté leur attention sur cette affection ont tenu compte de la totalité des cas, tandis que quelques autres n'ont eu en vue que les cas les plus évidents. On comprendra que des statistiques de cette nature ne puissent fournir aucun renseignement utile quant à l'influence du climat sur les affections auriculaires !

[1] ' Lesioni professionali e traumatiche dell' orecchio,' *Relazione alla Riunione della Società Ital. di Laringologia*, Rome, octobre 1903.

[2] *Centralblatt f. Ohrenh.*, vii, p. 431.

[3] *Die Hypertrophie der Rachentonsillen*, Jena, Fischer édit., 1901, p. 223.

Fréquence des maladies d'oreilles suivant les saisons et les mois

Afin d'étudier l'action des conditions météorologiques, on a cru devoir tenir compte du nombre de maladies d'oreilles soignées dans les cliniques à diverses époques de l'année.

R. Leymann et Bürkner [1], après avoir élaboré un matériel statistique abondant, sont arrivés à conclure que les affections auriculaires aiguës atteignent leur maximum de fréquence au printemps et qu'elles sont peu répandues en automne.

Le pourcentage des malades nouveaux soignés dans les cliniques irait en augmentant selon les mois, d'après les statistiques tirées par Bürkner du *Klinisches Jahrbuch* allemand pour cinq années, de 1887 à 1892.

décembre 5,83 %	février 8,65 %
octobre 6,50 %	mars 8,70 %
septembre 6,54 %	août 8,80 %
novembre 7,23 %	juin 10,30 %
janvier 7,36 %	juillet 10,80 %
avril 8,03 %	mai 11,26 %

Nous n'avons rapporté cette statistique qu'à titre documentaire, attendu que ces chiffres ne sont rien moins que certains. Il suffira de savoir que Leymann ne put établir de corrélation entre le temps pluvieux et les affections auriculaires ; de même Hessler [2], qui a calculé pour chaque mois les cas de catarrhes des bronches, d'inflammations tonsillaires et d'otites dans les quatre garnisons de Berlin, Kœnigsberg, Munich et Strasbourg durant quatre années ne put démontrer aucun rapport direct en envisageant les variations météorologiques des mêmes mois.

Ultérieurement nous verrons que des observations faites dans d'autres années ont mis en évidence d'importants facteurs étiologiques à côté des variations climatériques au point de vue de la genèse des maladies d'oreilles.

Tous ceux qui fréquentent les cliniques otologiques aux différentes époques de l'année savent combien il est difficile de reconnaître l'influence des phénomènes météorologiques sur la fréquence des otites. Il est certain que les otites aiguës sont moins impressionnées par les changements de climats que par l'existence de maladies infectieuses épidémiques, ainsi il advient que l'on observe un pourcentage variable d'otites au cours du même mois de plusieurs années selon la production ou l'absence d'épidémies de grippe, de rougeole, etc. De plus, on considérera que la présence aux consultations d'un certain nombre de malades pendant un mois ne veut pas dire que leur affection ait débuté au cours de ce mois. Mon observation personnelle m'a enseigné qu'à Turin, la plupart

[1] *Schwartze's Handbuch f. Ohrenh.*, Bd. i, p. 373.

[2] *Blau's Encyklopädie f. Ohrenh.*, Leipzig, Vogel édit., 1900, p. 183.

des otites aiguës se manifestent au début de l'hiver ou du printemps. Beaucoup de malades de cette catégorie ne viennent pas consulter à la période aiguë, vu qu'ils guérissent par les soins de leur médecin ordinaire, ils n'ont recours aux spécialistes que s'ils tardent à se rétablir ou lorsque l'affection assume un caractère grave ou engendre des complications éloignées. Pour toutes ces raisons, les registres des cliniques n'ont qu'une valeur relative en ce qui regarde la date initiale de la maladie ; d'autre part, on sait avec quelle difficulté certains malades fournissent des indications précises au sujet de l'anamnèse.

Les maladies d'oreilles dans l'armée

Lorsque l'on veut disposer, pour les statistiques, d'un vaste matériel, suffisamment homogène, il faut étudier les affections auriculaires dans l'armée. En effet, on se trouve en présence de sujets du même sexe, et d'âge à peu près égal, originaires de diverses régions et dont le nombre est en un rapport déterminé avec la masse de la population des régions respectives. Bien entendu ces statistiques peuvent contenir des erreurs, mais, puisqu'elles nous donnent des résultats relatifs, elles nous renseignent utilement sur les maladies d'oreilles dans des pays différents.

Pour l'étude des affections auriculaires relativement au climat, il convient de prendre en considération soit les maladies qui affectent les soldats en garnison dans diverses villes soit les conscrits originaires d'une région déterminée qui sont exonérés du service militaire après avoir été reconnus atteints de lésions auriculaires graves.

Proportion des maladies d'oreilles observées parmi les soldats aux diverses époques de l'année et dans différentes régions

On a tenté, ainsi que nous l'avons vu, de recueillir des indications sur l'influence des conditions météorologiques en calculant le nombre de malades qui fréquentent les consultations otiatriques selon les saisons et on procède de même en consignant chaque mois, sur un registre, les cas d'otites survenus chez les militaires.

Un excellent résumé, publié en 1904 par Ostino[1], permet de voir qu'à côté du climat il faut encore envisager d'autres facteurs étiologiques.

La fréquence des maladies varie selon les mois dans les diverses armées. Pour l'Italie, elle atteint le maximum durant les mois de mars et d'avril, c'est-à-dire au moment du recrutement de l'infanterie ; les chiffres sont élevés aussi en juin et en juillet, vu que l'on soigne encore les maladies d'oreilles non encore réformées au cours des mois précédents et en raison de l'appel des réservistes. On observe une seconde poussée en janvier, lors de l'arrivée des recrues de la cavalerie, par le fait de l'influence du climat et des maladies infectueuses telles que la rougeole et l'influenza

[1] *Centralblatt f. Ohrenh.*, ii, vol. viii.

qui sévissent durant les premiers mois de l'année. Si, comme le fait s'est produit en 1899, la grippe et l'influenza se manifestent au printemps, le pourcentage des affections auriculaires sera plus élevé au cours des mois de mars et d'avril.

L'armée austro-hongroise présente presque les mêmes particularités. Durant la période de 1897 à 1901 le maximum fut atteint en octobre, lors de l'arrivée des conscrits ; ensuite on note par ordre les mois de janvier, mars, février, novembre, décembre : ce sont les mois d'août et septembre qui sont moins favorisés sous le rapport de l'éclosion des maladies d'oreilles tant dans l'armée autrichienne que pour les soldats italiens.

Dans l'armée prussienne, Villaret [1] a enregistré le maximum en juillet pour la période allant de 1878 à 1898 et il attribue cette constatation aux exercices de natation, à la détonation des fusils et des canons et aux traumatismes auriculaires plus fréquents pendant les manœuvres. Les affections tympaniques seraient très répandues surtout en juin et en juillet.

Scholze [2] prétend au contraire que les maladies d'oreilles se rencontrent principalement en octobre et en novembre lors de la réception des recrues. La moyenne de la période estivale (1,03 $^{1}/_{000}$) est inférieure à la moyenne des mois d'hiver (janvier, février et mars), aussi l'auteur conclut que l'influence du climat est plus nocive pour l'oreille que celle des bains.

Pour l'armée française, c'est durant les trois premiers mois de l'année que l'on observe le plus de maladies d'oreilles, tandis qu'elles sont très rares en septembre et en octobre, telles sont les conclusions adoptées par Chauvel [3] d'après les observations qu'il a recueillies dans le service d'otologie du Val-de-Grâce.

Au sujet de ces statistiques, nous remémorerons ce que nous avons énoncé ci-dessus, à savoir : que Hessler, à la suite de ses recherches portant sur des militaires, nie toute relation directe entre la fréquence des affections auriculaires et le nombre des journées pluvieuses.

Mancioli [4] a dressé pour l'armée italienne une statistique précise, dans laquelle il énumère seulement les cas d'otites aiguës, catarrhales ou suppurées, observés soit aux diverses mois des années 1898 et 1899 dans plusieurs régions d'Italie où des militaires tiennent garnison, soit par année durant 20 années (de 1880 à 1900). Mancioli établit que la moyenne des affections auriculaires observées dans l'armée atteint 6,89 $^{1}/_{000}$; les maladies sont surtout répandues en avril et en mai, elles diminuent de fréquence par la suite pour reparaître à la fin de l'été et décroître en novembre et décembre.

La fréquence des maladies d'oreilles au cours de plusieurs années atteint, en Italie, son maximum tous les quatre ans, en corrélation avec le cycle des

[1] *Deutsche militär-ärztliche Zeitschr.*, 1900, p. 25, et 1899, p. 462.
[2] Ibid., 1891, p. 693.
[3] *Archives de médecine et pharmacie militaires*, 1892, p. 161.
[4] *Archivio ital. di Otologia e Laringol.*, vol. xiv, 1903, p. 261.

maladies infectieuses aiguës. Ce sont les régions extrêmes et occidentales de la Péninsule qui sont les plus frappées. Sauf Rome et Pérouse, l'Italie centrale présente au contraire un chiffre d'otites au-dessous de la moyenne. Il semble résulter de ces observations que les otites atteignent particulièrement, dans l'armée italienne, les soldats originaires des Alpes et des pays méridionaux, soit des climats les plus froids et les plus chauds.

Ostmann [1] s'est livré à une série d'observations dans l'armée prussienne et il a remarqué que la plupart des otites ont une origine traumatique, ou sont déterminées par des tares héréditaires ou des infections locales et généralisées ; toutefois le climat joue aussi un rôle assez important. D'après Ostmann, les affections nasales et rhino-pharyngiennes seraient fort communes dans la Prusse orientale soumise à de brusques variations atmosphériques par suite de l'humidité de l'air et de la fréquence des vents. On observe la moyenne la plus basse en Prusse, dans les corps d'armée qui tiennent garnison dans la portion occidentale de la monarchie, tandis que la moyenne est très élevée dans la partie orientale ; le 4e corps d'armée résidant en Saxe est également au-dessus de la moyenne. Ostmann conclut que dans les corps d'armée habitant des régions où la température est plutôt froide (6 à 8 degrés centigrades) et où il pleut modérément (550 à 770 millimètres par an), les affections auriculaires sont plus répandues, tandis que les chiffres les plus bas sont observés dans les régions où la température annuelle moyenne atteint 8 à 10° centigrades avec une pluie modérée.

Pour la monarchie austro-hongroise, c'est en Bohême que l'on rencontre le moins d'affections auriculaires parmi les soldats.

Maladies d'oreilles évaluées parmi les conscrits des diverses régions

On peut se documenter sur la distribution géographique des affections auriculaires dans des pays différents et peut-être aussi sur l'influence du climat au point de vue de l'oreille en tenant compte du nombre des conscrits réformés pour des maladies d'oreilles. Des articles concernant cette question ont été publiés par Nimier [2] pour l'armée française et par d'Aguanno [3] pour les troupes italiennes.[4]

Les statistiques dont les matériaux ont été empruntés à l'armée offrent le réel avantage d'être la plupart du temps basées sur un très grand nombre d'observations et rédigées d'après des données uniformes. Mais il ne faut leur attribuer qu'une valeur relative, attendu que le diagnostic est souvent fait par des médecins non-spécialistes et ainsi

[1] *Deutsche militär-ärztliche Zeitschr.*, 1894.

[2] *Annales des Mal. de l'Oreille*, p. 749, 1892.

[3] *Boll. delle Mal. dell' Orecchio*, vol. xii, 1894, p. 97 et suivantes.

[4] Dans un rapport sur les maladies professionnelles publié à Rome en 1903, Della Vedova donne des statistiques relatives aux affections observées chez les conscrits de l'armée italienne durant la période de 1891 à 1900, mais ces chiffres ne peuvent nous servir pour établir un pourcentage puisqu'ils ne fournissent nulle indication sur le nombre des conscrits pour chaque région en particulier.

que le fait remarquer justement Nieddu,[1] il est impossible d'examiner attentivement l'audition au conseil de revision vu la rapidité de la visite, le milieu bruyant dans lequel on évalue et l'agitation des conscrits. Une autre cause d'erreur importante provient de ce que puisque le règlement italien prescrit pour nombre d'affections auriculaires une période d'observation dans un hôpital militaire avant de prononcer la mise en réforme ou l'ajournement, les statistiques ne comprennent-elles pas tous les sujets pour lesquels la réforme ou l'ajournement ont été décidés pour cause d'imperfections physiques ou d'altérations d'autres organes ne nécessitant pas le séjour à l'hôpital. On sait que les affections auriculaires affectent souvent des sujets atteints d'autres maladies, aussi peut-on s'expliquer que nombre de lésions auriculaires échappent pour ladite raison à la régistration du conseil de revision. Dans les statistiques de cette catégorie, on n'établit généralement pas de distinction entre les lésions traumatiques et les véritables maladies d'oreilles, ce qui expliquerait que nombre d'affections auriculaires ne figurent pas sur les statistiques des conseils de revision. Nous voyons, par exemple, d'après certains chiffres que j'ai recueillis concernant l'armée italienne, que sur 8 à 10,000 individus soumis à l'examen, on ne découvrit aucun cas ou seulement 3 ou 4 cas d'otites moyennes suppurées ; cette proportion est évidemment très inférieure à la moyenne normale.

Malgré ces lacunes, la statistique des conscrits possède une valeur relative, en admettant que les causes d'erreur pèsent d'une manière uniforme sur l'ensemble des chiffres.

Pour l'armée française, Nimier s'est occupé de la période allant de 1887 à 1890 ; il a conclu que 55 sur 10,000 conscrits étaient réformés ou versés dans les services auxiliaires en cause des lésions auriculaires.[2] En France l'influence de la race est très atténuée, en raison de la fusion des races primitives. Nimier a remarqué la rareté des affections auriculaires parmi les conscrits méridionaux, tandis que le climat des côtes de la Manche et de l'Océan exerce une influence pernicieuse sur l'ouïe des populations côtières. Le climat méditerranéen n'offre pas les mêmes inconvénients : le climat d'altitude n'a pas une action aussi marquée que celui du littoral, attendu que les plaines sises aux pieds des montagnes sont bien moins influencées par ce voisinage que les pays limitrophes de la mer.

D'Aguanno a dressé, pour l'armée italienne, une série de statistiques ayant trait aux conscrits examinés et réformés pour des affections auriculaires de 1854 à 1863.

J'ai cru devoir me servir des indications très précises recuillies par

[1] *Giornale medico del R. Esercito (italiano)*, Maggio-Giugno, 1905.

[2] La moyenne oscille de 159,96 à 12,97 pour les divers départements sur 10,000 sujets examinés, tandis que l'on verra que pour l'armée italienne la moyenne varie de 50 à 4,5 pour 10,000. Il est probable que cet écart provient du règlement ; le degré d'abaissement de l'audition compatible avec le service militaire est laissé au choix de l'expert en Italie, au lieu qu'en France il est fixé à 4 mètres pour la voix parlée et à 12 mètres pour la voix élevée.

un médecin militaire, le Dr Balla, assistant honoraire de ma clinique, pour compiler une nouvelle statistique embrassant, pour l'armée italienne, une période de neuf ans (1900 à 1909, classes 1881–89) dans le but de comparer les chiffres recueillis par d'Aguanno et par moi à 50 années d'intervalle. Les réformés ont été classés par province et par arrondissement. D'après le règlement italien, les causes de réforme sont : 1° l'absence totale du pavillon de l'oreille ou les lésions entraînant d'importantes déformations; 2° les otites suppurées chroniques ; 3° la surdité.

La surdi-mutité, dont il serait utile de connaître la répartition géographique, n'est pas mentionnée dans les publications du Ministère de la Guerre relatives aux recrues. Les calculs sont établis à la mensuration des conscrits ; on n'a tenu aucun compte des inscrits qui, pour une raison quelconque, n'ont pas répondu à l'appel et ne furent pas mesurés. On peut contrôler sur les tableaux annexés à ce rapport[1] les résultats de ma statistique personnelle et ceux de d'Aguanno. Il résulte de la comparaison que le pourcentage des conscrits réformés en Italie pour des affections auriculaires s'est accru au cours des 50 dernières années. Très probablement cette augmentation n'est qu'apparente, et provient de ce que les médecins sont plus instruits en matière d'otologie. En outre, alors que d'Aguanno, ainsi qu'on le voit sur le tableau, déclare que la région des Alpes, presque toute la chaîne des Appennins de Ligurie et en particulier la province de Massa, occupent le premier rang en Italie au point de vue de la fréquence des affections auriculaires, la statistique plus récente montre que les maladies d'oreilles sont réparties d'une façon plus irrégulière. Le chiffre le plus élevé est fourni par Côme où l'on compte 50 réformés sur 10,000 inscrits. Ensuite vient Udine qui, dans l'ancienne statistique, avait une moyenne très basse ; le contraste est frappant pour Mantoue qui, de 4 sur 10,000, atteint dans la seconde statistique le chiffre de 38 sur 10,000. On remarquera que pour certaines régions de l'Italie méridionale et des îles, la proportion est demeurée presque la même pour les deux statistiques et même certaines villes, Sassari, Lecce, Ancône, fournissent récemment des chiffres inférieurs à ceux de l'ancienne statistique. Toutefois, la plupart du temps l'augmentation est assez marquée. On ne peut, avec d'Aguanno, admettre l'influence nocive des altitudes, du voisinage des glaciers, des vents froids et humides et des variations climatiques pour expliquer la fréquence des maladies qui résultent dans ma statistique pour certaines régions. Les divergences existant entre les deux statistiques ne peuvent être expliqués par l'action du climat. Si l'on considère que les provinces où le pourcentage est actuellement le plus élevé sont celles dans lesquelles, à l'exemple de Côme, de nombreuses industries se sont implantées au cours de ces dernières années, on est fondé à croire que la fréquence des affections auriculaires est favorisée par ces industries peu salubres qui exposent la population aux maladies infectieuses entraînant à leur suite les altérations auditives. Pourtant on peut seulement avancer

[1] Ces tableaux graphiques seront présentés au Congrès par le Prof. Gradenigo.

l'hypothèse, selon laquelle les milieux industriels exerceraient une influence analogue et peut-être supérieure à celle du climat ; on manque de bases suffisantes pour pouvoir exprimer un jugement définitif à cet égard.

Conclusions

1° Nos connaissances actuelles ne nous permettent pas d'émettre une opinion exacte quant à l'influence du climat sur les maladies de l'oreille.

2° Il faudra, à l'avenir, recueillir des indications précises, et, avant tout, les praticiens devraient acquérir des connaissances otologiques suffisantes, ce qui n'adviendra que lorsque l'étude de cette spécialité sera obligatoire dans les universités.

3° Les statistiques ne devraient pas être limitées aux affections auriculaires mais s'étendre aux maladies des premières voies aériennes, attendu que la plupart du temps, les conditions climatériques n'influencent les oreilles qu'indirectement, par suite des maladies des voies aériennes.

4° On devrait uniformiser les statistiques pour tous les pays en fixant les procédés d'enquête et les modes de classification ; il faudrait aussi grouper les maladies en quelques catégories restreintes.

5° En examinant les statistiques, on tiendra compte non seulement de l'action du climat, mais encore de l'influence d'autres facteurs importants, parmi lesquels figurent en première ligne les races, les maladies infectieuses, le développement de l'industrie, etc.

6° Certaines statistiques donneraient à penser que les conditions industrielles d'une région peuvent influer davantage que les variations atmosphériques sur la genèse des affections auriculaires.

Conscrits réformés pour Affections auriculaires dans l'Armée italienne : $\frac{1}{10.000}$ distribués par Province

Régions.	*Provinces.*	*Années* 1854–1863 (*statistique d'Aguanno*).	*Années* 1900–1909 (*statistique Gradenigo*).
Piemonte.	Alessandria . .	10,59	15,46
	Cuneo	10,64	15,91
	Novara . . .	18,42	26,84
	Torino	14,88	18,08
Liguria.	Genova . . .	18,79	29,61
	Porto Maurizio .	10,60	14,35
Lombardia.	Bergamo	18,64	24,48
	Brescia	14,22	19,62
	Como	18,83	50,78
	Cremona . . .	11,45	17,89
	Mantova . . .	4,59	38,86
	Milano	14,19	27,09
	Pavia	13,26	13,20
	Sondrio . . .	22,40	33,80

Régions.	*Provinces.*	*Années* 1854-1863 (*statistique d'Aguanno*).	*Années* 1900-1909 (*statistique Gradenigo*).
Veneto.	Belluno . . .	17,20	24,18
	Padova . . .	5,45	14,63
	Rovigo . . .	12,35	15,01
	Treviso . . .	5,99	19,88
	Udine . . .	7,80	45,02
	Venezia . . .	11,21	20,66
	Verona . . .	8,68	17,45
	Vicenza. . . .	8,78	18,60
Emilia.	Bologna . . .	7,25	15,08
	Ferrara . . .	11,18	22,48
	Forlí	20,73	17,28
	Modena . . .	9,84	19,15
	Parma	14,32	19,01
	Piacenza . . .	11,16	12,50
	Ravenna . . .	11,84	17,94
	Reggio Emilia . .	4,17	21,88
Toscana.	Arezzo	10,06	17,25
	Firenze . . .	12,88	16,76
	Grosseto . . .	6,68	18,25
	Livorno . . .	15,54	19,45
	Lucca	10,88	27,02
	Massa Carrara . .	23,52	18,37
	Pisa	8,65	17,46
	Siena	8,63	20,36
Marche.	Ancona . . .	13,89	10,34
	Ascoli Piceno . .	11,97	11,73
	Macerata . . .	9,38	9,07
	Pesaro	13,40	25,34
Umbria.	Perugia . . .	4,69	10,19
Lazio.	Roma	9,36	17,73
Abruzzi Molise.	Aquila	12,05	14,59
	Campobasso . .	8,60	21,79
	Chieti	10,96	13,90
	Teramo	6,18	12,34
Campania.	Avellino . . .	10,10	11,52
	Benevento . . .	7,71	14,79
	Caserta . . .	11,83	19,46
	Napoli	10,59	17,25
	Salerno	15,52	12,97
Puglia.	Bari	12,42	16,34
	Foggia	11,38	15,13
	Lecce	15,54	8,91
Basilicata.	Potenza . . .	10,32	13,36
Calabria.	Catanzaro . . .	11,44	14,46
	Cosenza . . .	13,13	18,61
	Reggio Cal . . .	14,15	19,09
Sardegna.	Cagliari . . .	10,72	10,29
	Sassari	14,81	10,62
Sicilia.	Caltanisetta . .	12,16	15,32
	Catania . . .	13,09	13,35
	Girgenti . . .	8,96	15,40
	Messina . . .	12,59	15,19
	Palermo . . .	11,74	19,27
	Siracusa . . .	13,78	11,30
	Trapani . . .	15,87	15,51

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CONTENTS

SECTION XV: RHINOLOGY AND LARYNGOLOGY

Proceedings of Thursday Morning, August 7

PAGE

PRESIDENTIAL ADDRESS.
By Sir StClair Thomson, M.D., F.R.C.P., F.R.C.S. . . . 1

BUSINESS PROCEEDINGS 5

DISCUSSION No. 1

ON THE RECENT PROGRESS OF ENDOSCOPIC METHODS AS APPLIED TO THE LARYNX, TRACHEA, BRONCHI, ŒSOPHAGUS, AND STOMACH.
Report by Chevalier Jackson, M.D. (*see* Part I, pp. 1–34).
Report by Professor G. Killian 7
Additional Introductory Remarks by Dr. C. Jackson . . 33
Discussion by
Professor O. Chiari, Dr. W. Hill,
Dr. R. Lewisohn, Dr. H. Marschik,
Dr. W. Freudenthal, Mr. H. Tilley,
Professor I. Kubo, Dr. H. P. Mosher,
Dr. Braden Kyle, Dr. A. Logan Turner,
Dr. J. London, Mr. E. B. Waggett,
Professor J. Mouret, Mr. R. H. Skillern 37–58
Replies by Dr. Killian and Dr. Jackson 58–61

Proceedings of Thursday Afternoon, August 7

QUE DEVIENNENT LES SINUS OPÉRÉS ?
Paper by Professor E. J. Moure 63
Discussion by
Dr. Maurice, Dr. J. Guisez,
Sir Robert Woods, Mr. H. Tilley,
Dr. Marschik, Professor I. Kubo,
Dr. Trétrôp, Professor Jacques,
Dr. Luc, Mr. E. B. Waggett,
Dr. M. Halle, Dr. J. Donelan . 68–73
Reply by Professor Moure 73

DE CERTAINS PROGRÈS DANS LA THÉRAPEUTIQUE DES AFFECTIONS DE L'ŒSOPHAGE, DE LA TRACHÉE ET DES BRONCHES, ET EN PARTICULIER DE LA RADIUM-THÉRAPIE DU CANCER DE L'ŒSOPHAGE.
Paper by Dr. J. Guisez 75

REMARKS UPON FIFTY-FOUR CASES OF FOREIGN BODIES IN THE ŒSOPHAGUS, TRACHEA, AND BRONCHI.
Paper by Dr. E. Botella 89
Discussion by Professor I. Kubo 92

PAGE

RADIUM IN THE TREATMENT OF MULTIPLE PAPILLOMATA OF THE LARYNX.
Paper by T. J. Harris, A.M., M.D. 93
Discussion by Dr. W. Freudenthal 96

EXPERIMENTELLE UNTERSUCHUNGEN ZUR MECHANIK DER INTRACRANIELLEN UND CEREBRALEN KOMPLIKATIONEN DER STIRNHÖHLENENTZÜNDUNGEN.
Paper by Dr. H. Zwillinger 97

UNTOWARD RESULTS FOLLOWING THE EXTERNAL OPERATION ON THE FRONTAL SINUS. A CRITICAL REVIEW OF TWENTY CASES.
Paper by Dr. R. H. Skillern 103
Discussion by Dr. R. F. Ridpath 114

WEITERE ERFAHRUNGEN MIT MEINER METHODE ZUR DIREKTEN BESICHTIGUNG DES NASENRACHENS UND DESSEN DIREKTES PHOTOGRAPHIEREN.
Paper by Dr. A. Gyergyai 115

ZUR ANATOMIE DER UNTEREN NASENMUSCHEL.
Paper by Professor Dr. R. Hoffmann 121

EINE SCHLEIMCYSTE DES NASENSEPTUMS MIT POSTOPERATIVER HYDRORRHŒA NASALIS.
Paper by Dr. V. Frühwald 123

Proceedings of Friday Morning, August 8

DISCUSSION No. 2

(Jointly with Section XVI, Otology, Discussion No. 3)

THE METHODS AND RESULTS OF TREATMENT OF DISEASES OF THE THROAT, NOSE, AND EAR BY SALVARSAN AND OTHER ARSENICAL COMPOUNDS.
Report by Dr. A. Castex (*see* Part I, pp. 35–58).
Report by Professor Dr. P. Gerber (*see* Part I, pp. 59–110).
Discussion by
Dr. H. L. Whale, Professor Brühl,
Professor Alexander, Sir Robert Woods,
Dr. Maurice, Professor Finder,
Professor de Stella, Dr. Dundas Grant,
Dr. A. Lieven, Dr. Dan McKenzie,
Dr. H. Marschik, Dr. Kœnig . . 129–35
Professor E. V. Segura,
Replies by Dr. Castex and Professor Gerber 135

DIE BEHANDLUNG DER NARBENSTENOSEN DES ŒSOPHAGUS MIT RADIUM.
Paper by Dr. F. Neumann 137
Discussion by Professor I. Kubo 145

Proceedings of Friday Afternoon, August 8

POST-CRICOID CARCINOMA.
Paper by Mr. E. B. Waggett 147
Abstract of Paper by Mr. Wilfred Trotter 157
Discussion by
Mr. de Santi, Dr. W. Hill,
Dr. Logan Turner, Sir Robert Woods,
Dr. Marschik, Professor Uchermann 161–4
Reply by Mr. Waggett 164

PAGE

PACHYDERMIA LARYNGIS: SOME POINTS IN ITS ÆTIOLOGY AND DIAGNOSIS.
Paper by JOBSON HORNE, M.D. 165

Proceedings of Saturday Morning, August 9

DISCUSSION No. 3

INDICATIONS FOR AND RELATIVE VALUES OF TONSILLOTOMY AND TONSILLECTOMY.
Report by Professor Dr. H. BURGER (*see* Part I, pp. 111–118).
Report by Dr. J. L. GOODALE (*see* Part I, pp. 119–133).
Additional Remarks by Professor BURGER 169
Discussion by
Professor JACQUES, Sir STCLAIR THOMSON,
Mr. E. D. DAVIS, Dr. BROECKAERT,
Dr. TRÉTRÔP, Dr. W. H. KELSON,
Mr. J. F. O'MALLEY, Professor KUBO,
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Dr. F. C. PYBUS, Dr. MARSCHIK,
Dr. S. S. WHILLIS, Dr. W. M. STOOKEY,
Mr. TILLEY, Dr. M. HALLE,
Dr. W. S. SYME, Professor UCHERMANN 171–87
Reply by Professor BURGER 187

OPERATIVE BEHANDLUNG DER HYPOPHYSENTUMOREN.
Paper by Dr. O. HIRSCH 189
Discussion by
Dr. W. HILL, Dr. F. H. WESTMACOTT,
Dr. MARSCHIK, Dr. J. M. WEST,
Dr. BROECKAERT, Dr. HOLMGREN . 193–6
Dr. HALLE,
Reply by Dr. O. HIRSCH 196

Proceedings of Monday Morning, August 11

DISCUSSION No. 4

(Jointly with Section XVI, Otology, Discussion No. 5)

SPECIAL TREATMENT OF THE THROAT, NOSE, AND EAR DURING THE ACTIVE STAGES OF CERTAIN INFECTIOUS FEVERS.
Report by E. W. GOODALL, M.D. (*see* Part I, pp. 135–47).
Report by Dr. V. DELSAUX 717
Additional Remarks by Dr. GOODALL 729
Discussion by
Mr. A. CHEATLE, Dr. WATSON-WILLIAMS,
Professor JACQUES, Mr. H. TILLEY,
Dr. DUNDAS GRANT, Mr. S. SCOTT,
Dr. BRONNER, Dr. TRÉTRÔP,
Professor PICK, Professor MYGIND,
Dr. MCKENZIE, Dr. MAURICE,
Dr. KŒNIG, Professor SEGURA,
Mr. HARMER, Dr. LAUTMANN . 731–8
Reply by Dr. DELSAUX 738
Resolution 738

PAGE

DIE AKUT-INFEKTIÖSEN HALSENTZÜNDUNGEN.
Paper by Professor A. KUTTNER 197

THE PATHOLOGY OF THE VARIOUS ACUTE INFLAMMATIONS OF THE THROAT AND NECK, INCLUDING ACUTE ŒDEMA, PHLEGMON, ERYSIPELAS, AND ANGINA LUDOVICI, BUT EXCLUDING DIPHTHERIA.
Paper by P. R. W. DE SANTI, F.R.C.S. 225
Discussion on Papers by Professor KUTTNER and Mr. DE SANTI.
Sir F. SEMON, Mr. A. R. TWEEDIE,
Mr. DOUGLAS HARMER, Professor FINDER,
Sir STCLAIR THOMSON, Dr. MARSCHIK,
Dr. LAUTMANN, Mr. TILLEY . . 231–4
Reply by Mr. DE SANTI 234

DIE INTRANASALE DACRYOCYSTOSTOMIE.
Paper by Dr. L. POLYÁK 235
Discussion by Dr. M. HALLE, Dr. DAN MCKENZIE, and Mr. DOUGLAS HARMER 239–40
Reply by Dr. POLYÁK 240

Proceedings of Monday Afternoon, August 11

CHRONIC HYPERPLASIA OF THE SUPERIOR MAXILLA.
Paper by F. H. WESTMACOTT, F.R.C.S. (Eng.), B.Sc. (Vict.) . 243

CONTRIBUTION A L'ÉTUDE DES RAPPORTS BACTÉRIOLOGIQUES ET HISTOLOGIQUES ENTRE LES AFFECTIONS NASALES ET LES AFFECTIONS DES VOIES LACRYMALES.
Paper by Dr. L. DEWATRIPONT 249

LA PITUITRINE DANS LES HÉMORRAGIES OPÉRATOIRES ET SPONTANÉES DES VOIES RESPIRATOIRES.
Paper by Professor CITELLI 283

TREATMENT OF DISEASES OF THE NOSE AND THROAT BY THERAPEUTIC INOCULATION, EXCLUSIVE OF TUBERCULIN AND DIPHTHERIA ANTITOXIN.
Paper by W. DOUGLAS HARMER, M.C. (Cantab.), F.R.C.S. (Eng.) 285

NOTES ON FIFTY CASES OF NASAL DISCHARGE TREATED WITH VACCINES.
Paper by A. LOGAN TURNER, M.D., F.R.C.S.E., and N. HAY BOLTON, M.B., Ch.B. 313
Discussion on Papers by Mr. HARMER and Drs. TURNER and BOLTON by
Mr. E. B. WAGGETT, Dr. TRÉTRÔP,
Sir STCLAIR THOMSON, Dr. LYON SMITH . 317–18
Dr. DAN MCKENZIE,
Reply by Mr. HARMER 318

CONSIDÉRATIONS SUR LES MYCOSES DES MUQUEUSES DES VOIES RESPIRATOIRES ET DIGESTIVES SUPÉRIEURES.
Paper by Professor Dr. E. V. SEGURA 319

LES INFECTIONS PAR VOIE NASALE.
Paper by Dr. TRÉTRÔP 335

ÜBER DEN DIAGNOSTISCHEN UND PROGNOSTISCHEN WERT DER LOKALEN TUBERKULINREAKTIONEN AUF GRUND NEUERER UNTERSUCHUNGEN.
Paper by Dr. E. VON TÓVÖLGYI 339

PAGE

DIE THERAPEUTISCHE WIRKUNG DES BACTERIUM COLI COMMUNE IN FÄLLEN DER KEHLKOPF-TUBERKULOSE.
Paper by Dr. (E. von Tóvölgyi 349

Proceedings of Tuesday Morning, August 12

DISCUSSION No. 5

THE PATHOLOGY AND TREATMENT OF MALIGNANT GROWTHS OF THE NOSE AND NASO-PHARYNX (FIBROMA TO BE EXCLUDED).
Report by Professor G. Ferreri (*see* Part I, pp. 149–202).
Report by Dr. H. Marschik 361
Discussion by
Dr. Luc, Professor Segura,
Dr. Sargnon, Mr. T. Jefferson Faulder,
Dr. J. W. Bond, Sir StClair Thomson,
Professor I. Kubo, Mr. Tilley,
Dr. A. Jansen, Professor Jacques . 404–11
Replies by Professor Ferreri and Dr. Marschik . . 411–12

DIE ANWENDUNG EXPERIMENTELL-PHONETISCHER METHODEN FÜR DIE DIAGNOSTIK UND THERAPIE FUNKTIONELLER STÖRUNGEN DER STIMME.
Paper by Professor H. Gutzmann 413
Discussion by Mr. MacMahon and Professor Gradenigo . 421–2

WEITERE STUDIEN ÜBER DIE SOLITÄREN CHOANALPOLYPEN AUS DEN NASENNEBENHÖHLEN.
Paper by Professor Dr. I. Kubo 423
Discussion by Dr. Brown Kelly, Dr. H. Marschik, and Dr. Dan McKenzie 425–6
Reply by Professor Kubo 426

SUR UNE MÉTHODE TRÈS UTILE POUR GUÉRIR L'APHONIE HYSTÉRIQUE.
Paper by Professor Citelli 427

Proceedings of Tuesday Afternoon, August 12

CONTRIBUTIONS A LA LARYNGOSTOMIE.
Paper by Dr. Sargnon 429
Discussion by
Dr. H. Marschik, Professor von Eicken,
Mr. E. B. Waggett, Sir StClair Thomson . 442
Reply by Dr. Sargnon 442

ON THE DESIRABILITY OF A MORE EXTENDED AND DISCRIMINATIVE NOMENCLATURE FOR THE NERVES DISTRIBUTED TO THE NOSE.
Paper by L. Hemington Pegler, M.D. 445
Discussion by Sir StClair Thomson, Dr. J. Cameron . . 450

THE DEVELOPMENT OF THE OLFACTORY NERVE IN VERTEBRATES.
Paper by John Cameron, M.D., D.Sc., F.R.S.E., and William Milligan, M.D., M.S. 451

DEMONSTRATIONS.
Organized by E. D. Davis, F.R.C.S. 467

SECTION XV

RHINOLOGY AND LARYNGOLOGY

PRESIDENTIAL ADDRESS

By Professor Sir StCLAIR THOMSON,
M.D., F.R.C.P., F.R.C.S.

The last time that the International Congress of Medicine was held in London was in 1881, when, as a student, I attended some of the meetings. Although there were separate sections for otology, ophthalmology, dermatology, and odontology, yet thirty-two years ago our special department of practice had not attained to a separate, autonomous section. Rhinology was hardly recognized and laryngology was relegated to a sub-section and had no president. The sittings were presided over by a chairman selected from the vice-presidents of the section of medicine. That chairman was Sir George Johnson, my own chief, who, however, desired to be known as a general physician, although his observations on the innervation of the larynx are worthy of remembrance. The number of laryngologists who inscribed their names as members of this sub-section—this Cinderella of the specialities—was 342 and the average daily attendance at the sittings varied from 60 to 100. To-day, judging from this morning's gathering, our attendance will be numbered in hundreds.

In the year 1881 eighteen hours were given up to discussing thirty-eight papers. This year we are prepared to devote thirty hours to debating five reports and thirty-four communications.

The chairman of 1881 has been gathered to his forefathers, but all the three secretaries of that occasion are, I rejoice to say, still amongst us and in the full enjoyment of health and vigour. Dr. de Havilland Hall has been lured into the wider field of internal medicine ; Dr. T. J. Walker, in the provincial town of Peterborough, where I commenced my studies of medicine as his pupil, has won renown as a general surgeon ; but Sir Felix Semon has continued to be the devoted apostle of laryngology, and in his retirement from active practice he remains an attached follower and a detached critic.

It might be interesting if I briefly referred to the subjects of discussion in 1881, and left to all present to draw their own comparisons with the programme of work we have before us to-day, in this way showing how swift and mighty has been the progress of rhino-laryngology in these thirty-two years.

The chairman in his opening address in 1881 still felt it necessary to uphold the claims and benefits of the laryngoscope, and Manuel Garcia again narrated how the possibility of viewing the living larynx flashed across his imagination while he was strolling in the garden of the Palais Royal, that historical September day in 1854. The local treatment of diphtheria nowadays—thanks to the wonderful discovery of antitoxin, of such secondary, I might almost say, of such trifling importance—was one of the subjects of set discussion, and the reporters were Morell Mackenzie, Tobold, and Lennox Browne. Krishaber of Paris announced his firm conviction that tuberculosis of the larynx, while possibly healing in one part, yet always broke out in another, and so invariably ended fatally, generally within a year, although 'quelques cas résistent deux ans'.

A debate on laryngeal paralyses set the foundation of our now established classification of these affections and gave the early embodiment of what we know as the Semon law. It gives us pause to read that a subject of debate was 'the indications for extra- or intra-laryngeal treatment of benign tumours of the larynx', when at present all the world knows that every simple growth can be removed by the intra-laryngeal route according to the Hippocratic principle 'cito, tutto et jucunde'. But doubtless the subject was chosen to enlighten the medical public as to the possibilities of the laryngoscope, which was then only 25 years old.

Stenoses of the larynx, still in our day a *bête noire* to the laryngologist, was another subject of general discussion in 1881, one of the reporters being our *confrère* Heryng of Warsaw.

The indications for partial or complete extirpation of the larynx were discussed, but laryngo-fissure was not even mentioned during the debate. At that date and for some years afterwards the operation was so disastrous that there was little anticipation of the brilliant results we now obtain.

At our previous London Congress the galvano-cautery had hardly started on its useful, although sometimes wild, career, yet in a discussion introduced by Voltolini, Solis Cohen, Cadier, Lennox Browne, Foulis, and Victor Lange, one conclusion arrived at, even thirty-two years ago, was that the electric cautery was often used unnecessarily!

Adenoid vegetations were discussed in a masterly way by their discoverer, Wilhelm Meyer, followed by Loewenberg, Guye, and Woakes.

The everlasting subject of ozæna came in for consideration, and five other communications on various subjects completed the programme.

It is interesting to note that in 1881 there was not a single communication on the surgery of the nose, on diseases of the accessory sinuses, on the bacteriology of the air-passages, or on laryngo-fissure. At that date cocaine was unknown, adrenalin was undreamt of, and electricity was not available for illumination.

The trachea was only partly visible when reflected in the laryngeal mirror; but the hardihood of the early pioneers of rhino-laryngology

is shown by the record that Morell Mackenzie and Stoerk showed their œsophagoscopes, and the former even exhibited a patient on whom he had performed internal œsophagotomy.

Gentlemen, in the thirty-two years which have passed since an International Congress of Medicine was held in this city, rhinology has been created and laryngology has travelled far.

A glance at our programme will show that diseases of the larynx can now be viewed not only indirectly but directly, and can be surgically attacked both from the outside and through the mouth. The laryngologist has extended his province downwards until it embraces the lower air-passages and even the secondary bronchi, and we are now busy annexing the œsophagus.

Upwards the laryngologist has claimed and explored the nose and its various accessory sinuses. The sphenoidal sinus, a cavity which not so long ago was looked upon as for ever beyond the surgeon's interference, is now treated daily in every throat clinic, and before our Congress closes we shall, I hope, have shown how it can be traversed to remove disease within the very cranium itself.

Lermoyez divided the history of laryngology into three stages. The first was from the invention of the laryngoscope in 1854 up to the discovery of cocaine. This drug opened up a large field for observation and usefulness and started us on the second stage. The third stage was opened by the perfection of all the endoscopic methods for which we are indebted to Killian, his pupils and followers. Gentlemen, we are happy to have lived in these vigorous years of development of rhino-laryngology. Our speciality in the last ten years has made more marked progress than any of its fellows, and the public hardly realize the benefits it confers on the health and happiness of the community and particularly of the rising generation.

What will mark the next stage of our progress I will not venture to predict. It only remains for me as spokesman of the council of the section and on behalf of all British laryngologists to bid you one and all a hearty welcome, and to assure you, in the words of Shakespeare, that ' our true intent is all for your delight '.

Meine verehrten Herren Kollegen! Ich hoffe, dass Sie mit meinem schwachen Versuch, deutsch zu sprechen, freundlichst Nachsicht haben mögen. Wohl weiss ich, dass die meisten von Ihnen unsere Sprache mit Leichtigkeit beherrschen, ja, dass Sie zuweilen unsere Literatur besser kennen, als wir selber! Gestatten Sie mir daher, mich darauf zu beschränken, Sie im Namen unseres Komitees und im Namen der gesamten Britischen Aerztewelt herzlich willkommen zu heissen, willkommen auf unserer Insel, deren Einwohner demselben grossen Volksstamm angehören wie Sie auch. Und lassen Sie mich in diesen Willkommensgruss nicht nur Deutschland und Oesterreich einschliessen, sondern auch die Vertreter der Länder, welche mit Recht, wie ich glaube,

das Deutsch als gemeinsame Verkehrssprache für unsere Wissenschaft adoptiert haben.

Sie sind alle willkommen !

Miei cari amici e Colleghi della Penisola, — Voi, che venite dal paese che fu la culla della nostra civiltà, e la sorgente delle nostre arti, parlate una dolce lingua che deve essere sempre cara a tutti quelli, che, come noi, debbono per la professione loro interessarsi anche della voce pel canto.

Non è il caso di dirvi che personalmente io esulto al pensiero di avere ora l' opportunità di salutare quelli che vengono dal caro paese, che è per me una seconda patria, dove io ho passato sette dei più felici anni della mia vita :

Siate tutti i ben venuti.

Mes chers Confrères et Amis, — Nous savons tous que chacun de nous a deux patries — la sienne et la France. Cette situation est très agréable pour les autres nationalités, mais elle est très dure pour les Français, qui par le fait sont les seuls n'ayant qu'une seule patrie ! Nous avons l'intention de porter remède à ce grief, et avant la fin de ce Congrès, nous osons espérer qu'en retournant dans ses foyers tout Français aura deux patries, sans compter que passé ce délai les représentants des autres nations en auront peut-être trois !

Nous souhaitons cordialement la bienvenue à nos confrères de France, de Belgique, et de la Suisse, où j'ai fait mes études et où j'ai obtenu mon doctorat, aussi qu'à nos amis d'autres pays dont la langue de la culture et des rapports sociaux est le français.

Mes amis, à vous tous je vous souhaite la bienvenue.

La séance est ouverte.

[Thursday Morning, August 7]

SECTION XV

RHINOLOGY AND LARYNGOLOGY

BUSINESS PROCEEDINGS

The Secretary read a letter from Dr. H. v. Schrötter, Vienna, inviting the co-operation of the members of the Section in the investigation of scleroma, and requesting to be kept informed of all researches, publications, &c., bearing upon the subject.

SECTION XV

RHINOLOGY AND LARYNGOLOGY

DISCUSSION No. 1

ON THE RECENT PROGRESS OF ENDOSCOPIC METHODS AS APPLIED TO THE LARYNX, TRACHEA, BRONCHI, ŒSOPHAGUS, AND STOMACH

(For Report by Dr. Chevalier Jackson, see Part I, pp. 1–34)

Referat von GUSTAV KILLIAN, Berlin

Die direkten Methoden in den Jahren 1911 und 1912

Mit den direkten Untersuchungsmethoden der Luftwege und der oberen Speisewege haben wir uns seit Erfindung der direkten Laryngoskopie und besonders im letzten Jahrzehnt ausserordentlich eifrig beschäftigt. Viele zusammenfassende Arbeiten sind erschienen. Auf den internationalen Laryngologen-Kongressen in Wien, Budapest und Berlin wurden Referate gehalten. Ich glaubte mich daher auf eine Durchsicht der Literatur beschränken zu sollen, welche in den letzten beiden Jahren 1911–12 erschienen ist. Das ausführliche Referat Kahlers auf dem Berliner Kongress reicht bis zum 1. Januar 1911.

Auch bei dieser Beschränkung blieb mir noch eine grosse Arbeit zu leisten. Handelte es sich doch darum, über 460 Titel und Arbeiten zusammenzubringen und durchzusehen.

Die genannte umfangreiche Literatur erstreckt sich auf viele Nationen und ist zum Teil in wenig geläufigen Sprachen niedergelegt. Sie konnte daher nur unvollständig und an der Hand zum Teil recht kurzer, den Inhalt keineswegs umfassender Referate bearbeitet werden. Trotzdem glaube ich, dass die Uebersicht über die Literatur der letzten beiden Jahre, welche ich Ihnen zu geben gedenke, alles Wesentliche umfassen wird.

Mehr wie früher haben wir uns in den letzten Jahren mit der Geschichte der direkten Methoden befasst. Es gelang mir, zu zeigen, dass sich von kleinen Anfängen nach und nach alle Kulturnationen eifrig mit diesen Methoden befasst haben. Genaue historische Darstellungen gaben in dem Ausstellungskatalog des Berliner Kongresses 1911 Mann und Gottstein.

In einer Reihe von Lehrbüchern wurden die direkten Methoden und

die damit behandelten Krankheiten ausführlich beschrieben. Besonders hebe ich hervor die neuen Darstellungen von Starck über die Oesophagoskopie und von Mann über die direkte Laryngo-Tracheo-Bronchoskopie in dem grossen Handbuch von Katz, Preysing und Blumenfeld. Ferner das Buch von Lemaître, *Application de la Broncho-Oesophagoscopie à la chirurgie du Thorax.* Eine kurze Darstellung der Oesophagoskopie habe ich selbst in der ' Neuen chirurgischen Operationslehre ' von Bier, Braun und Kümmel gegeben. Eine wertvolle Bereicherung erfuhren unsere Kenntnisse über die Krankheiten der Speiseröhre durch die zweite Auflage des Buches von Kraus, welche von Ridder mitbearbeitet wurde. Auch die Hoffmann'sche *Darstellung der Krankheiten der Bronchien* hat eine neue Auflage erlebt.

Was nun die direkten Methoden selbst angeht, so sei gleich eingangs erwähnt, dass sie durch v. Gyergyai und Yankauer eine Anwendung auf den Nasenrachenraum erfahren haben, wobei hervorgehoben werden muss, dass schon A. Hartmann, Lindt und andere die direkte Untersuchung des Nasenrachens in Angriff nahmen. Wie es scheint, kann mit der direkten Rhinoscopia posterior in diagnostischer und therapeutischer Hinsicht einiges geleistet werden. Auch die Ohrtrompete lässt sich jetzt mit dem Rohr erreichen und kann eine Strecke weit besichtigt werden.

Was die direkte Laryngoskopie betrifft, so gehen wir kaum fehl in der Annahme, dass das, was die Literatur der letzten beiden Jahre darüber gebracht hat, keineswegs auf Vollständigkeit Anspruch erheben kann. Die direkte Laryngoskopie wird in der Praxis oft verwendet. Viele halten es aber nicht für der Mühe wert, darüber besonders zu berichten. Wir müssen sie ausserordentlich hoch einschätzen, denn sie hat uns in diagnostischer und therapeutischer Hinsicht wesentlich weiter gebracht. Dies lehren auch die neueren Arbeiten.

Bezüglich der Anwendung der Narkose bei kleinen Kindern zeigt sich neuerdings, besonders in Amerika (Jackson), die Tendenz, möglichst ohne Betäubung auszukommen. Paunz betont aber mit Recht, dass wir uns die Vorteile der Narkose nicht sollten entgehen lassen. Schon früher hat Jackson einen Spatel angegeben, bei dem das Chloroform durch ein besonderes Röhrchen geblasen wird. Neuerdings kam Neumann in Wien wieder auf diese Technik zurück, um sich dadurch eine gleichmässige und hinreichende Betäubung des Patienten zu sichern. Es unterliegt keinem Zweifel, dass es schwer ist, die Kinder in eine genügend tiefe Narkose zu bringen. Auch wachen sie leicht daraus auf, wenn man mit der Untersuchung beginnt. Die Narkose wird ungleichmässig, weil sie während der Manipulationen im Halse stets unterbrochen werden muss. Das Jackson'sche und Neumann'sche Nebenröhrchen erlaubt eine kontinuierliche gleichmässige Chloroformzufuhr. Jedoch muss unter erhöhtem Druck mit einem Gebläse gearbeitet werden. (Junker'scher Narkose-Apparat.)

Hartmann und Yankauer haben den Röhrenspatel konisch gestaltet. Das obere Ende ist weit, das untere eng. Ihnen folgte Hill und fügte

noch einen seitlichen Schlitz bei. Derselbe hat einen gewundenen Verlauf und ist so breit, dass er den Stiel eines durchzuschiebenden Innenrohres hindurchgleiten lässt.

Die Einführung der Röhren und Spatel geschieht beim Erwachsenen wie immer im Sitzen. Auch bei Kindern in Narkose hält Paunz diese Haltung für die bessere. Anstatt der Rückenlage gebe ich in neuerer Zeit der Seitenlage den Vorzug und erlaube mir, daran zu erinnern, dass ich dies in New York seiner Zeit in dem Hubbard'schen Falle einer Reihe von Kollegen vorgemacht habe. Später wurde die Seitenlage von Mosher und neuerdings von Jackson besonders empfohlen. Sie erleichtert die Einführung des Röhrenspatels wesentlich. Man steht vor und nicht hinter dem Kopf des Kindes. Der Speichel läuft aus dem Mundwinkel heraus. Die Kopfhaltung kann leicht reguliert werden. Es empfiehlt sich, bei gewöhnlicher gerader Kopfhaltung oder mässig rückgebeugtem Kopf zu beginnen, eine Methode, auf die schon früher Kirstein und später Johnston hingewiesen haben. Kirstein machte darauf aufmerksam, dass man bei vorgebeugtem Kopf die hintere Larynxwand besonders leicht sieht. Auch von Claoué wird dies neuerdings betont.

Der Brünings'sche Gegendrücker wird wenig erwähnt, leistet jedoch gelegentlich gute Dienste. Wie ich selbst angab und jetzt auch Pollatschek hervorhebt, kann man den Gegendrücker leicht durch Fingerdruck auf den Ringknorpel ersetzen. Den Patienten ist es oft unangenehm, sich zwischen Rohr und Gegendrücker eingeklemmt zu fühlen.

Eine neue Verwendung hat die direkte Laryngoskopie bei der Melzerschen Narkose gefunden. Man muss dabei einen dicken Katheter, durch welchen ein Gemisch von Sauerstoff und Aether geblasen wird, durch die Stimmritze tief in die Trachea einführen. Es handelt sich um die verlängerte Kuhn'sche Tubage. Die Einführung des Katheters hat sich Elsberg mit der direkten Laryngoskopie erleichtert. Lautenschläger verwandte meinen teilbaren Röhrenspatel dazu.

Der kongenitale Stridor der kleinen Kinder bedarf einer noch weiteren Aufklärung durch die direkte Laryngoskopie. Auch Paunz fand die Epiglottis eigentümlich schnabelförmig zusammengedrückt und beobachtete im Anfall, wie sie geradezu in den Larynx hineingesaugt wurde. Aehnliches sah ich in Bezug auf die Arygegend. Man fängt jetzt an, diese Erscheinungen als sekundäre aufzufassen. Da Kehlkopf und Luftröhre meist normal weit gefunden werden, Thymuskomplikationen in der Regel nicht vorliegen, so muss an eine primäre Neurose gedacht werden, die wohl in Beziehung mit dem Stimmritzenkrampf steht. Beim Laryngismus stridulus werden die Stimmlippen inspiratorisch genähert.

Akute Athembehinderungen sind bei kleinen Kindern oft durch eine subglottische Schwellung, wie bekannt, herbeigeführt. Die direkte Untersuchung zeigt dies mit aller Klarheit. Selten dürfte, wie in dem Fall von Caussade und Willett, eine Perichondritis dahinter stecken.

Stimmbandknötchen hat Guisez in 10 Fällen beim Erwachsenen direkt entfernt. Er bediente sich dabei besonders exakt gebauter Zangen.

Die Resultate waren sehr befriedigend. Auch Goldbach empfiehlt das gleiche Vorgehen. Gelegentlich wurden Kehlkopfpolypen direkt entfernt (Setien und Quix), wenn der indirekten Entfernung besondere Schwierigkeiten entgegenstanden. Auch in Simanowski's Klinik ging man bei Larynxtumoren direkt vor (Wojatschek).

Die wichtigste Rolle spielt die direkte Laryngoskopie bei den Kehlkopfpapillomen der Kinder. Wir lesen neuerdings wieder viel darüber. Paunz unterscheidet die leichter zu behandelnde umschriebene Form von der schwereren diffusen, bei der die Papillome schnell und immer wieder rezidivieren, und die manchmal unheilbar erscheint. Ich werde bei der Schwebelaryngoskopie auf dieses Thema zurückkommen.

Zur Behandlung der Kehlkopftuberkulose versuchte Brünings die Röntgenbestrahlung endolaryngeal und direkt durchzuführen, stiess jedoch auf erhebliche praktische Schwierigkeiten. In dem Bestreben, möglichst lange Belichtungszeiten zu erzielen, werden die Kranken zu sehr angestrengt. Auch macht die Angst vor den Funken viel zu schaffen. Ausser einer schmerzstillenden Wirkung und einer Reinigung der Geschwüre waren die bisherigen Erfolge wenig befriedigend.

Bemerkenswert sind die Angaben von Paunz über Kehlkopfsyphilis bei kleinen Kindern, die an Heiserkeit litten und stridorös atmeten. Er fand Anschwellungen der Kehlkopfschleimhaut und entdeckte in einem Fall ein Geschwür an der hinteren Wand. Die antisyphilitische Behandlung ermöglichte, die Tracheotomie zu vermeiden.

In Fällen von erschwertem Dekanülement leistet die direkte Laryngoskopie fortgesetzt grosse Dienste. Sie gibt Aufschluss über die Ursache und führt zu einer rationellen Behandlung. Dies zeigt sich wieder in den Mitteilungen von Sargnon und Abrand, Schmiegelow und Uffenorde. Abrand hat bei einem dreijährigen Kinde durch Intubation herbeigeführte Verwachsungen im Kehlkopf direkt getrennt. Uffenorde entfernte von oben Granulationen. Schmiegelow empfahl die Behandlung der Stenosen nach Tracheotomie und Intubation mittelst Drainröhren, welche mit Faden oder Draht am Halse befestigt werden. Das Gummirohr wird später von oben entfernt. Mayer und Yankauer haben eine subglottische Stenose vom Munde aus mit bronchoskopischen Röhren dilatiert.

Bemerkt sei, dass man gelegentlich anstatt eines Fremdkörpers eine subglottische Schwellung oder subglottische Diphtherie findet (Zytowitsch). Ich kann dies aus eigener Erfahrung bestätigen. Auch ist es vorgekommen, dass anstatt Diphtherie ein Fremdkörper entdeckt wurde.

Zur Entfernung von Fremdkörpern aus dem Kehlkopf erwies sich die direkte Laryngoskopie wieder sehr nützlich. Wittmaack entfernte bei einem 11 monatigen Kinde ein Knochenstück, bei einem 2 jährigen den Wirbel eines Aals aus dem Kehlkopf. Von Paunz wurden in zwei Fällen ebenfalls Knochen glücklich entfernt. Reinking extrahierte ein Knochenstück direkt von oben, nachdem das $1\frac{3}{4}$ jährige Kind wegen Atemnot hatte tracheotomiert werden müssen. Ich erwähne noch eine

Schraube (Goldbach), Agraffe (Claoué), Fischschale (Guisez), eine Nadel und eine Sicherheitsnadel (Paunz). In diesen 5 Fällen handelt es sich ebenfalls um kleine Kinder.

Beim Erwachsenen haben Uffenorde und Kubo eine Münze und Fischgräten direkt entfernt. Middleton versuchte einen Kürbiskern aus dem Kehlkopf herauszuziehen. Derselbe fiel dabei in die Bronchien und wurde ausgehustet.

Die Schwebelaryngoskopie

Eine Modifikation der direkten Untersuchung des Kehlkopfes ist meine Schwebelaryngoskopie, über welche ich schon auf dem Berliner Kongress Mitteilung gemacht habe. Das Instrumentarium wurde seitdem wesentlich verbessert. Ausser mir hat auch mein Schüler Albrecht mit bestem Erfolg daran gearbeitet. Die Methode hat jetzt eine allgemein brauchbare Form erlangt. Es sind Mitteilungen von Albrecht, Hölscher, Wolff, Brieger, Hinsberg, Spiess, Lautenschläger, Davis, Pollatschek, Freudenthal und Kämpfer und mir gemacht worden.

Mittelst der Schwebelaryngoskopie gelang es, besonders bei den Kehlkopfpapillomen der kleinen Kinder, gute Erfolge zu erzielen. Auch bei der Behandlung der Kehlkopftuberkulose hat sie uns gute Dienste geleistet.[1] Ueble Zufälle haben sich bisher nicht ereignet. Die Aufhängung des Kopfes an einem Spatel wird leicht ertragen. Man kommt leichter zum Ziel, wenn man, wie ich das neulich auf der Versammlung Deutscher Laryngologen demonstriert habe, zuerst den Zungengrund in die Höhe hebt und dann mit besonderem Heber den Kehldeckel aufrichtet und das Larynxinnere einstellt. Ein Gegendruck auf den Ringknorpel erleichtert wesentlich die Einstellung der vorderen Commissur. Albrecht gibt den Gegendruck mit der Brünings'schen Vorrichtung, welche für seinen Hebehaken besonders eingerichtet wurde. Aus der Literatur und den Verhandlungen in Stuttgart gewann ich den Eindruck, dass die Schwebelaryngoskopie nicht allein in meinen Händen, sondern auch in denen anderer sich bewährt hat.

Die obere und die untere Tracheoskopie

Die Technik dieser Verfahren hat in den letzten beiden Jahren verschiedene Verbesserungen erfahren, was bei Besprechung der Bronchoskopie noch weiter ausgeführt werden wird.

Von angeborenen Veränderungen der Trachea ist ein Divertikel zu erwähnen, welches Kahler bei einer Frau auf direktem Wege gefunden hat.

Aus der Klinik der direkten Tracheoskopie erwähne ich einen Fall Gerbers, der von Max Zimmermann genau beschrieben wurde. Es handelte sich um eine Fraktur der Luftröhre, gefolgt von Stenosenbildung.

[1] Wie Brieger und Davis zeigten, lassen sich in der Schwebe auch Fremdkörper aus Rachen und Speiseröhre bequem entfernen. Auch Operationen im Nasenrachen und Rachen, insbesondere die Tonsillektomie wurden mit Vorteil in der Schwebe ausgeführt (Albrecht, Killian, Freudenthal).

Zimmermann stellte die ganze Literatur von Trachealfrakturen zusammen (19 Fälle). In dem Gerber'schen Falle gelang es, mit der direkten Tracheoskopie nicht allein die Verengerung sichtbar zu machen, sondern auch mit dem Rohr bis zu den unterhalb der Stenose gelegenen Gebieten vorzudringen und diese aufzuklären.

Blutungen aus Venektasien der hinteren Trachealwand beseitigte Ephraim mit Chromsäureätzungen.

Was die akuten Infektionskrankheiten angeht, so hat die untere direkte Tracheoskopie zur Entfernung von Membranen wieder gute Dienste geleistet (Paunz, Zytowitsch). Wichtig ist, zu wissen, dass diphtherische Prozesse in der Luftröhre mit Stenosenbildung ausheilen können. Solche Fälle wurden von E. Mayer und Yankauer und von Wojatschek beschrieben. Hier handelte es sich um ein 16 jähriges Mädchen, das nach Diphtherie seit 8 Jahren kurzatmig geworden war. Es hatte sich in der Luftröhre eine Membran gebildet, die nur ein sehr enges Lumen übrig liess. Aehnliches dürfte nach den Untersuchungen von Oppikofer auch nach Scharlach zu erwarten sein. Von 128 Scharlachsektionen ergaben 14 nekrotisierende Entzündungen in der Trachea. Paunz hatte Gelegenheit, bei einem Kanülenkind im Verlauf von Masern mit unterer Tracheoskopie eingetrocknete Sekretmassen aus den Bronchien zu entfernen. Auch betont er mit Recht den Wert der Methode bei Tracheotomierten, insbesondere, wenn sich Schwierigkeiten für die Atmung einstellen.

Guisez berichtet von einer Hernie der hinteren Trachealwand, Tóvölgyi hat die chronische Tracheitis erfolgreich mit dem Spray behandelt. Die Lösungen wurden durch das Rohr eingeblasen.

Die Literatur über die syphilitischen Veränderungen der Luftröhre hat eine wesentliche Erweiterung erfahren. Gerber fand 3 cm unterhalb des Larynx ein Gumma ; es ging von der hinteren Trachealwand aus und ragte tumorartig vor. Antisyphilitische Behandlung führte Heilung herbei. Durch Zerfall der gummös erkrankten Partien entstehen Ulcerationen, welche einen grossen Umfang annehmen können, wie in dem Fall von Denker. Hier zeigten sich bei der Sektion auch die Lungenspitzen erkrankt. Patient starb an einer Pneumonie im rechten Unterlappen, welche sich im Anschluss an eine langdauernde Demonstration entwickelt hatte.

Offenbar handelte es sich bei der Kranken, von welcher Iwanoff erzählt, um Syphilis. Autoptisch fand man bei ihr die ganze Trachealschleimhaut wegulceriert. Alles war eine grosse Wunde.

Wenn der Zerfallsprozess weiter fortschreitet, so kann ein Durchbruch in die Speiseröhre erfolgen (Basch). Der Patient geht dann leicht an Bronchopneumonie zu Grunde (Schmiegelow). Interessieren dürfte es, dass pathologisch-anatomisch auch ein Durchbruch in den Aortenbogen beobachtet wurde (Stumpf).

Bei der Heilung der syphilitischen Ulcerationen entwickeln sich Narbenstenosen. Szmuolo berichtet über eine hochsitzende, Lorenz über eine tiefsitzende diaphragmenartige Narbe. Auch zeigte dieser Fall

narbige Veränderungen im peritrachealen Gewebe. Durch solche Vorgänge können bekanntlich Kompressionsstenosen der Luftröhre und der Bronchien entstehen.

Die Kasuistik über Sklerom erfährt durch Mitteilungen von Gerber, Mayer und Yankauer, Galebski, Erbrich und Wojatschek eine Erweiterung. Bald handelte es sich um kleine, blasse, wandständige Infiltrate, bald um ringförmige Veränderungen der Luftröhre. Auch die Gegend der Carina wurde infiltriert gefunden, wodurch die Eingänge in die Hauptbronchien eine Verengerung erfuhren. Gleichzeitig waren meist Nasenrachen und Nase erkrankt. Die tracheoskopische Behandlung, Auskratzungen und Dilatationen, leisteten einigen Nutzen.

An gutartigen Geschwülsten in der Trachea sind beobachtet: ein Fibrom (Gereda), es sass an den letzten Ringen; Papillome (Guisez, Döderlein); ein Enchondrom (Guisez). Der letztere erwähnt auch einen Granulationspolypen. Ueber die Tracheopathia osteoplastica hat Hagar eine interessante Arbeit veröffentlicht.

Kompressionsstenosen durch Strumen, Aneurysmen dürften wohl kaum noch viel interessieren. Anders verhält es sich mit Veränderungen des Trachealrohres durch tuberkulöse Lymphdrüsen. Meist handelt es sich um Kinder. Paunz beobachtete allein 4 Fälle. Zwei waren merkwürdiger Weise durch Fremdkörperaspiration kompliziert. Schwere Erscheinungen ruft der Durchbruch der Drüsen in die Luftröhre hervor (Koch). Zytowitsch eröffnete mit dem Röhrenspatel einen in die Trachea vorgewölbten Drüsenabscess.

Auch einfache Induration der Drüsen wirkt gelegentlich deformierend auf die Luftröhre. Näheres haben wir auf tracheoskopischem Wege neuerdings durch Gerber und Guisez erfahren. Gerber fand bei einem 43 jährigen Manne einen kleinen fingerdicken, zapfenartigen, graurothen Tumor im Bereiche der Carina. Es handelte sich um eine grosse anthrakotische Drüse, welche die Bronchialwand durchbrochen und die Knorpel zur Usur gebracht hatte. Ueber dem Tumor bemerkte man eine grosse, pigmentierte strahlige Narbe, offenbar von einem früheren Durchbruch herrührend. In Guisez's Fall war die Trachea durch Drüsen von hinten nach vorn komprimiert.

Am häufigsten gelangen in der Trachea Carcinome zur Beobachtung, und zwar sowohl primäre (Tetens Hald), als auch sekundäre nach Pharynxcarcinom (Botella) oder Uteruscarcinom (Döderlein). Mediastinaltumoren beschreiben Schlemmer und Caboche. Der Tumor des ersteren war ein Lymphosarkom der Thymus und hatte die Trachea durchwachsen. Die Geschwulst von Caboche hat offenbar aus dem Mediastinum die Luft- und Speiseröhre durchbrochen.

Ueber Fremdkörper der Trachea haben eine ganze Reihe von Autoren berichtet. Leider konnten einige Arbeiten nicht verwertet werden, weil die Referate nicht ausreichten. Auch ist in einer ganzen Reihe von Fällen die Natur des Fremdkörpers nicht angegeben. Dies sind allein 30. Die obere Tracheoskopie kam in 31, die untere in 5 weiteren Fällen zur

Beobachtung, so dass wir im ganzen über 65 Fälle berichten können. Bei weitem die meisten wurden glücklich von oben extrahiert. Dabei handelt es sich, wie immer, in der Mehrzahl der Fälle um Kinder. Das jüngste war 9 Monate alt. Nur in zwei Fällen werden posttracheoskopische subglottische Schwellungen erwähnt. Die Art der Fremdkörper war eine sehr differente. Ausser Nadeln, Gräten, Fruchtkernen, Metallhaken, Gebissplatten, sei auch ein Blutegel sowie das Stück eines Schrötter'schen Rohres und eine Intubationskanüle, welche beim Extubieren in die Luftröhre gerutscht war, besonders erwähnt. Es befindet sich auch ein Fall darunter, bei welchem bei der Operation in Narkose ein Stück Rachentonsille aspiriert worden war (Guisez). Trotz der schweren Atemnot gelang die Extraktion von oben. Makuen hat in der Trachea eine Sicherheitsnadel geschlossen und dann extrahiert. Die untere Tracheoskopie wurde in 5 Fällen zur Entfernung von Kanülenstücken, Agraffen und einem Pflaumenkern mit bestem Erfolg ausgeführt.

Manche Autoren haben ganze Serien von Fremdkörpern tracheoskopisch entfernt. So Segura, Holmgren, De Lens, Mink, Guisez und andere. Unglücksfälle scheinen sich keine ereignet zu haben.

Die obere und die untere Bronchoskopie

Wesentliche Verbesserungen hat die Technik der Bronchoskopie in den letzten zwei Jahren nicht erfahren. Die Fortschritte beziehen sich meistens auf das Instrumentarium. Einiges wird bei der Oesophagoskopie noch erwähnt werden.

Für die Untersuchung von kleinen Kindern habe ich zur Vermeidung von subglottischen Schwellungen einen Satz von besonderen Röhren angegeben. Das System der Verlängerung des Röhrenspatels mittelst eines durchgeschobenen Rohres musste der engen Raumverhältnisse des subglottischen Gebietes wegen aufgegeben werden. Ich verwandte nur ein einfaches, möglichst dünnwandiges Rohr, das vorne zur Vermeidung von Kratzeffekten gut abgerundet war. Die Einführung geschieht mittelst eines hohlen Mandrins vorsichtig durch den subglottischen Raum. Die Weite desselben wurde zuerst an frischen Kehlköpfen von Kinderleichen mittelst durchgeführter Bolzen genau bestimmt und danach die Weite der bronchoskopischen Röhren gewählt. Ich blieb jeweils $1-\frac{1}{2}$ mm hinter der zulässigen Weite zurück. Die Untersuchungen ergaben, dass die Weite des subglottischen Raumes in viel innigerer Beziehung zur Körperlänge, als zum Alter der Kinder steht. Deswegen ist auf jedem Rohr verzeichnet, für welche Körperlänge es passt. Meine bisherigen Erfahrungen mit diesen Röhren waren alle günstig. In neuerer Zeit habe ich sie etwas länger gemacht.

Das bronchoskopische Rohr versah Neumann mit einem Nebenröhrchen, durch welches das Chloroform eingeblasen wird. Zur Zerstäubung von Flüssigkeiten innerhalb der Trachea und der Bronchien gab Ephraim besondere Instrumente an. Brünings empfiehlt einen Bronchialschützer,

der zwei Kränze von Borsten trägt und vor der Extraktion in den gesunden Bronchus eingeführt wird, um die Aspiration des Fremdkörpers auf die andere Seite zu verhindern. Von Viren enthält der Fischer'sche Katalog eine neue Nadelzange und einen neuen Nadelbrecher. Für Gebissplatten gab Kahler ein besonderes Zerkleinerungsinstrument an. Die Sekrete sucht Yankauer dadurch zu entfernen, dass er am Rohrende einen Bläser rechtwinklig anbringt, der mit einer halben bis zwei Atmosphären Ueberdruck arbeitet. Einen neuen Untersuchungsstuhl verdanken wir De Lens, Kahler einen hochstellbaren Untersuchungstisch, der dem Untersucher erlaubt, im Stehen in das Rohr zu sehen.[1]

Hinsichtlich der Verwendung der Narkose habe ich das Nötige schon bei der direkten Laryngoskopie erwähnt. Die Einführung des Rohres geschieht in verschiedenen Körperhaltungen, nach meiner Ansicht am besten am narkotisierten Patienten in linker Seitenlage. Wenn nötig, dreht man ihn dann auf den Rücken. Johnston und Hill stellen den Larynx zuerst mit dem Spatel ein und führen dann das Rohr ein. So habe ich es in der allerersten Zeit der Bronchoskopie auch gemacht. Paunz bevorzugt die sitzende Haltung bei Kindern in der Narkose und legt die Kinder dann eventuell um.

Ein Bronchialdivertikel wird von Kahler beschrieben. Scoloff gelang es, eine Bronchialblutung mit bronchoskopischer Anwendung von Adrenalin zu stillen. Die Blutung war durch einen aspirierten und bald wieder ausgehusteten Knochen entstanden und stammte aus dem linken Bronchus.

Aus dem Gebiete der akuten Infektionskrankheiten finde ich die Mitteilung von Oppikofer beachtenswert, dass bei Scharlach die nekrotisierenden Entzündungen in seltenen Fällen bis in die Bronchien reichen. Die chronischen Bronchialkatarrhe erfahren durch die Spraybehandlung Ephraims bedeutende Besserungen. Guisez behandelte eitrige und fötide chronische Bronchitis mit 'huile gomélonée' und hatte dabei gute Erfolge. Auch bei Bronchiektasien soll Novocain-Adrenalin-Spraybehandlung nutzen (Ephraim).

Aehnliche Veränderungen wie in der Trachea finden wir bei der Syphilis in den Bronchien. Hier kommen ebenfalls gummöse Verdickungen, Ulcerationen, Granulationen und Narbenstenosen vor (Finder, Jurasz, Denker, Cimenez). In dem Denker'schen Falle reichten die Narben und Ulcerationen, wie die Sektion ergab, bis zu den kleinsten Bronchien. Die Dilatationsbehandlung ergibt bei syphilitischen Bronchialstenosen sehr günstige Resultate.

Zwei Fälle von skleromatösen Veränderungen der Bronchien und Dilatationsbehandlung mit dem Bronchoskopierohr erwähnte Wojatschek.

Von gutartigen Tumoren in den Bronchien nenne ich ein kleines Fibrom, welches den linken Bronchus verstopfte (Gereda) und einen Fall von E. Mayer, in dem leider die Natur des Tumors nicht genauer zu

[1] Zum Aufsuchen von Fremdkörpern mittelst Röntgenschirmes hat die Instrumentenfabrik von Scherer (Bern) einen sehr brauchbaren Tisch angegeben.

bestimmen war. Ein Bronchialcarcinom bei einem 23 jährigen Mädchen beschreibt Ephraim.

Nicht allein die Trachea, sondern auch die Bronchien werden durch anthrakotische Drüsen komprimiert und deformiert. Die Anwesenheit solcher Drüsen gibt sich bronchoskopisch durch schwarze Narbenflecken zu erkennen, welche man in der Bronchialwand beobachtet (Mann). Wenn die anthrakotischen Drüsen verkalken, so bilden sich Steine, die in die Luftwege durchbrechen können und dann ausgehustet werden. In einem solchen Fall von Lungensteinen konnte ich bronchoskopisch die Stelle feststellen, an der der zuletzt ausgehustete Stein durchgebrochen war. Sehr bemerkenswert finde ich, dass Rehn, wie Betke mitteilt, anthrakotische Drüsen, welche die Trachea und den rechten Hauptbronchus umlagerten, mittelst Mediastinotomia longitudinalis operativ entfernt hat, wodurch die Atemnot, an welcher der Patient litt, wesentlich vermindert wurde.

Tuberkulöse Lymphdrüsen brechen leicht in die Bronchien durch, wofür Koch ein prägnantes Beispiel anführt. Die Bronchialdrüsentuberkulose fand in neuerer Zeit durch Silbermann eine gute zusammenfassende Darstellung. Menzel sah die Teilungsstelle der Luftröhre und die medialen Wände beider Bronchien durch Einwirkung von Drüsen verbreitert und vorgewölbt.

Sehr bemerkenswert sind die ausführlichen Mitteilungen von Kahler über die Vorhofskompressionen des linken Bronchus. Durch Vergrösserung des linken Vorhofs wird der linke Bronchus stärker auf die Seite gedrängt. Er kann sogar abgeknickt werden, denn dieser Bronchus liegt auf dem linken Vorhof wie auf einem Kissen. Kahler hat 11 hierhergehörige Fälle bronchoskopisch untersucht.

Auf dem Gebiete der Fremdkörper in den Bronchien hat sich wieder eine lebhafte Bewegung gezeigt. Es ist eine bemerkenswerte Tatsache, dass in den letzten 14 Jahren mehr Bronchialfremdkörper publiziert wurden wie in dem ganzen Jahrhundert vorher. Man achtet jetzt vielmehr auf Fremdkörperaspirationen. Man glaubt mehr an die Anwesenheit der Fremdkörper, auch wenn nur geringe Zeichen vorhanden sind. Man findet sie leichter, wenn das Röntgenbild positiv ausfällt. Man sucht eifriger nach ihnen. Man hat den Wert einer guten Fremdkörperanamnese mehr zu schätzen gelernt. Die Aerzte führen die Fälle viel häufiger dem Spezialisten zu. Leider gehen doch noch viele Patienten an Fremdkörpern zu Grunde, wie gelegentliche Sektionsberichte zeigen. Auch werden oft akute und chronische Lungenleiden durch Fremdkörper herbeigeführt, deren Anwesenheit nicht beachtet wird. Die Wirkung der aspirierten Fremdkörper hat Wojatschek experimentell studiert. Die Schwierigkeiten aber erwiesen sich als sehr grosse, sodass vorläufig noch nicht viel bei diesen Experimenten herauskam.

Dass die Bronchoskopie zum Nachweis von Fremdkörpern viel wertvoller ist als die Röntgenuntersuchung, zeigt eine Arbeit v. Eicken's. Wir ersehen daraus, wie oft die Röntgenuntersuchung negative Resultate

ergibt. Von 40 Fällen mit Knochenaspiration waren nur 4 im Röntgenbild positiv. Auch metallische Fremdkörper können sich vor dem Röntgenschirm verbergen.

Die mittelst der bronchoskopischen Extraktion erzielten Heilerfolge zeigen in sehr günstigem Licht die Zusammenstellung von Guisez. In 22 eigenen Fällen erzielte er 20 Heilungen. Nur zwei starben trotz der Extraktion an präbronchoskopischen Lungenkomplikationen.

Aus den letzten beiden Jahren konnte ich 128 Fremdkörperfälle zusammenstellen, welche mit oberer Bronchoskopie behandelt wurden. Dies sind aber keineswegs alle. Eine ganze Reihe konnte, weil ungenügend beschrieben oder referiert, nicht berücksichtigt werden. Und wie viele mögen nicht publiziert worden sein, meiner eigenen gar nicht zu gedenken.

Unser Material stammt von vielen verschiedenen Laryngologen aus aller Herren Länder. Einzelne Namen anzuführen, würde sich kaum lohnen. Sie werden, soweit erforderlich, im Folgenden zitiert.

Manche Autoren haben neue und alte Fälle vermengt dargestellt, so dass es nur bei einer Gesamtbearbeitung aller Fälle möglich sein wird, genau zu sichten.

Was alles beobachtet und extrahiert wurde, könnte ein kleines Museum füllen. Ich erwähne 15 Fälle von Nadeln verschiedenster Art, von der einfachen Näh- und Stecknadel bis zur Haar-, Krawatten-, Tuch- und Hutnadel. Metallische Körper waren es 21, darunter Nägel, Agraffen, Eisenfeilspähne, Münzen, Bleistiftschützer, Gebissplatten, Pfeifchen, 2 Intubationsröhren und ein offenes Federmesser (Guisez). Dazu kommen eine Cigarrenspitze, ein Stück eines Bleistiftes, eine Puppe aus Celluloid.

Knochenstücke sind in 25 Fällen beobachtet worden, in einem Falle zwei bei demselben Patienten.

Eine besondere Abteilung bilden die Zähne, Zahnfragmente, Cement- und Goldkronen. Es folgen Perlen, Knöpfe, ein Kork, ein Wattetampon.

Besonderes Interesse verdienen die Rachentonsillenstücke, welche bei der Adenoidenoperation in Narkose aspiriert worden waren (Guisez).

Aus dem Pflanzenreich habe ich alle möglichen Fruchtkerne: harte, bröckelnde, weiche und quellbare zu erwähnen (42). Etwas schwierig haben sich die Kaffeebohnen erwiesen; auch die Pflaumenkerne. Viel Sorgen und Mühe haben wieder 12 aspirierte Bohnen verursacht und trotz allem einige üble Ausgänge herbeigeführt.

Die überwiegende Zahl der Fremdkörper betrifft Kinder (von 79, in denen das Alter angegeben ist, 54, also $\frac{2}{3}$). Besonders stark tritt in der Statistik das Alter von 6 Jahren abwärts hervor. Unter 1 Jahr zähle ich 5 Fälle. Das jüngste Kind war 3 Monate alt. Es folgen solche mit 6, $8\frac{1}{2}$, 9 und 11 Monaten. Etwa 1 Jahr alt waren 7. $1\frac{1}{2}$ Jahre: 5, 2 Jahre: 5 Kinder. Der älteste Patient zählte 60 Jahre.

Die meisten kamen in den ersten Tagen zum Arzt. Doch hatten manche Fremdkörper Wochen (8), Monate (8) und 1–3 Jahre (5) in den Bronchien gesteckt.

Die rechte Lungenseite herrschte wieder vor (55), doch war auch der linke Bronchus reichlich bedacht (36). 3 Mal ist der Fremdkörper von einer auf die andere Seite gewandert ; in einem Fall erst nach $\frac{5}{4}$ Jahren.

Die Röntgenuntersuchung hat sich wieder bei vielen Fremdkörpern als negativ erwiesen, insbesondere auch bei fast allen Knochen, Pflaumenkernen etc.

Die Extraktion gelang mit der oberen Bronchoskopie von 128 Fällen in 106 ganz, in 7 weiteren in Bezug auf Stücke des Fremdkörpers. Der Rest wurde ausgehustet oder mit unterer Bronchoskopie entfernt.

In einem Falle veranlasste die zurückgebliebene Nadel (deren Rosette glücklich extrahiert worden war) Haemoptoe und Tod. 3 Mal kam es nicht zur Extraktion, weil der Fremdkörper, nachdem er gesichtet worden war, ausgehustet wurde. Mehrere Male war der Fremdkörper zu gross, um direkt durch den Larynx extrahiert werden zu können. Die Möglichkeit der Extraktion war auch ausgeschlossen in einem Fall, in dem über die ganze Lunge zerstreute Nussteilchen zu zahlreichen kleinen pneumonischen Herden geführt hatten. Patient starb.

Von den 12 Bohnen haben 5 den Tod herbeigeführt. Drei fielen bei dem Extraktionsversuch auf die gesunde Seite und bewirkten Erstickung, weil die kranke nicht mehr atmen konnte. Zwei Kinder gingen trotz Aushustung, bezüglich nach Extraktion mit unterer Bronchoskopie an schon vorher bestandener, also präbronchoskopischer Pneumonie zu Grunde. Auch eine Kaffeebohne fiel auf die andere Seite und führte den Tod herbei. Ein Nusskern, der den Exitus veranlasste, war von oben nicht gefunden worden. Zwei Kirschkerne und Perlen bereiteten Schwierigkeiten und konnten von oben nicht entfernt werden, ebenso ein Zwetschgenkern. Die bewegliche Perle machte einen Erstickungsanfall und nötigte zur Tracheotomie. Im Ganzen betrug die Zahl der Todesfälle 11.

Es will viel bedeuten, wenn von 128 Fremdkörpern der Bronchien 117 mit *oberer* Bronchoskopie zu einem guten Ende kamen. In Prozenten betrug die Zahl der Todesfälle 8,7 gegen 9,6 in den Jahren 1909–11 und 13 Prozent in den früheren (304) Fällen. In der laryngoskopischen Zeit zählte man 30 Prozent und vordem 52 Prozent Todesfälle.

An üblen Folgen erwähne ich : 3 mal subglottische Schwellungen bei kleinen Kindern, welche die Tracheotomie oder Intubation nötig machten. 1 mal entstand nach schwierigen Extraktionsmanövern ein Pneumothorax, der aber wieder zur Ausheilung kam.

Ausser bei Bohnen sind auch sonst noch Pneumonien beobachtet worden in 6 Fällen, jedoch kamen diese alle zur Heilung. Ein Stück Feige bewirkte einen Lungenabscess und führte zum Tode. 1 mal ereignete sich eine Blutung bei einer Knochenextraktion. Sie kam wieder zum Stehen.

Von den Extraktionen ist zu beachten, dass eine Nadel in der Mitte gefasst und ins Rohr gezogen wurde, eine andere eingespiesste musste zuerst zurückgeschoben, die Zahnkrone aus Gold musste gedreht werden. Bei einem Knochenstück gelang die Extraktion durch Ansaugen.

Untere Bronchoskopie bei Fremdkörpern

Im Ganzen konnten 61 Fälle zusammengestellt werden. Darunter Nadeln (3), Nägel (4), Messinghaken, Schuhösen, eine Glaskugel mit Haken, eine Cigarrenspitze, eine Gummiblase mit Mundstück, eine Bleistiftkappe. Ferner Knochen (4), Kanülenstücke (6), Glasperlen (3), ein Stück Kork, eine Eierschale, ein Pflaumenkern, Kirschenkerne (2), eine Kaffeebohne, Mandeln- und Nussstücke (2), Sonnenblumenkerne (4), Kürbiskerne (5), Melonenkerne (3), Fichten- und Johannisbrotkerne (2), eine Erbse, neun Bohnen, ein Apfelgehäuse, eine Pflanzenhülse.

Unter diesen Fällen befinden sich 33 Kinder und nur drei Erwachsene, Sechs Patienten waren Kanülenträger, von den übrigen waren zehn wegen Atemnot tracheotomiert worden. Bei einem musste die Trachea eröffnet werden, weil der Fremdkörper zu gross war und nicht durch den Kehlkopf hindurchging. Bei elf hatte die obere Bronchoskopie nicht, bei zwei nur teilweise zum Ziele geführt. Es musste daher die untere Bronchoskopie versucht werden. In einem Falle bekam Patient bei der oberen Bronchoskopie einen Erstickungsanfall und bedurfte des Luftröhrenschnittes.

Der Fremdkörper sass 24 mal im rechten und 18 mal im linken Bronchus. Die Extraktion gelang 40 mal glatt und 4 mal nur teilweise, d. h. es wurden nur Stücke entfernt. Bei den Extraktionsmanövern wurde der Fremdkörper ausgehustet 2 mal, eine Zeit lang später 2 mal. Die Extraktion misslang 4 mal, 3 mal wurde nichts mehr vorgefunden.

Ein von Kümmel beobachteter Fall kam mit einem Pneumothorax an, der draussen durch Extraktionsmanöver im Blinden (von der Tracheotomiewunde aus) entstanden war, eine Warnung für solche, welche das Vorgehen unter Führung des Auges für unnötig halten.

Gestorben sind im Ganzen 11 Kinder (von 61) = 18 Prozent. Dieser Prozentsatz ist doppelt so hoch, wie der bei der oberen Bronchoskopie beobachtete. Es ist das nicht zu verwundern, denn eine Reihe von schwierigen Fällen bleibt stets der unteren Methode vorbehalten.

Von zwei Bohnenkindern starb eines an präbronchoskopischer Pneumonie ; das andere kam schon moribund auf den Operationstisch und starb, bevor der gesuchte Bohnenrest heraus war. Der schon genannte Pneumothoraxfall von Kümmel hatte einen Kürbiskern im linken Bronchus und kam zum Exitus trotz gelungener Extraktion. Aehnlich ging es mit einem Melonenkern. Ein Kind starb an der Aspiration einer Pflanzenhülse, welche auch mit der unteren Methode nicht gefunden worden war.

Dass eine Erbse bei einem Erwachsenen schwere Atemnot und schliesslich den Tod herbeiführen kann, sollte man nicht für möglich halten. Und doch musste ein Patient von Sargnon an einer aspirierten Erbse zu Grunde gehen. Er war Phthisiker und konnte nur noch mit seinem rechten Unterlappen atmen. In diesen aber war die Erbse

begreiflicherweise aspiriert worden. Die hochgradige Atemnot zwang alsbald zur Tracheotomie. Eine durch die Bronchoskopie entstandene Excoriation in der Trachea führte zu einer heftigen Blutung und beschleunigte den Exitus.

Ein Nagelfall starb 11 Tage nach gelungener Extraktion noch an Pneumonie. Ein zweiter ähnlicher Fall kam zum Exitus dadurch, dass bei der Extraktion der Nagel in den linken Bronchus fiel.

Wojatscheks Fall starb an Pneumonie, nachdem der Metallkörper $2\frac{1}{2}$ Monate im linken Bronchus gesteckt hatte und die Extraktionsversuche sich als vergeblich erwiesen hatten. Paunz verlor ein Kind mit einer Bleistifthülse an Erschöpfung. Besonders unglücklich verlief ihm ein Fall mit einer Cigarrenspitze im Bronchus. Bei der Extraktion hatte das spitze Ende der Cigarrenspitze sich in die Trachea eingespiesst, was Emphysem, eine Hautphlegmone und eine tödliche Mediastinitis zur Folge hatte.

Rechnen wir obere und untere Bronchoskopie zusammen, so ergeben sich 10 Prozent Todesfälle.

Es unterliegt keinem Zweifel, dass das Gesamtresultat noch wesentlich verbesserungsfähig ist. Vor allem wird es beeinflusst durch die vielen übel ausgegangenen Bohnenfälle. Wenn man darauf dringt, dass bei Bohnen so rasch als möglich und *stets mit Tracheotomie* vorgegangen wird, so brauchen die meisten dieser unglücklichen Kinder nicht zu sterben. Die obere Bronchoskopie ist bei Bohnen nur zulässig, wenn man sie ein paar Stunden nach der Aspiration ausführen darf. Die Bohne ist dann noch klein, hart, nicht gequollen und lässt sich leicht entfernen, wie ein Fall von Paunz lehrt.

Die von Pieniazek und Nowotny inaugurierte Asthmabehandlung mit oberer Bronchoskopie und Einpinselung bezüglich Einsprayen von Cocain und Adrenalin in die Bronchien hat in den letzten Jahren einen grossen Aufschwung genommen. Man beschäftigt sich jetzt allgemein damit und sammelt Erfahrungen. Es liegen eine ganze Reihe von Mitteilungen vor (Mahler 27 Fälle, Freudenthal 11 Fälle, Erbrich, v. Schrötter, Yankauer, Kafemann, Gallusser, Heilkow, Sobernheim etc.). Viele, wie ich selbst, haben ihre Erfahrungen noch nicht mitgeteilt. Wir verdanken diesen grossen Aufschwung den anregenden Arbeiten von Ephraim, der unermüdlich für die Adrenalinspraybehandlung Propaganda macht und auch durch wissenschaftliche Studien Klarheit in die Frage der Adrenalinwirkung und des Asthmas überhaupt zu bringen sucht.

Um dem Kranken die öftere Einführung des Rohres zu ersparen, hat er eine biegsame Röhre empfohlen, welche durch die Glottis zu den Bronchien hinabgeleitet wird und bei passender Körperhaltung in den rechten oder linken Bronchialbaum gelangt.

Aus der grossen Zahl von behandelten Fällen konnte Ephraim 88 längere Zeit weiterverfolgen. 16 zeigten keine Dauererfolge. In 72 aber ergaben sich befriedigende Resultate.

Von 48 Patienten waren rezidivfrei:

nach $1\frac{1}{2}$–3 Monaten	3
nach 3–6 Monaten	12
nach 6–9 Monaten	9
nach 12–18 Monaten	10
über 18 Monate	8

Von diesen Kranken waren

7 nur 1 mal,
6 nur 2 mal,
5 nur 3–4 mal gesprayt worden.

Die Erfolge sind also sehr vielversprechend. Dies will um so mehr bedeuten, da es sich um ein Leiden handelt, dem auf andere Weise sehr schwer beizukommen ist. Am wenigsten günstig sind nach Ephraim die Fälle mit nervösen Komponenten. Die andern Autoren haben ebenfalls im allgemeinen Befriedigendes zu verzeichnen. Freudenthal hat 8 geheilt und 3 gebessert. Mahler hat von 27 nur 5 geheilt und 10 gebessert (mit 2–6 maliger Behandlung), bei 12 keinen Erfolg.

Wie es scheint, darf man mit der Adrenalindosis nicht zu zaghaft sein; 10–15 Tropfen einer 1 $^0/_{00}$ Lösung dürfen beim Erwachsenen eingesprayt werden.

Dass das Adrenalin der heilende Faktor ist, beweist seine ausgezeichnete Wirkung gegen den Anfall bei subkutaner Injektion. Nach Ephraim bringt diese Substanz die kleinen und kleinsten Gefässe in den Bronchien zur Kontraktion, beseitigt Hyperämie, Stauung, Sekretanhäufung und macht die Wege frei. Nach seinen neuesten Beobachtungen verändert sich auch die Schleimhautoberfläche durch reichliche Abstossung des Flimmerepithels. Vielleicht ist darauf die Dauer der Wirkung zurückzuführen. Nach eigenen Erfahrungen kann ich den Adrenalin-Novocainspray warm empfehlen.

Die Erkrankungen der Lunge, insbesondere auch die akuten, müssen die Bronchoskopie nicht kontraindizieren. Die Erfahrungen mit den Fremdkörpern der Bronchien haben gelehrt, dass man bei schweren akuten Bronchitiden mit reichlichem flüssigem, eitrigem, fötidem Sekret bronchoskopieren darf, ohne Schaden anzurichten. Ja selbst bei pneumonischer Erkrankung wird die Untersuchung ertragen. Crosby Greene hat bei zwei Pneumonikern nach Fremdkörpern bronchoskopisch gesucht, ohne zu schaden.

Bei der Lungengangrän verspricht die Einträufelung von Gomenolöl gute Erfolge. Guisez empfiehlt es warm und behauptet, damit wesentliche Besserungen und selbst Heilungen erzielt zu haben. Auch Lereboullet erzählt von einem solchen Fall.

Guisez hat sich, wie es scheint, auf Einträufelung des Oels in die Trachea mit der Spritze unter Leitung des Spiegels beschränkt. Es werden auf einmal 10 (bis 20) ccm eingebracht. Es war eine Reihe

solcher Applikationen nötig. Anstatt des 5- bis 10 proz. Gomenolöls wurde auch eine Mischung mit Guaiacolöl zu gleichen Teilen verwandt. Das Hineinlaufen der Substanzen in die rechte oder linke Lunge sucht man durch entsprechende Körperhaltungen zu begünstigen. Es ist klar, dass die direkte Tracheo- und Bronchoskopie hier nützlich werden kann. Auch beim Lungenabscess versucht Guisez diese Behandlung.

Bei Lungenechinokokken hat die Bronchoskopie noch nicht viel Gelegenheit zur Verwertung gefunden, und doch könnte sie einiges leisten, zumal die Blasen recht häufig in die Bronchien perforieren. Diagnostisch leistet allerdings das Röntgenbild schon sehr Gutes. Man sieht ring- und scheibenförmige Zonen. Es dürfte doch wohl möglich sein, mit dem Bronchoskop Veränderungen festzustellen und das Platzen der Blasen zu begünstigen oder herbeizuführen.

Bei den Lungengeschwülsten wird wohl mehr bronchoskopiert worden sein, als die Literatur zeigt. Ich verfüge auch über einige nicht publizierte Fälle. Am meisten hat Ephraim berichtet. Nach Garrè und Quincke kommen auf 11,000 Sektionen 31 primäre, 135 metastatische Lungencarcinome.

Die Lungentumoren sind häufiger, als man gewöhnlich annimmt (Garrè und Quincke). Die meisten gehen von den Bronchien aus. Die Diagnose wird nur in einem Teil der Fälle gestellt. Die Bronchoskopie muss das ändern. Ephraim sah 2 Carcinome, eines im rechten, das andere im linken Bronchus bei einem Mädchen von 23 Jahren, ein metastatisches (vom Uterus aus) Endotheliom im linken Bronchus und einen Amyloidtumor in der Bifurkationsgegend.

Auf diesem Gebiet gibt es noch viel zu tun. Ebenso auf dem der Lungenchirurgie, wo die Bronchoskopie noch gar nicht nennenswert herangeholt wurde, es sei denn zur Einführung des Katheters bei der Meltzer'schen Narkose. Wir müssen, da die Fälle meist nicht direkt zu uns kommen, unsere Hilfe mehr anbieten. Bronchoskopie und Probeexcision können rasch Aufklärung und Frühdiagnosen herbeiführen.

Die Oesophagoskopie

Die Oesophagoskopie hat in den letzten beiden Jahren eine weitere Ausgestaltung erfahren.

Die Lehre von den Krankheiten der Speiseröhre und ihrer Behandlung wurde durch vielfältige Arbeit wesentlich gefördert. Auch haben wir ausführliche lehrbuchmässige Darstellungen erhalten, so von Lemaître, Hill, Einhorn, Starck und insbesondere von Kraus und Ridder. Ihre zweite Auflage bringt die Speiseröhrenerkrankungen mit erschöpfender Verwertung der pathologischen Anatomie. Wir können uns da sehr viel Wissen holen.

Was die Oesophagoskopie im Speziellen angeht, so hat Guisez zusammenfassend über 930 Untersuchungen, Gottstein über 1100 berichtet. Die Ausführung erwies sich nur in 1 bis 2 Prozent unmöglich. 3 Patienten

Gottsteins sind an Perforationen, welche sich durch Hautemphysem anzeigten, gestorben. Jackson giebt 3 Prozent Todesfälle bei Fremdkörpern und grossem Material an, für die einfache oesophagoskopische Untersuchung 1 Prozent.

Das oesophagoskopische Instrumentar wurde von Gottstein eingehend historisch dargestellt. Auch Lemaître beschäftigt sich mit der Geschichte der Methode.

Die Beleuchtung wird von den verschiedenen Schulen in der bekannten Weise gehandhabt. Eine warme Empfehlung liess neuerdings Aach der Innenbeleuchtung angedeihen. Er gibt eine Beleuchtungsprobe auf photographischem Papier (in der Dunkelkammer aufgenommen), die anscheinend zu Gunsten der Innenbeleuchtung spricht.

Die Brünings'schen Röhren beherrschen das Feld nach wie vor. Doch haben einzelne Aerzte Neuerungen versucht, die sich wohl kaum allgemein einbürgern werden. Hill konstruierte ein langes konisches, seitlich geschlitztes Rohr, welches oben reichlich weit ist und excentrisch am Stiel sitzt. Diese Anordnung erlaubt, mit den Zangen neben dem Beleuchtungsspiegel des Brünings'schen Handgriffes vorbeizugehen. Solche Röhren sind auch auf Verlängerung eingerichtet. Durch dieselben kann ein Rohr von entsprechendem Kaliber geführt werden. Der Stiel gleitet durch den Schlitz des primären Rohres abwärts.

Optik haben bei der Oesophagoskopie nur Glücksmann und Lewisohn beibehalten. Der letztere glaubte auf die alten Nitze-Leiter'schen Winkelrohre zurückgreifen zu müssen mit dem Unterschied, dass er Aussenbeleuchtung anwandte.

Der grosse praktische Wert der Erweiterung des Oesophagus bei der Entfernung voluminöser Fremdkörper wurde erst neuerdings anerkannt. Glücksmann hat schon lange an seinem Instrument vorn einen Ballon, der mit Wasser gefüllt den Oesophagus dilatiert. Brünings gab ein speculumartig dilatierbares Instrument an. Killian modifizierte diese Form; ihm folgten Hill und Tapia. Den schon von Brünings empfohlenen Modus der Aufblasung des ganzen Oesophagus hat Aach als besonders nützlich bezeichnet.

Speziell zur Fremdkörperextraktion gab Mosher einen Tubus an, der zugleich als Zange dient. Corbin verwandte zu demselben Zweck ein Doppelrohr; das Innenrohr trägt federnde Krallen, die durch Vorschieben des Aussenrohres geschlossen werden.

Botey glaubte die Einführung des oesophagoskopischen Rohres dadurch erleichtern zu können, dass er das Rohr mit einem Mandrin versah, der den Durchblick erlaubte und vorne mit Glas halbkuglig abgeschlossen war.

Erwähnt seien noch Instrumente von Mosher zum Zerschneiden von Zahnplatten und Schliessen von Sicherheitsnadeln, sowie ein Münzenextraktionshaken von Botey.

Zur Ausführung der Oesophagoskopie verwendet Jackson bei kleinen Kindern im allgemeinen weder Narkose noch Cocain. Nur in besonderen

Fällen macht er davon Gebrauch. Auch Johnston fängt erst bei Kindern über sechs Jahren damit an. Er fürht sein Rohr unter Leitung des direkten Laryngoskops von Jackson in den Oesophagus. Botella stellt sich hinter den Patienten, drückt mit dem graden Spatel die Zunge nach vorn und leitet so das Rohr in den Oesophagus. Den Verzicht auf den Mandrin hat Wojatschek als besonders nützlich bei Fremdkörpern empfunden.

Wenn man die Ergebnisse der oesophagoskopischen Untersuchungen der Lehre von den eigentlichen Erkrankungen der Speiseröhre gegenüberstellt, so ergibt sich, dass vieles am Lebenden noch nicht oesophagoskopisch festgestellt wurde, was die pathologische Anatomie und die Klinik beschreibt. Es ist daher unsere Aufgabe, unsere Kenntnisse über alle möglichen Vorkommnisse tunlichst zu bereichern, damit wir an unsere Untersuchungen genügend vorbereitet herantreten. Auch die einfachen Sektionsbefunde verdienen unsere Aufmerksamkeit. So hat Schlaefke gefunden, dass Oesophagusblutungen nicht ausschliesslich bei Störungen im Pfortaderkreislauf vorkommen. Auch einfache Erosionen der Speiseröhrenschleimhaut können schwere letale Blutungen veranlassen, die manchmal schleichend erfolgen. Bronner sah eine Fissur im Oesophagusmund, Glücksmann einen Herpes der Cardiagegend, Gottstein einen Ulcus pepticum. Oppikofer fand unter 128 Scharlachsektionen 15 mit Scharlachdiphtherie im Oesophagus, davon 3 ohne gleichzeitige Pharynxerkrankung. Die 15 Patienten waren zumeist Kinder aus dem ersten Lebensjahr, vorwiegend war der obere Speiseröhrenabschnitt befallen, doch blieb auch die Cardia nicht verschont. Bei einem $1\frac{1}{2}$ jährigen, nach abgelaufenem Scharlach an Pneumonie gestorbenen Kinde fand man neben nekrotischen Stellen auch feine Narben im Oesophagus. Bleibt ein solches Kind am Leben, so können sich Narbenstenosen ausbilden (Sargnon) und darüber kann es noch zu einer Dilatation kommen. Auch die reine Diphtherie befällt die Speiseröhre (Rolleston, Réthi, Sargnon). Die vernarbenden Geschwüre stenosieren den Weg.

Subakute und chronische Abscesse wurden mehrfach beobachtet. Roure sah einen mit Recurrenslähmung komplizierten Fall. Der Abscess wurde durch das Rohr geöffnet. Auch Albrecht erzielte durch Spaltung eines wahrscheinlich nach Erysipel entstandenen hochsitzenden Abscesses Heilung. Kubo hat in einem derartigen Falle 16 mal die Punktion ausgeführt und schliesslich Heilung erzielt. Der Abscess komprimierte die Trachea so stark, dass die Tracheotomie nötig war. Gottstein erlebte nach einem Punktionsversuch am 8. Tage Tod an Mediastinitis.

Dass auf dem Boden einer Verätzungsstriktur eine Tuberkulose sich entwickelt, dürfte zu den grössten Seltenheiten gehören (Leto).

Ueber die Syphilis der Speiseröhre wissen wir noch nicht viel. Sie kann Veränderungen machen, die für Carcinom gehalten werden. Schmiegelow erzielte bei einem derartigen Patienten durch Jodgebrauch Heilung. Walker Downie behauptet, öfter Syphilis im Oesophagus gesehen zu haben. In einem Falle von Schmiegelow hatte sich zwischen Speise- und

Luftröhre eine Fistel gebildet. Gottstein beobachtete ein Gumma im Antrum cardiacum.

An gutartigen Tumoren erwähne ich einfache Polypen, welche mit der Schlinge entfernt wurden (Morelli, Olavarria). Ein Papillom des Oesophaguseinganges gewann allmählich die Charaktere eines Carcinoms. Tumorähnlich wölbte sich in Grabowers Fall eine peritracheale Lymphdrüse vor. Sie machte in der Speiseröhre eine kirschgrosse Prominenz. Schliesslich sei noch ein Myxom im unteren Oesophagusteil (Abrand) genannt.

Dass die Carcinome in der Speiseröhre sehr häufig vorkommen, wissen wir schon lange. Guisez beobachtete unter 930 oesophagoskopischen Fällen 650 Carcinome, also $\frac{3}{4}$ der ganzen Zahl. Lemaître unterscheidet eine ulcerierte, eine wuchernde und eine infiltrierte Form. Andere sprechen nur von wandständigen und ringförmigen Carcinomen. Ein tiefsitzendes Carcinom kann durch Drüsenmetastasen eine hochsitzende Stenose machen (Glücksmann). Mehrfach fand man das Carcinom in die Trachea durchgewachsen, indem es dort einen Tumor bildete. Sehr leicht entwickelt sich unter solchen Umständen eine oesophago-tracheale Fistel (Tetens, Hald, Gerber). Ein hochsitzendes Carcinom kann Posticuslähmung veranlassen. Nach der Probeexcision erlebte Gottstein bei einem Cardiacarcinom Tod an Peritonitis.

In die Therapie des Speiseröhrenkrebses ist durch das Radium und Mesothorium neues Leben gekommen. Hill und Guisez haben damit Besserungen und anscheinend sogar Heilung erzielt. Wallace entfernte ein hochsitzendes Carcinom durch Operation. Danach trat Heilung ein.

Die verschiedenartigsten Vorgänge in der Speiseröhre verlegen den Weg oder machen dauernde Verengerungen. Die oesophagoskopisch vorgefundenen Stenosen bleiben manchmal hinsichtlich ihres Entstehungsgrundes dunkel. Manche Fälle können durch Scharlach und Diphtherie entstanden sein, insbesondere wenn sie kleine Kinder betreffen. Vielleicht gehört auch der Fall von Mosher hierher, bei dem am oberen Ende der Speiseröhre sich ein Diaphragma mit kleiner zentraler Oeffnung gebildet hatte. Einen ganz analogen Fall habe ich selbst beobachtet.

Eine angeborene Atresie der Speiseröhre ist oft beschrieben worden, nicht aber, dass man, wie Lubliner ein solches Kind am zweiten Lebenstage oesophagoskopiert hat. Wie es scheint, führen die Entwicklungsstörungen nicht immer zu einem vollständigen Verschluss. Es kann sich auch eine Stenose ergeben (Sargnon und Payson Clark). Am häufigsten sind immer noch die Verätzungsstrikturen, Guisez erwähnt allein 21 Fälle bei kleinen Kindern im Alter von 2–4 Jahren. Bei einem Kinde fand er 3 Stenosen nacheinander. Ausser Diphtherie, Scharlach, Pocken kommt auch die Syphilis als narbenbildende Ursache in Betracht. Auch nach Oesophagoskopien können sich Narbenstenosen ausbilden. Wenn bei einer Verätzung der Speiseröhre die Wirkung in die Tiefe geht, so kommt es in der Umgebung zu Bindegewebswucherungen, bei deren narbiger Umwandlung die Speiseröhre verzogen wird.

Ist die Gastrostomie notwendig gewesen, so können die Veränderungen in der Speiseröhre auch von unten her betrachtet werden. Für die Therapie ergeben sich daraus gewisse Vorteile. Auf die verschiedenartigen, sehr fein ausgearbeiteten Behandlungsformen der Narbenstenosen gehe ich nicht näher ein. Nur möchte ich mit Jackson vor der inneren Oesophagotomie warnen, welche doch gelegentlich auch den Tod veranlasst. Zur Dilatation mit elastischen Bougies gab Brünings eine Leitsonde an mit verstellbarer Scheibe, damit das Ende der Sonde vom Ende des Bougies entfernt bleibt. Sehr viel Erfolge sind mit Hilfe des verschluckten Fadens erzielt worden, der das Durchziehen von Oliven und Bougies nach der Gastrostomie von unten und von oben her erlaubt.

Eine besondere Form der Stenosierung der Speiseröhre wird durch die krampfartigen Kontraktionen des Oesophagusmundes und der Cardia bewirkt. Derartige Verengerungen treten nur periodisch auf und sind reflektorisch erregt durch die andrängenden Speisen. Doch beschreiben die französischen Autoren auch Fälle, in welchen zu dem Krampf eine Entzündung hinzukommt. Dadurch kann die Cardia dauernd verengt werden.

Es gibt auch Kompressionsstenosen der Speiseröhre durch retrotracheale Strumen, Mediastinaltumoren und Aneurysmen. Was die letzteren angeht, so ist ihre Einwirkung auf die Speiseröhre von Corbin und von Vygodsky oesophagoskopisch beobachtet worden. Erwähnt seien noch die perioesophagealen Lymphdrüsen, welche gelegentlich auch in die Speiseröhre durchbrechen können.

Es kann als Regel gelten, dass jede temporäre oder dauernde Verengerung der Speiseröhre, wenn sie lange genug dauert, eine Erweiterung des darüber gelegenen Abschnittes herbeiführt. Guisez fand in seinem dreifachen Strikturfall über jeder Striktur ein *gedehntes* Stück der Speiseröhre. Auch über carcinomatösen Stenosen kann sich, wie ich beobachtet habe, die Speiseröhre stark ausdehnen.

Der Oesophagospasmus führt bekanntlich zu einer partiellen Erweiterung des Hypopharynx, dem sogenannten Pulsionsdivertikel. Im Gefolge von Cardiospasmus dilatiert sich die ganze Speiseröhre im Laufe der Zeit. Neuerdings sind wieder enorme Dilatationen beobachtet worden (Kofler, Obakewitch, Heyrovsky). Der letztere gibt an, dass in seinem Falle durch Verschlucken von Luft die Luftröhre komprimiert wurde.

Erweiterungen der Speiseröhre durch einfache Atonie, wahrscheinlich im Gefolge von Neuritis des Vagus (Kraus) kommen vor. Marschik zeigte, dass dabei die Röntgenuntersuchung die Peristaltik der Speiseröhre vermissen lässt. Beim Cardiospasmus ist nach Brünings die Peristaltik im Oesophagus abnorm gesteigert. Bei dieser Krankheit hat die Behandlung mit Dilatatorien vielfach gute Resultate erzielt.

Bekanntlich kann auch Narbenzug am Oesophagus partielle Dilatationen hervorrufen, die sogenannten Traktionsdivertikel. Man hatte sie bisher nur an der Leiche beobachtet. Neuerdings aber wurden von

Frese zwei Fälle am Lebenden oesophagoskopisch festgestellt. In einem sass das Divertikel bei 20, im andern bei 25 cm an der vorderen Wand. Beides waren Frauen. Auch van Swieten beschreibt einen derartigen Fall. Man sah im Oesophagoskop auf der Seite der Speiseröhre bei 23 cm eine Oeffnung, aus der Speisereste herauskamen. Die Röntgenuntersuchung führte ebenfalls zum Nachweis des Divertikels. Es lag im Niveau des Sternums. Goris versuchte operativ einzugreifen. Der Patient starb jedoch am dritten Tage an Mediastinitis. Die Sektion ergab ein $\frac{1}{2}$ cm tiefes und 5 cm breites Divertikel, das an der hinteren Oesophaguswand mündete. Die Verwechslung mit einem Pulsionsdivertikel scheint mir nicht ausgeschlossen.

Fremdkörper in der Speiseröhre

Eine unübersehbare Menge von Fremdkörpern wurde wieder mittelst der Oesophagoskopie aus der Speiseröhre entfernt. Es ist nicht allein das wissenschaftliche Interesse, welches die Autoren dazu drängt, sondern auch die Freude an der gelungenen Extraktion und Ueberwindung von Schwierigkeiten. Sehr dankenswert finden wir die Mitteilung vieler Autoren über Zwischenfälle und üble Ausgänge. Es zeigt sich daraus deutlich, wie viel durch unzweckmässiges Vorgehen im Blinden geschädigt werden kann, und dass das Manipuliren durch das Oesophagoskop nicht Jedermanns Sache ist. Uebung und Selbstzucht (um vor allen Dingen jeden Schaden zu vermeiden) haben leider da und dort zu wünschen übrig gelassen. So haben sich denn eine Reihe von üblen Folgen ergeben, und manchem Kranken wurde der verschluckte Fremdkörper zum Verhängnis.

Ueber die Häufigkeit von Fremdkörperfällen im Verhältnis zu anderen macht uns Guisez eine interessante Angabe. Er hat 930 mal oesophagoskopiert und dabei 54 Fremdkörper bearbeitet. Gottstein allein gab einen Ueberblick über 109 Fälle, welche aus früheren Jahren stammen. Solche aus der Kasuistik vollständig auszuscheiden und dieselben nur auf die Jahre 1911–12 zu beschränken, war leider nicht möglich, weil eine Reihe von Mitteilungen nur summarische sind. Nach meiner Berechnung dürften 287 Fremdkörper in den Jahren 1911 und 1912 beobachtet worden sein. Bei einer grossen Zahl derselben ist leider nicht angegeben, worum es sich gehandelt hat. Sie sind nur einfach als Fremdkörper in der Speiseröhre bezeichnet. Ich kann daher nur in 170 Fällen die Art des Fremdkörpers in Rücksicht ziehen. Gebissplatten und Geldstücke herrschen vor. Von den ersteren habe ich 54, von den letzteren 44 notiert. Die Knochenstücke stehen erst an dritter Stelle mit 25 Nummern. Metallische Körper verschiedenster Art zählte ich 15. Darunter ist so ziemlich alles vertreten, was sich verschlucken lässt. Unter den übrigen sehr verschiedenartigen Dingen sei eine Sicherheitsnadel erwähnt. Von Fischgräten wird siebenmal berichtet. Ein 10 Monate altes Kind hatte einen Kragenknopf verschluckt. Dieser erwies sich jedoch nicht so schwierig, wie die Kragenknöpfe in den Luftwegen.

Wenn auch die Kinder eine grosse Anzahl von Fällen geliefert haben, so sind doch auch alle übrigen Lebensalter reichlich vertreten bis zum 92. Lebensjahre. Die jüngsten Kinder waren 4 und 13 Tage alt. Bei weitem die meisten Extraktionen sind glücklich gelungen. Jedoch ist auch eine ganze Anzahl von Fremdkörpern bei den Extraktionsversuchen in den Magen gerutscht. Einige wurden ausgewürgt.

Im Oesophagus werden die Fremdkörper im Allgemeinen bei weitem nicht so lange ertragen, wie in den Luftwegen. Es sind daher Fälle, in denen sie sich in der Speiseröhre längere Zeit aufgehalten haben, selten. Der erwähnte Kragenknopf hatte 10 Monate lang, ein Hornknopf 14 Monate lang (bei einem 4 jährigen Kinde) und eine Münze $3\frac{1}{2}$ Jahre lang in der Speiseröhre gesteckt.

Wenn ein Fremdkörper einige Zeit in der Speiseröhre verweilt, kann er in verschiedener Weise schädigend wirken. Vor allen Dingen kommt es zu Decubitus. Aus diesem kann sich eine Mediastinitis entwickeln, wie in dem Fall von Wojatschek. Brown Kelly erzählt von einem Geisteskranken, der eine ganze Reihe von Fremdkörpern verschluckt hatte: Eicheln, Erbsen, Knochenstücke etc. Trotz der oesophagoskopischen Entfernung ging der Patient an Lungengangrän zu Grunde, weil die Fremdkörper multiple Perforationen herbeigeführt hatten. In den Fällen von Guthrie und Schousboe hat die Gebissplatte nicht allein die Oesophaguswand perforiert, sondern auch eine tracheale Fistel gemacht. Fortschreitende Eiterung führte bei dem Patienten von Milligan zu einer Arrosion der Aorta und zu Verblutung. Unter 200 aus der Literatur zusammengestellten Fremdkörpern fand Lerche 2 mal Perforation der Speiseröhre und der Aorta innerhalb 24 Stunden, 8 mal denselben Vorgang nach 5–10 Tagen. Es handelte sich dabei um spitze Fremdkörper.

Als seltenes Ereigniss muss es bezeichnet werden, wenn ein in der Speiseröhre verweilender Fremdkörper eine Recurrenslähmung macht. Sargnon berichtet von einem solchen Falle. Es handelte sich um eine Gebissplatte.

Bei der oesophagoskopischen Untersuchung und Extraktionsversuchen entstehen leicht oberflächliche Abschürfungen. Sie verdienen keiner besonderen Beachtung und heilen von selbst. Handelt es sich aber um einen Hämophilen, so sollten sie ängstlich vermieden werden. Botey erlebte unter solchen Umständen starke Blutungen.

Mehrfach kamen gröbere Verletzungen zur Beobachtung (Claoué, Kümmel, Guthrie, Yankauer etc.), welche bei Extraktionsversuchen entstanden waren. Für die meisten dürfte der Münzenfänger verantwortlich gemacht werden. Bei einem solchen Manöver war der Fremdkörper, eine Münze, durch die Perforation unter die Schleimhaut geschoben worden. Die Muskelwand blieb offenbar unverletzt. Deswegen kam der Patient mit dem Leben davon. Die Extraktion war erst nach Spaltung der Schleimhaut über der Münze möglich.

In dem Yankauer'schen Fall hatte der frühere Operateur die Speiseröhre durchbohrt und einen falschen Weg gemacht, zwischen dieser und

der Wirbelsäule. Das Oesophagoskop schlug regelmässig die falsche Bahn ein. Erst nach vielen Schwierigkeiten gelang es, in die Speiseröhre selber einzudringen und den Fremdkörper (ein Metallstück) von dort zu entfernen. Wie zu erwarten war, wurde der Patient dadurch nicht gerettet. Auch ein 2 jähriges Kind von Stanton starb an Verletzungen der Speiseröhre durch vorausgegangene Manipulationen. Dabei handelte es sich noch um die leichteste Art von Fremdkörpern, um eine Münze. Aehnlich liegen zwei Fälle von Kümmel und Claoué. Die Fälle von Wojatschek und Milligan habe ich bereits erwähnt. Es kommen noch zwei weitere dazu, in welchen die Extraktion nicht gelungen war und Mediastinitis entstand. Bei einem derselben hat auch die Oesophagotomie nicht geholfen.

Den wenigen misslungenen und übelverlaufenen oesophagoskopischen Extraktionsbemühungen steht die grosse Zahl derjenigen gegenüber, welche vollen Erfolg geben, selbst nach Ueberwindung grosser Schwierigkeiten. Als besondere Leistung hebe ich die intraoesophageale Zerlegung einer Gebissplatte mit einem besonders konstruierten galvanokaustischen Brenner hervor, welche Tapia gelungen ist. Das Gebiss sass 32 cm tief. Der Kauter war wie ein grosses lateinisches A geformt ; nur das Querstück konnte zum Glühen gebracht werden. Hubbard hat eine Sicherheitsnadel im Oesophagus gedreht und dann extrahiert. Beiläufig sei erwähnt, dass Simpson auf dem Röntgenschirm eine Sicherheitsnadel in der Speiseröhre beobachtete, die am ersten Tage nach oben und am zweiten nach unten offen stand, ohne dass etwas Besonderes geschehen war. Die Drehung musste beim Schlucken von Nahrungsmitteln stattgefunden haben. Ein durch eine Gräte hervorgerufener intraoesophagealer Abscess wurde von Jacques durch das Rohr geöffnet. Der Erfolg war ein guter.

Die Oesophagotomie wurde gemacht, wenn die Extraktion sich als undurchführbar erwies. Dies hing aber offensichtlich nicht allein von der Natur des Fremdkörpers und der Eigenart des Falles ab, sondern auch von der Technik des Operateurs. Auch kam mehrfach in Betracht, dass die Speiseröhre durch die Beschaffenheit des Fremdkörpers oder durch vorausgegangene unzweckmässige Extraktionsmanöver Schaden gelitten hatte. So erklären sich etwa 15 operative Eingriffe von aussen (bei Zahnplatten, Metallkörpern, Knochen etc.).

Corbin erzählt von einer Gebissplatte, welche eine 21 jährige Frau verschluckt hatte und die oesophagoskopisch nicht entfernt werden konnte. Vier Tage nachher wurde von aussen operiert und dabei ein zwischen Luft- und Speiseröhre gelegener Abscess eröffnet. Karevsky hat einen peri-oesophagealen Abscess mit der Oesophagotomie geheilt und aus der Abscesshöhle den Fremdkörper, ein Drahtstück, entfernt.

Eine besondere Stellung nehmen die Fremdkörper über Stenosen ein. Stenosen können Dinge zurückhalten, welche sonst mit Leichtigkeit geschluckt werden. Schon durch eine weiche Masse kommt es an engen Stellen zum vollständigen Verschluss. Der Patient befindet sich dann in der üblen Lage, überhaupt nichts mehr schlucken zu können. In dem

Falle von Schmilinsky hat eine Pflaumenschale genügt, um die Speiseröhre an ihrer engen Stelle zu verstopfen. Auch Divertikel können Fremdkörper zurückhalten. In dem Falle von Willaume-Jantzen steckte ein Fleischbrocken in dem Pulsionsdivertikel. Der 78 jährige Patient konnte infolgedessen 2 Tage lang nicht schlucken.

Gastroskopie

In den letzten beiden Jahren hat die Untersuchung des Magens mittelst Röhren einige Fortschritte gemacht, doch ist die Zahl der Mitteilungen noch eine recht beschränkte. Chevalier Jackson empfiehlt nach wie vor seine Methode mit dem offenen Rohr ohne Optik. Er hat bis jetzt 160 Fälle in dieser Weise gastroskopiert und damit gute Erfolge gehabt. Ein neues Gastroskop wurde von Sussmann in Berlin empfohlen. Dasselbe hat einen starren mit Oculartrichter versehenen und einen beweglichen mit Gummi überzogenen Teil, welcher Objectiv und Lampe trägt. Das Instrument wird in beweglichem Zustand bis in den Magen geschoben und dann durch Drehung eines Rades gerade gestreckt.

Sussmann verwahrt sich dagegen, dass dieser Modus gefährlich sei. Ueber 100 Fälle wurden von ihm untersucht, ohne dass sich dabei eine Störung ergab. Ich habe jedoch zufällig Kenntniss von einem Fall erlangt, in welchem der durch pathologische Prozesse fixierte Magen bei der Gradstreckung des Sussmann'schen Gastroskops an der kleinen Curvatur einriss. Auch durch einen raschen operativen Eingriff und Naht der Wunde konnte Patient nicht gerettet werden.

Besonders eifrig haben sich H. Elsner in Berlin und William Hill in London mit der Gastroskopie beschäftigt. Beide geben monographische Darstellungen. Jeder von beiden arbeitete mit einem selbstkonstruierten Instrumentar. Das Elsner'sche Gastroskop habe ich anwenden sehen und halte es für sehr brauchbar. Es handelt sich um ein gerades starres Rohr, dessen Spitze biegsam konstruiert ist. Sie besteht aus einer Drahtspirale mit Gummiüberzug und soll das Rohr leichter durch den Rachen in die Speiseröhre und durch den Hiatus oesophageus nach vorn und besonders nach links in den subphrenischen Teil der Speiseröhre und durch die Cardia in den Magen leiten. Es handelt sich um ein Doppelrohr. Das innere ist exzentrisch mit dem äusseren verbunden. Durch den restierenden, mondsichelförmigen engen Raum sind die elektrischen Leitungen und der Luftkanal zum Aufblasen des Magens geführt. Dabei beträgt der Gesamtdurchmesser 11 und die Länge 76 mm. Die Beleuchtung bewirkt ein über dem Gummiansatz angebrachtes elektrisches Lämpchen. Die Optik ist in einem besonderen Rohr untergebracht, welches in das Doppelrohr vor Beginn der Untersuchung eingeführt wird. Die optische Einrichtung ist so getroffen, dass man seitlich aus dem Gastroskop heraussieht. Vor dem rechtwinklig spiegelnden Prisma befindet sich eine kleine Linse. Durch Drehung des optischen Apparates wird diese gedeckt und bei der Einführung vor Beschmutzung bewahrt.

Das optische System ist nach den neuesten Grundsätzen der geometrischen Optik gebaut unter Verwendung einer grösseren Zahl von Linsen. Dadurch werden besonders lichtstarke und scharfe Bilder und ein weites Gesichtsfeld erzielt. Auch erhält man ein aufrechtes Bild.

Elsner giebt genaue Vorschriften über die Vorbereitungen zur Untersuchung und diese selber. Der Patient wird nicht narkotisiert, sondern bekommt nur eine Morphiuminjektion von 2 cg· und wird mit Cocain gepinselt. Das Rohr wird in linker Seitenlage eingeführt und möglichst weit bis an die grosse Kurvatur vorgeschoben. Wenn der Magen aufgebläht ist, dreht man die Optik, damit das Ocular frei wird und betrachtet dann das Innere des Magens.

Elsner wählt seine Fälle sorgfältig aus und sucht sich vor Unglücksfällen dadurch zu bewahren, dass er sich streng an bestimmte Indikationen hält.

William Hill's Gastroskop ist eine Modifikation des von Herschel in Breslau gebauten Instrumentes. Es besteht aus einem geraden Rohr mit einem besonderen Luftkanal für die Aufblähung des Magens. Beleuchtung und Optik befinden sich in einem besonderen Rohr. Ein weiches Mundstück ist nicht an dem Instrument angebracht. Das Brünings'sche Rohr wird mit Brünings'schem Handgriff beleuchtet eingeführt. Erst danach schiebt man die Optik mit endständiger Lampe durch. Es kann also zuerst nach Jackson und dann mit Optik untersucht werden. Die Untersuchung ohne Optik wird dadurch erleichtert, dass das Rohr oben einen Glasabschluss erhält, um die Aufblähung des Magens zu gestatten. Hill scheint mit seinem Instrument gute Erfahrungen gemacht zu haben. Das Aussenrohr ist nur 55 und das optische 65 cm lang. Erwähnt sei noch ein Speculum ' à grand champ visuel ' von Robert Rendu. Dasselbe hat eine eigentümliche Konstruktion. Von einem Trichter gehen 6 elastische Stäbe aus, die sich beim Zurückziehen einer Scheibe spreizen und dadurch dem Beschauer ein grosses Gesichtsfeld freihalten.

Die Gastroskopie hat entschieden noch gegen grosse Vorurteile zu kämpfen. Ihrer Anwendung durch die Laryngologen stellen sich praktische Schwierigkeiten entgegen, weil diesen nicht genügendes Material zuströmt. Diejenigen, welche sich mit den Erkrankungen des Magens speziell befassen, führen zumeist die Gastroskopie selber aus. Es ist fraglich, ob sich darin in Zukunft vieles ändern wird.

SECTION XV

RHINOLOGY AND LARYNGOLOGY

DISCUSSION No. 1

ON THE RECENT PROGRESS OF ENDOSCOPIC METHODS AS APPLIED TO THE LARYNX, TRACHEA, BRONCHI, ŒSOPHAGUS AND STOMACH

(For Report, see Part I, pp. 1–34)

Additional Introductory Remarks

By CHEVALIER JACKSON, M.D., Pittsburg, U.S.A.

Killian, like Lister, is a pathfinder. It remains for us to follow. My report in the presessional proceedings will, I hope, not be misunderstood to be a *complete* report of the recent progress of peroral endoscopy. Many replies to my circular letter were necessarily omitted because they came too late, especially some very valuable data from D. Braden Kyle, Bryan, Stucky, Jervey, Birkett, Freudenthal, Harris, and many others; and of course much good work remains unmentioned because of lack of space.

I beg to offer the following points not as dogmatic assertions but as postulates for discussion.

1. Direct laryngoscopy for diagnosis should be done by every laryngologist but, for the best results, bronchoscopy, œsophagoscopy, and gastroscopy should be done by a few men to whom all cases should be referred in order that the highest perfection in armamentarium, organization, and personal skill may be attained.

2. Eliminating the deaths due to lack of promptness in performing bronchoscopy the mortality of bronchoscopy in the hands of various operators is about 3·5 per cent.

3. Failures to remove foreign bodies known to be in the bronchi are almost all failures to find a small foreign body located in a minute bronchus far down and far out toward the periphery, and very rarely failures to remove a foreign body when found. Therefore the development of localization aids is of paramount importance.

4. Œsophagoscopy for diagnosis, in skilled hands, has practically no mortality. For the removal of foreign bodies it has a mortality of less

than 3 per cent., and this mortality is due to trauma incident to the foreign bodies and their extraction.

5. The mortality of gastroscopy, in skilled hands, is less than 1 per cent. There have been over 1,000 gastroscopies done by various operators, so that gastroscopy is established as a safe procedure in skilled hands. Practical instruments are now developed; it remains to develop the gastroscopists who will use them.

6. The best instruments for peroral endoscopy are those with which the operator has had the most practice. Successful work depends more upon the skill and training of the endoscopist than upon his instrumentarium, provided the latter is ample. Professor Killian, I believe, prefers the Kirstein head-lamp for much of his work.

7. For peroral endoscopy, except in the most urgent cases, the patient should be prepared generally as for any other operation. Locally, oral antisepsis is highly important. Before undertaking a prolonged course of peroral endoscopic treatment for chronic conditions, diseased lymphoid tissue anywhere in Waldeyer's ring should be removed. Diseased faucial tonsils are especially potent sources of dangerous complications.

8. The conclusions as to anæsthesia in my report are too long satisfactorily to epitomize further than to state that the greatest of recent progress is towards the use of no anæsthetic at all. General anæsthesia introduces into peroral endoscopy a peculiarly grave risk out of all proportion to the anæsthetic risks in the general run of surgical cases. In œsophagoscopy, intratracheal ether-insufflation anæsthesia, as devised by Melzer and Auer and perfected by Elsberg and Janeway, is safe. It cannot be used in bronchoscopy.

9. The essentials of the best position of the patient were first demonstrated by Kirstein in his early work, and they consist chiefly in an extreme anterior placement of the cervical spine with extension only at the occipito-atlantal joint. The flexed position of Mosher and Johnston is often useful for direct laryngoscopy. Removal of laryngeally lodged foreign bodies should be attempted only in the Trendelenburg position in order to have gravitation in our favour instead of against us.

10. The causes of subglottic œdema are chiefly the use of too large a tube and the trauma in introduction of the tube or of the sojourn or the removal of the foreign body. Many cases reported as subglottic œdema are essentially an incipient drowning of the patient in his own secretions. A few years ago Dr. Logan Turner made special studies on the general subject of œdema of the larynx, and I hope he will enlighten us.

11. Tracheotomic bronchoscopy is very rarely, if ever, necessary unless the endoscopist is unaccustomed to very small tubes.

(An error exists in the report in stating that the author has had but one case of tracheotomic bronchoscopy for foreign body. There were two, both in the author's earliest work.)

Tracheotomy may be required for dyspnœa, but in such cases it is better to do the bronchoscopy through the mouth.

12. Bronchoscopic oxygen insufflation, preferably with amyl nitrite added, is the best method of resuscitation in cases of respiratory arrest.

13. In all endolaryngeal operative work it is essential to avoid injury to the arytenoids, as upon their subsequent activity largely depends the formation of a useful adventitious vocal cord.

14. Endoscopic cauterization is the best method of curing hypertrophic subglottic post-diphtheritic stenoses. Endoscopic divulsion and excision are often curative in cicatricial laryngeal stenosis, and are worthy of a trial before resorting to laryngostomy.

15. The word ' cardiospasm ' should either be dropped altogether as misleading or else should be restricted to those rare cases of spasm limited to the cardiac orifice. I would suggest the following nomenclature :

(*a*) Hiatal œsophagismus.
(*b*) Cardial œsophagismus.
(*c*) Abdominal œsophagismus.

16. The operation of external œsophagotomy for foreign bodies is rarely, if ever, justifiable. Any foreign body, no matter how large or sharp, that has entered the œsophagus through the mouth can be brought out the same way. When swallowed, the intruder turns to the position in which it offers the least resistance. It remains for the œsophagoscopist to turn it so as to offer the least resistance on the return journey. A notable case in point is that of D. Braden Kyle, who skilfully removed a large tooth-plate from a bed of cicatricial tissue by careful manipulation under cocaine.

17. Pleuroscopy for the removal of foreign bodies, and especially for the diagnosis and treatment of disease, is full of promise.

18. Finally, the future progress of endoscopy depends upon those who devote to it the time necessary to educate the *eye* and the *fingers*. It requires but little brains, now, since Killian has pointed out the way.

SECTION XV

RHINOLOGY AND LARYNGOLOGY

DISCUSSION No. 1

ON THE RECENT PROGRESS OF ENDOSCOPIC METHODS AS APPLIED TO THE LARYNX, TRACHEA, BRONCHI, ŒSOPHAGUS AND STOMACH

DISCUSSION

Professor Dr. Ottokar Chiari (Wien). Die Wichtigkeit der direkten Untersuchungsmethoden der oberen Luft- und Speisewege wird wohl dadurch am besten bewiesen, dass wir sie täglich brauchen. An meiner Klinik werden sie in der grössten Zahl sowohl zu diagnostischen als auch zu therapeutischen Zwecken vorgenommen.

Fremdkörper des Oesophagus. Ich will nur darauf verweisen, dass an meiner Klinik von 1909 bis jetzt 182 Fremdkörper durch Oesophagoskopie entfernt wurden. Darunter waren 69 Knochen, 42 Fleischstücke, 14 künstliche Gebisse, 11 Münzen, 6 Fischgräten, 9 Fruchtkerne, 3 Nadeln, 3 Knöpfe und verschiedene andere Dinge. Einmal extrahierte ich zwei platt aneinander liegende Münzen auf einen Griff. In schon früh bestandenen Strikturen sassen 13 dieser Fremdkörper. Die Extraktion gelang in allen Fällen, manchmal leichter, manchmal schwerer; zwei der Patienten starben an Mediastinitis in Folge von Verletzung durch den Fremdkörper; drei konnten durch Mediastinotomie von diesem Schicksale gerettet werden, obwohl man bei dieser Operation schon deutliche Perioesophagitis nachweisen konnte.

Drei Fremdkörper der Trachea. Zwei Bohnen sassen an der Carina und eine Bohne wurde in gequollenem Zustande nach Tracheotomie bei einem kleinen Kinde entfernt. Die meisten Fremdkörper wurden tiefer aspiriert.

23 *Fremdkörper der Bronchien.* 18 im rechten, 5 im linken Bronchialbaum, meistens in den Hauptbronchien. Es handelte sich um 8 Knochen- oder Knorpelstücke, 4 Erbsen oder Bohnen, 4 Fruchtkerne oder Fruchtteile, 2 künstliche Gebisse, 2 Nägel, 2 Glasperlen und 1 Münze. Einmal wurde die Extraktion verweigert. Alle anderen wurden extrahiert; bei kleinen Kindern musste in einigen Fällen die Tracheotomie vorausgeschickt werden.

Neubildungen der Trachea oder Bronchien. Ambulatorisch wurden zwei Fälle von Karzinom untersucht. Acht klinische Fälle. Ein Endotheliom im oberen Teile der Trachea, ein Zylindrom ebendort und ein Rundzellensarkom unter dem 8. Ringe wurden mit direkter Tracheoskopie mit Pincetten, Locheisen und Schlinge abgetragen und einmal galvanokaustisch verätzt und schliesslich geheilt. Ein Sarkom von Daumengliedgrösse im rechten Bronchus wurde nach Bronchoskopie spontan ausgehustet. Ein harter, die halbe Peripherie der Trachea in der Höhe des 2. Ringes umfassender, wulstförmiger Tumor wurde mit Morcellement und Galvanokaustik entfernt. Unsicherer histologischer Befund. Zwei Karzinomen des unteren Teiles der Trachea und ein Adenokarzinom des rechten Bronchus wurden mit Schlinge, Pincette, Locheisen und auch mit Einlegen eines Dominiciröhrchens von 17,6 mg metallischem Radium (einmal nach Tracheotomie) mit wechselndem Erfolg behandelt.

Tumoren ausser der Trachea. Sechsmal wurde die Trachea, resp. einer oder beide Bronchien, durch Mediastinaltumoren verengert. Einmal Kompression der Trachea durch Struma maligna von vorn nach hinten. Einmal brach die Struma maligna durch die vordere Wand in die Trachea durch, während die Hinterwand nur vorgewölbt war. Einmal wurde nach Anlegung einer Lungenfistel bei der Obduktion ein apfelgrosses Medullarkarzinom zwischen Oesophagus, Trachea und Aorta gefunden. Einmal liess sich der Mediastinaltumor nicht nach seiner Natur bestimmen.

Fünf Karzinome des Oesophagus mit Perforation in die Trachea und einmal auch in den linken Bronchus wurden histologisch durch Bronchoskopie genau bestimmt. Zweimal erfolgte Exitus; zehnmal war die Trachea oder auch ein Bronchus nur komprimiert.

Eine besondere Besprechung verdient eine ausgedehnte Metastasenbildung von einem Karzinom der Schilddrüse ausgehend:

Klinik. (Carc. thyreoid. et bronchi dextri.) Marie Höring, 56 J., Hausbesorgerin, XII, Steinbauergasse 32. Aufnahme 14. V. 13. Januar 1913, strumektomiert (Klinik Eiselsberg). Histologischer Befund der ektom. Struma: Adenocarcinoma thyr. Februar, Tracheotomia sup.; wegen zunehmender Atembeschwerde zu uns transferiert. *Larynx*: Stimmlippen stehen unbeweglich in der Medianlinie, nur die rechte macht ruckende Bewegungen. *Oesophagus*: Bougie 20 passiert anstandslos, nur das Rohr ruft Spasmus am Oesophagusmund hervor und geht nicht weiter; Patient kann gut essen, also jedenfalls keine nennenswerte Stenose. *Bronchi*: Bifurkation u. *linker* Bronchus normal, 1 cm. von der Bifurkation im *rechten* Hauptbronchus ein wie eine gequollene Fisole aussehender Tumor mit der Basis an der lateralen Wand. Entfernung mit der scharfen Pincette, worauf sich weiter unten eine zweite kugelige, weisse Verwölbung zeigte, welche mit scharfer Pincette leicht als Tumor *in toto* von zwei Fingerbeerengrösse entfernt werden konnte. Exitus nach mehreren Wochen. Obduktionsbefund: Durchbruch eines Carc. thyr. subglott. in den Larynx, metast. Infiltration der peritrach. Lymphdrüsen, sowie der Bifurkationsdrüsen, und Durchbruch in den *rechten* Stammbronchus, 6 cm. unter der Carina; ausgedehnte Metastasen in der *rechten* Lunge und Pleura.

Während der Bronchoskopie trat nur einmal der Tod ein ; die Sektion ergab eine Pankreasapoplexie, so dass also die Bronchoskopie nicht schuld war.

Die Schwebelaryngoskopie wurde etwa 20 mal vorgenommen mit dem Instrumentarium von Killian oder Albrecht, bei Kindern nur in der Narkose. Sie ergab sehr gute Resultate bei 2 Fällen von Papilloma laryngis, 12 Fällen von Tuberkulose, 3 Fällen von Polypen, und in einigen Fällen, um die Ventrikel oder den subglottischen Raum zu untersuchen. Ich ziehe einen breiten Zungenspatel vor, weil neben dem schmalen sich die Zunge an beiden Seiten zu sehr vorwölbt. Mein Assistant, Dr. Kofler, wird die hier nur kurz angedeuteten Fälle ausführlich publizieren.

Dr. RICHARD LEWISOHN (New York). Though a great number of different œsophagoscopes have been constructed in the last thirty years, no instrument has so far proved to be entirely satisfactory. The straight tube œsophagoscope is not very popular on account of the great discomfort to the patient. I have devised a new model, a rectangular telescopic œsophagoscope, which adapts itself better than any other instrument heretofore constructed to the normal relations between mouth and œsophagus. The advantages of this instrument are : 1, The introduction of the œsophagoscope is possible in the normal position of the head, and, 2, the instrument is so constructed that it actually passes in the longitudinal axis of the œsophagus, and not at an angle to this axis. The telescopic part consists of six tubes which can be easily evolved with the aid of a spring. The uppermost tube carries two metal guides which act as a kind of obturator, and simplify the introduction of the instrument very considerably. A series of cases examined with this new model, especially of cancer of the œsophagus, was reported.

Dr. WOLFF FREUDENTHAL (New York). For the past year or more I have tried suspension laryngoscopy and found it a valuable aid in the treatment of many cases.

The most important cases are those of laryngeal tuberculosis. It is true that with the aid of this method we can curette ulcerations all over the larynx in one sitting and can remove masses of infiltrations such as we never dreamt of.

Another class of case in which the method is valuable is that of papillomata of the larynx in children. In these cases it is less dangerous to operate without an anæsthetic, as pointed out by Jackson, and Johnston of Baltimore. For the incision of deep-seated *abscesses* of the pharynx and larynx it is the easiest and best method possible. Any one who has lost a patient at the moment of opening a laryngeal abscess will appreciate that fact. The position of the epiglottis often prevents us from obtaining a good view of the larynx. The speaker has constructed a new spatula to overcome that obstacle, the details of which will be published later.

Professor Dr. INO KUBO (Fukuoka, Japan). Ich fühle mich verpflichtet, Ihnen über den gegenwärtigen Zustand der direkten Untersuchungsmethoden in Japan zu berichten, da sie erst nach meiner Heimkehr (1907) hauptsächlich von mir und meinen Schülern eifrig studiert und gefördert wurden. An dieser Stelle spreche ich dem Vater der Bronchoskopie, meinem früheren Chef Herrn Geheimrat Killian herzlichen Dank aus. Vor meiner Heimkehr wurde das Instrumentarium allerdings von Fachkollegen, z. B. Tanaka, Nishiyama u.s.w. eingeführt und zeitweise benutzt, aber die allgemeine praktische Anwendung fand erst seit 1907 statt.

Die Oesophagoskopie wird meist bei Fremdkörperfällen angewendet. Die Zahl der seit 1907 veröffentlichten Fremdkörperfälle mit ösophagoskopischer Behandlung ist 150; darunter 127 in meiner Klinik und 23 von anderen Aerzten behandelt. Auf Grund meiner Erfahrungen werde ich einige Punkte hervorheben. Die Fälle betreffen meist Kinder, bisweilen aber auch alte Leute, da sie oft schlechte Zahnprothesen tragen und unvorsichtig essen. Das männliche Geschlecht ist vorwiegend betroffen (männlich 84 : weiblich 43). Die Arten der Fremdkörper sind bei uns etwas anders als in Europa, da die Gewohnheiten und Nahrungsmaterialen verschieden sind. Wie die Abbildungen von meinen Fällen und einige Exemplare meiner Fremdkörper Ihnen erklären, handelt es sich meistens um Münzen, Fischgräten, Zahnprothesen, Spielzeuge u.s.w.

Die Oesophagusfremdkörper wurden bis jetzt ausschliesslich durch Oesophagotomie, Angeln oder Hinunterstossen behandelt. Der Münzenfänger ist noch heute ein wichtiges Instrument bei Oesophagusfremdkörpern, trotzdem er oft schauderhafte Folgen mit sich brachte. Die Vorwürfe gegen die Oesophagoskopie sind hauptsächlich, dass die Technik schwierig und das Instrumentarium teuer ist. Aber unser Bestreben hat die Fachwelt so weit angespornt, dass man heute die Wichtigkeit der Oesophagoskopie versteht und das Instrumentarium in jedem grossen Hospital vorhanden ist. Die Vermehrung der ösophagoskopischen Fremdkörperfälle auch ausserhalb meiner Klinik[1] beweist diese Neigung:

1906, 1; 1907, 0; 1908, 2; 1909, 0; 1910, 2; 1911, 2; 1912, 6; 1913, 10.

Beiläufig sei erwähnt, dass japanische Laien aus Aberglauben Amuletspapier der Tempel schlucken, wenn sie einen Fremdkörper im Oesophagus haben, um denselben hinunterzufördern, was aber natürlich nur dazu dient, das Lumen des Oesophagus noch weiter zu verengern.

Die Oesophagoskopie wird bei uns fast ausschliesslich von Spezialisten

Oesophagoskopisch untersucht:	*Positiv und ösophagoskopisch extrahiert*:
1907 . . . 9	7
1908 . . . 12	9
1909 . . . 15	13
1910 . . . 19	15
1911 . . . 22	9
1912 . . . 34	21
1913 (bis Juni) 16	10
127	84

geübt. Unter denselben sind zu nennen: Tanaka, Tatsumi, Saito, Azai, Matsuzaki, Yoshii, Yokote u.s.w., und meine Schüler Kaji, Katsuyza, Matsui, Nogawa, Kosokabe, Sakurama, Mashida, Inouye, Kaku u.s.w.

Bei der Extraktion einer Zahnprothese muss man eine Ecke derselben suchen, von der Schleimhautwulst losmachen und sie an dieser Ecke fassen. Dazu benutzt man am besten ein schräg geschnittenes Rohr. Selbst ein rundlicher Fremdkörper wie eine Münze macht bei langem Verweilen einen Recessus an der Oesophaguswand, was die Sondierung erschwert. Bei Fällen von Fremdkörpern fand ich keine Kontraindikation gegen Oesophagoskopie, sogar bei Abszessbildung wegen Fremdkörpers konnte ich immer mit gutem Resultate ösophagoskopieren.

Bei Tumoren, besonders bei Karzinomen, ösophagoskopiere ich nicht nur für diagnostische Zwecke, sondern auch für therapeutische, z. B. bei Exzision kleiner Geschwülste, oder *bei Einführung* einer Dauerkanüle, welche dem Kranken Gastrostomie erspart. Beim Einführen des Radiumstiftes in den Oesophagus benutze ich ebenfalls die Oesophagoskopie, und auch bei der Untersuchung und Behandlung der Oesophagusstenose infolge chemischer Aetzungen, blinde Sondierung ist hierbei sehr gefährlich. Ich habe seit zwei Jahren bei 4 solchen Patienten ösophagoskopische Dilatation nach Gastrostomie probiert, 2 von ihnen genasen, während 2 sich noch in Behandlung befinden. Meine erfolgreiche Punktion eines periösophagealen Senkungsabszesses infolge von Brustwirbelkaries ist bereits veröffentlicht worden (*A. f. L.*, Bd. xxv, H. 3).

Da ich denke, dass die Thoraxchirurgie mit direkten Untersuchungsmethoden Hand in Hand gehen muss, so untersuche ich die Oesophaguskarzinomkranken systematisch mittels der Oesophagoskopie und bestimme exakt die Beschaffenheit und Lokalisation der Tumormasse, bevor ich sie nach der chirurgischen Klinik schicke. Jedes Leiden des Oesophagus, besonders bei alten Leuten, muss ösophagoskopisch untersucht werden, damit eine frühzeitige Diagnose ermöglicht wird.

Die Benutzung der direkten Laryngo-tracheo-bronchoskopie ist im allgemeinen viel seltener in Japan als die Oesophagoskopie, aber ich selbst jedoch habe ziemlich reiche Erfahrungen. Der erste mittels der oberen Bronchoskopie mit Erfolg behandelte Fall datiert vom 15ten September 1907, an welchem Tage ich eine Knopfnadel bei einem 5 jährigen Knaben aus dem linken Bronchus glatt entfernte, der vor 6 Monaten aspiriert worden war und dem Kranken eine schwere Pneumonie verursacht hatte. Mir sind 28 Fälle von Fremdkörpern in den Luftwegen aus der Literatur und Privatmitteilung bekannt, welche in Japan durch die direkte Methode behandelt wurden. Von diesen wurden 20 Fälle in meiner Klinik behandelt. Bronchialfremdkörperfälle betragen im ganzen 17, darunter 14 in meiner Klinik. Die sämtlichen Fälle zerfallen folgendermassen: Nishiyama, 2 Fälle (Bronchosc. inf., gerettet); Mishima, 1 Fall (Broch. inf., gestorben); und in meiner Klinik: Kubo, 10 Fälle (in 7 Fällen wurden die Fremdkörper durch Bronchoscopia superior glatt extrahiert, Heilung; in 2 Fällen nach und vor der Oberbronchoskopie ausgehustet und in einem

Falle glatte Extraktion mit Heilung durch Bronchosc. inf.) und je 1 Fall (Bronch. sup.) von meinen Assistenten, Nogawa (gerettet), Matsui (gestorben), und Sakurama (gestorben). Die Bronchialfremdkörper waren Bohnen (4 mal), Obstkerne (4 mal), Pfeifen (3 mal), Pflanzensamen (3 mal), Nagel (1 mal), Trachealkanüle (1 mal) und Hummerschale (1 mal). Ich ziehe stets die obere Bronchoskopie vor, und habe nur einmal bei einem Trachealkanülenfalle durch Bronchosc. inf. die Extraktion vorgenommen, da der Patient schon eine Kanüle trug. Man muss bei der oberen Bronchoskopie der Kinder die Kopflage des Kranken und die Dauer der Untersuchung berücksichtigen. Das Rohr soll natürlich nicht zu dick sein. Postoperative Dyspnoe habe ich noch nicht beobachtet.

Ueber die Diagnose und Behandlung der Trachealtumoren (1 primäres Karzinom, 1 Fibrom) und der Stenosen habe ich schon beim letzten internationalen Kongresse (1909 in Budapest) berichtet.

Bei der Indikationsstellung für das Decanulement ist die direkte Untersuchung mir unentbehrlich. Bei asthmaähnlicher Atembeschwerde der Kinder findet man oft durch Tracheobronchoskopie deutliche Vorwölbung einer Bronchialdrüse und Verengerung der Bronchialeingänge (Kubo und Toyama). Bei Asthma bronchiale nehme ich als Regel Bronchoskopie vor, und entdeckte gelegentlich maskierte Aneurysmen. Intrabronchiale Bepinselung von Kokain-Adrenalinlösung bei Asthmaanfall geschieht sehr gut durch das Rohr.

Die Kehlkopfbefunde der kakke- oder beriberikranken Säuglinge[1] wurden von mir im Jahre 1911 durch direkte Untersuchung entdeckt. Ich fand solche seither in 5 Fällen mit Recurrenslähmung (meist links), Oedem der Arygegend oder Anästhesie der Kehlkopfschleimhaut. Es ist sehr merkwürdig, dass das Kakkegift bei Säuglingen den Kehlkopf mehr als bei Erwachsenen angreift. Als neu möchte ich bezeichnen, dass ich (1913) bei einem 15 jährigen Mädchen eine linksseitige Recurrenslähmung ohne bekannte objektive Ursache durch sorgfältige Untersuchung auf eine Säuglingskakke zurückführen konnte. Dieses Mädchen wurde als Kind von ihrer kakkekranken Mutter gestillt und war schon damals heiser. Hiermit glaube ich, dass ein neues Moment zur Aetiologie der Recurrenslähmung hinzugefügt ist.

Was das Instrumentarium angeht, so ist es teilweise Geschmackssache. Man benutzt in Japan bald Killians, bald Brünings' Elektroskop. Kleinere Modifikationen sind selbstverständlich auch bei uns vorhanden. Das Motto für die direkten Untersuchungsmethoden ist, wie Killian einst mir schrieb: Erstens Geduld, dann Hand und Auge!

Dr. BRADEN KYLE (Philadelphia). Of the many interesting points

[1] Unter Säuglingskakke oder Beriberi versteht man eine Krankheit mit Heiserkeit, Erbrechen, Unruhe, Appetitlosigkeit, Oedem oder Abmagerung u.s.w., welche durch Stillen mit der Milch einer kakkekranken Frau hervorgerufen wird. Prof. Ito nennt dieses Leiden Muttermilchvergiftung, wenn Kakkesymptome der Stillenden fehlen.

brought forward in the excellent papers read, those of anæsthetic and position of the patient are of great importance.

Personally, I prefer local anæsthesia in every case possible. The question of position of the patient depends upon whether the foreign body is located in the upper part of the air passage or œsophagus. In such cases the Trendelenburg position should be used in order to avoid the danger of the foreign body being dislodged and drifting further down so as to make its location and extraction more difficult. The case I reported at the National Congress held in Washington this year to which Professor Jackson was kind enough to refer, was one in which the patient, aged 72, had a plate of false teeth embedded in his œsophagus, which he had swallowed during his sleep eighteen years previously. I shall not give details of the case, as these are contained in the original paper which will be published in the Transactions of the National Congress. I wish, however, to bring out one or two special points. The manipulation necessary to dislodge the plate from its bed of fibrous tissue was carried out as gently as possible in order to avoid further injury to the œsophageal structures. The fibrous tissue was pushed away from the upper edge of the plate, and then by patient interrupted traction and external manipulation the plate was dislodged and withdrawn without further trauma or loss of blood. The patient had been unable to swallow any solid food for eighteen years, and the muscles of the œsophagus from disuse had atrophied, so that after the removal of the foreign body the ability to swallow was still lost. I gave the patient a soft rubber catheter to insert into the upper part of the œsophagus in the hope that by continued training the muscles might be restored at least to partial action. After six months the improvement has been sufficient to enable him to swallow semi-solid food, and he has gained some 10 or 12 pounds in weight. By the increased muscular action a considerable amount of the fibrous tissue has been absorbed.

Dr. JULIUS LONDON (New York) described a new principle in stomatoscopy, and exhibited his gastroscope and cystoscope. The gastroscope consists of three major parts, an outer, middle, and inner sheath.

The outer sheath is simply a rigid straight round metallic tube, $\frac{1}{2}$ inch in internal diameter. It is bevelled at its distal end and flattened in one diameter so that the distal opening is oval while the proximal one is round. To the proximal end are attached a handle and a pin for holding the light reflector. This tube acts as a direct œsophagoscope and also as an introducer for the inner sheaths.

The middle sheath is 23 inches long and oval in shape. At the proximal end are attached two cocks for distension of the stomach and a catch for electric connexion. The distal end carries two electric lights and a terminal rubber tip. The distal end is connected to the main body of the instrument by two strong bars which carry the electric wires for the lamps.

The inner tube contains the optical system. This is composed of

a straight telescopic system and my objective lens, which is placed in front of the telescope and connected to it by two slender rods. The objective lens is made in two forms, one the bi-convex lens, and the other the perfect sphere. One half of this lens is silvered, the unsilvered portion being nearer the telescope, so that rays of light reflected from the mirrored surface can enter the telescope and be conveyed to the eye. This spherical objective has an angle of vision of 150° in water and 100° in air, and it shows the entire horizon in the same field without any rotation of the instrument, i.e. 360° in a plane at right angles to the long axis of the instrument. This objective has a practical working near point of $\frac{1}{4}$ inch. The telescopic system is of the terrestrial telescopic type, as in the Nitze cystoscope and subject to the modifications and refinements of the same, as in the Acmi Brilliant lens system which is used in present models. The image formed is inverted and reversed.

Practical Application. The stomach should be kept empty for at least twelve hours, then morphine administered one hour before anæsthesia is begun. I use the dorsal posture with elevation of the thorax and intratracheal anæsthesia of ether.

Method of Introduction. The operator stands behind the subject and passes the outer tube in at the right angle of the mouth until the larynx is seen, then engaging the distal end in the right pyriform sinus, the instrument is advanced until the opening of the œsophagus presents. Usually a moderate degree of pressure is necessary to shift the larynx to the left, before we can enter the œsophagus. The œsophagus is traversed under direct inspection, keeping the lumen fully in view throughout. After the stomach is entered, the inner tubes are inserted and the stomach distended with oxygen or air. If fluid is present it must first be aspirated.

Views obtained. In two dogs, on which I tested the instrument through Dr. Droper's courtesy at N.Y.U. and Bellevue Medical College, the view was satisfactory, the mucosa appearing clear and glistening, while the respiratory movements were marked. These were so characteristic as to suggest the possibility of diagnosing perigastric adhesions as well as gastric lesions. The pyloric antrum was distinctly seen.

The mechanical work was executed by M. R. Wappler. My cystoscope is built along similar lines.

Professeur docteur J. MOURET (Montpellier). — Au Congrès français d'Oto-Rhino-Laryngologie du mois de mai dernier j'ai exposé mon procédé d'œsophagoscopie et de bronchoscopie, qui consiste à mettre le patient en position du tronc fortement incliné en avant et la tête non en déflexion forcée, mais en simple extension. Je vous demande la permission de vous présenter les photographies de plusieurs malades de mon service, prises pendant les différents temps de l'introduction du tube endoscopique et de vous résumer les avantages de mon procédé sur le procédé classique. Je vous présente aussi les figures schématiques que j'ai dessinées, afin de mettre plus en relief les avantages et les inconvénients des deux procédés.

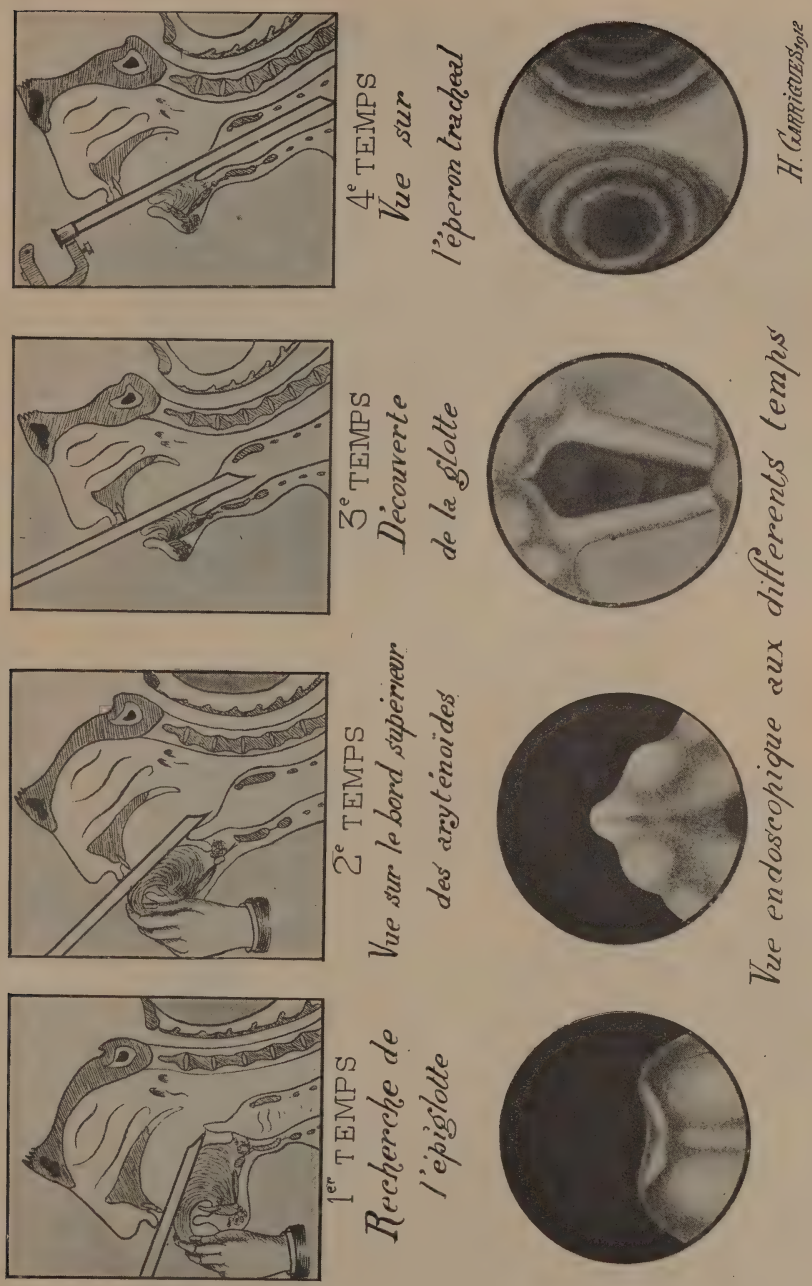


FIG. 1. Les schémas représentés dans cette figure montrent le passage du tube endoscopique dans les voies aériennes et les vues endoscopiques sur l'épiglotte, sur le bord supérieur des aryténoïdes, sur les cordes vocales et sur l'éperon trachéal.



FIG. 2. 1er temps : vue sur la luette. — L'opéré est assis à califourchon sur une chaise : le tronc et le bassin fortement inclinés en avant, le cou tendu et l'axe de la bouche horizontal. Il prend appui avec sa main gauche sur le dossier de la chaise. Avec sa main droite il tire la langue. L'opérateur placé en face de lui et fléchi sur ses jambes introduit le tube horizontalement au fond de la bouche et repère d'abord la luette.



FIG. 3. 2e temps : vue sur l'épiglotte. — L'opérateur relève un peu le manche de l'instrument et l'enfonce légèrement vers l'épiglotte qu'il reconnaît.



FIG. 4. 3e temps : vue sur la glotte. — Pour pousser le tube en arrière de l'épiglotte et aller vers l'entrée du larynx le manche de l'instrument doit être encore un peu relevé. A ce moment le patient lâche sa langue, qu'il tirait avec sa main droite, et complète son point d'appui sur le dossier de la chaise avec cette main devenue libre. L'opérateur, qui était fléchi sur ses jambes, se relève doucement en suivant le redressement du manche de l'instrument. Le tube endoscopique arrive alors au contact des dents de la mâchoire supérieure. Si les dents sont courtes et si le patient ouvre bien la bouche on pousse facilement le tube dans l'intérieur du larynx. Mais si les dents sont longues et si le patient ouvre mal la bouche (c'était le cas pour celui qui est représenté dans cette Fig. 4) le tube appuie contre les dents et l'opéré a tendance à redresser la tête et le tronc. L'opérateur doit alors mettre sa main gauche sur le sommet de la tête de l'opéré, afin d'arrêter ce mouvement, et, au lieu de lui permettre de redresser un peu le tronc, il faut, au contraire, lui ordonner de l'*incliner encore plus* et de *tendre* fortement le cou en avant. En agissant ainsi l'angle bucco-laryngo-trachéal s'ouvre encore davantage par son côté inférieur (côté colonne vertébrale) et reste horizontal par son côté supérieur (côté tête). Dans certains cas où le patient ouvre mal la bouche on peut, comme dans le procédé classique, introduire le tube par un des côtés de la bouche, au lieu de l'introduire suivant la ligne médiane.



FIG. 5. 4e temps : vue sur l'éperon trachéal. — Dès qu'on a franchi le larynx l'éperon trachéal apparaît aussitôt au fond de la trachée. Si l'opérateur ne l'aperçoit pas et voit seulement a paroi postérieure de la trachée, c'est que le patient n'est pas assez incliné en avant : il faut le faire incliner un peu plus, afin d'ouvrir au maximum l'angle bucco-trachéal par son côté inférieur. Le tube descend avec la plus grande facilité jusqu'à l'éperon trachéal.

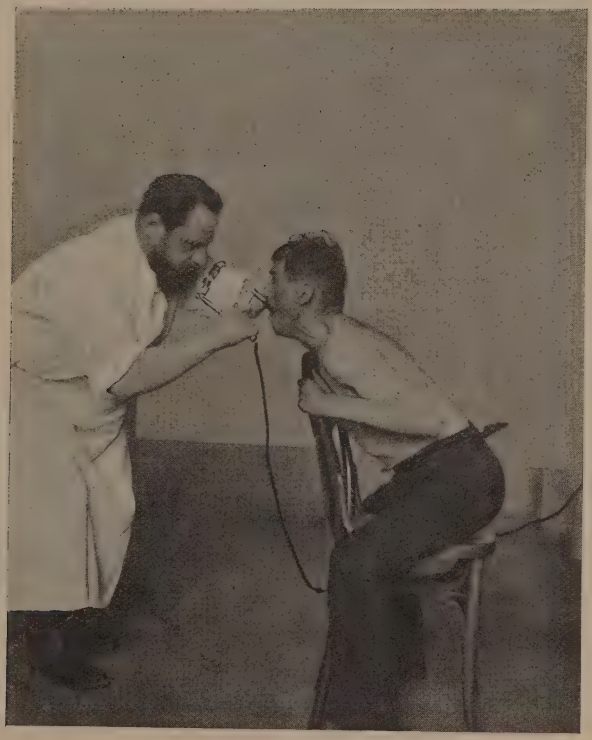


FIG. 6. 5e temps : vue dans la bronche. — Le tube bronchoscope est ici enfoncé dans la bronche droite à 32 centimètres de l'arcade dentaire supérieure. — Au niveau de l'éperon trachéal l'introduction du tube dans la bronche droite ou dans la bronche gauche se fait avec la plus grande facilité, en obliquant le bout de l'instrument du côté où l'on veut aller.

Les Figs. 5 et 6 montrent clairement que dans mon procédé d'endoscopie de l'œsophage ou des voies aériennes le redressement de l'angle bucco-œsophagien ou bucco-laryngo-trachéal se fait non par *en haut*, par le redressement de la tête et du côté buccal de cet angle, mais bien par *en bas*, par l'ouverture du côté vertébral. Le côté tête (axe buccal) reste horizontal, le côté vertébral s'ouvre en bas et en arrière.

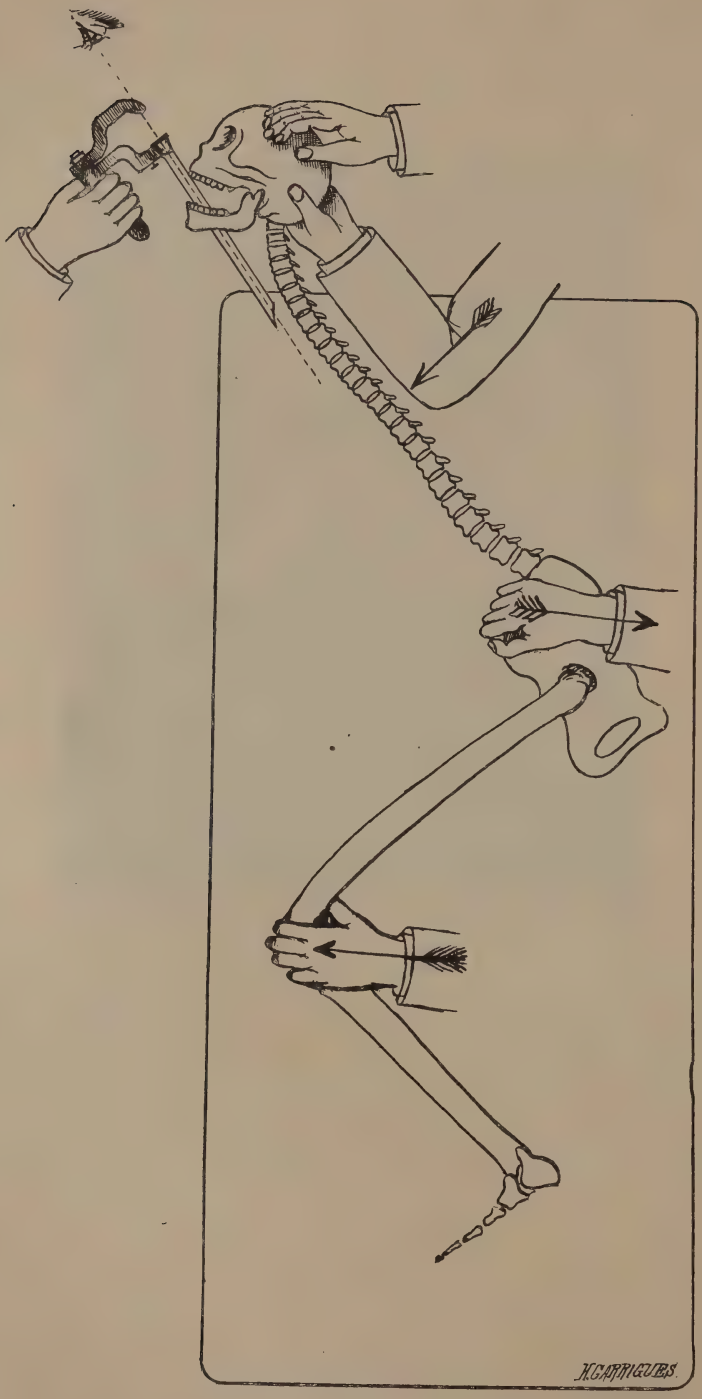


FIG. 7. Endoscopie en position couchée latérale droite.

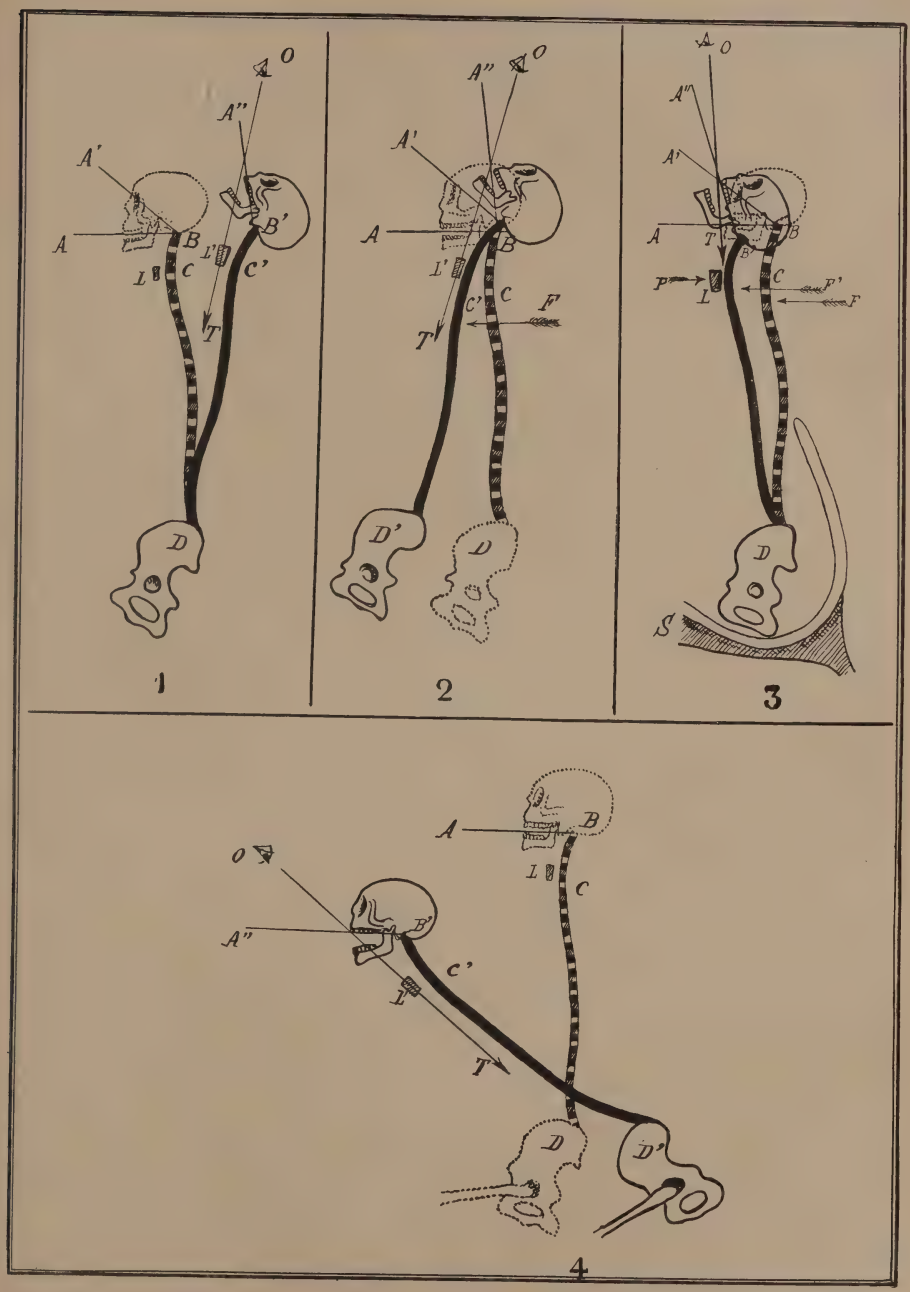


FIG. 8. Dans les schémas 1, 2, 3, j'ai représenté *en pointillé* le patient assis : tronc vertical et tête en position naturelle. En *traits pleins* j'ai représenté les trois principaux inconvénients qui, dans la bronchoscopie en position assise, tronc vertical et tête défléchie fortement, rendent difficile l'introduction du tube endoscopique.

Dans le schéma 4 j'ai représenté les rapports de la position assise naturelle avec la

position assise : bassin et tronc très inclinés en avant et cou tendu en avant. En comparant cette position avec celles qui sont représentées dans les schémas 1, 2, 3, on voit nettement les principaux avantages de mon procédé sur le procédé classique.

Les mouvements de flexion ou de déflexion de la tête se passent dans l'articulation occipito-atloïdienne. L'étendue des mouvements, que le jeu normal de cette articulation permet de faire, est limitée et moins grande qu'il ne le faut pour obtenir l'ouverture de l'angle bucco-laryngo-trachéal, qui est *nécessaire* pour qu'on puisse faire passer le tube de la bouche dans le larynx et dans la trachée. Dans chaque schéma *A'B D* représente le redressement de cet angle, que chacun peut faire par *en haut* naturellement et sans douleur : mais ce redressement est insuffisant. Pour que le tube endoscopique puisse passer de la bouche dans l'œsophage ou dans les voies aériennes il faut que l'angle s'ouvre davantage et que son côté supérieur (buccal) *A B*, s'ouvrant plus qu'il ne l'est en *A'B*, prenne une disposition pareille à celle de *A"B'*. Or comme la position *A'B* est la position-limite que le jeu naturel de l'articulation occipito-atloïdienne permet de faire, il faut, pour passer de la position *A'B D* dans la *A"B'D*, que l'angle bucco-laryngo-trachéal soit forcé (schéma 1). Pour forcer cet angle il faut que son sommet *B* puisse s'ouvrir davantage, ou bien que l'un de ses côtés soit suffisamment flexible pour permettre, quand même, une ouverture plus grande de l'espace compris entre ses branches. Or lorsque l'articulation occipito-atloïdienne a permis l'ouverture de l'angle *A B C D* jusqu'à la position *A'B C D*, c'est là le maximum d'ouverture qu'elle puisse donner. D'autre part le côté supérieur de l'angle (côté tête) est rigide et ne peut, pour sa part, présenter le moindre jeu d'extension. Au contraire la branche inférieure de l'angle (côté colonne vertébrale) peut, alors que l'articulation occipito-atloïdienne a atteint le maximum de son jeu, permettre encore une certaine ouverture de l'angle, grâce à la constitution même de la colonne vertébrale, qui forme le côté *flexible* de l'angle *A'B C D*. En effet la colonne vertébrale, grâce à ses articulations multiples, représente une colonne *flexible*, qui peut se laisser tordre sous l'influence de pressions excentriques : 1° Si le *flexible* est fixé à une seule de ses extrémités, on peut en tirant sur l'autre extrémité donner une obliquité plus ou moins prononcée à son axe ; par exemple, si l'on tire le point *B* en arrière, l'ensemble du *flexible*, c'est-à-dire la colonne vertébrale, sera tiré en arrière par cette extrémité qui prend la position *B'*, tandis que son extrémité inférieure *D* reste fixe (schéma 1) ; le mouvement inverse sera obtenu (schéma 2) si l'on tire sur l'extrémité *D*, celle-ci étant mobile et l'extrémité supérieure *B* étant fixe. 2° Lorsque les deux extrémités *B* et *D* du *flexible* sont immobilisées (schéma 3) on peut encore, grâce à l'élasticité propre au flexible (et ici l'élasticité de la colonne vertébrale est représentée par le jeu de l'ensemble des articulations vertébrales) lui faire exécuter certains mouvements en pressant latéralement sur une de ses faces. Par exemple : si l'on fait une pression sur sa face postérieure, suivant la flèche *F*, le *flexible* s'incurve en avant. Cette incurvation a pour résultat de diminuer un peu la ligne droite qui va du point *D* au point *B* et, par suite, de donner au point *B* qui est ici moins *immobilisé* que le point *D*, un léger déplacement en bas et aussi avant. Cette comparaison de la colonne vertébrale avec un *flexible* est indiscutable et il *faut* en tenir le plus grand compte, car c'est dans la *flexibilité* de la colonne vertébrale (côté vertébral de l'angle bucco-œsophagien ou bucco-laryngo-trachéal) que se trouve l'explication des difficultés que présentent la bronchoscopie et l'œsophogoscopie faites suivant le procédé classique, en même temps que se trouve aussi l'explication de la suppression de ces mêmes difficultés en employant mon procédé (schéma 4).

Schéma 1. — *A B C D*, angle bucco-œsophagien ou bucco-laryngo-trachéal dans la position verticale naturelle. — *A B*, axe antéro-postérieur de la bouche. — *C*, convexité de la colonne cervicale. — *D*, position du bassin. — *L*, larynx.

A'B D redressement de l'axe buccal par en haut, que chacun peut faire sans effort. — *A"B D* redressement *forcé* de l'angle bucco-œsophagien ou bucco-laryngo-trachéal, *nécessaire* pour pouvoir introduire le tube indoscopique. Pour l'obtenir la tête doit être mise en déflexion *forcée*. Mais alors, le jeu de l'articulation occipito-

atloïdienne ayant atteint sa limite possible et la déflexion forcée devenant douloureuse pour le patient, celui-ci produit l'ouverture *nécessaire* en portant en arrière l'extrémité supérieure de la colonne vertébrale. Dans cette position le patient perd la position verticale, *les épaules et la tête fuient en arrière.* — La flèche *T* montre le trajet du bronchoscope à travers la bouche et le larynx *L'*. — *O* indique la situation de l'œil de l'opérateur. Celui-ci doit dominer le patient et, étant donnée l'obliquité du bronchoscope, il est obligé de se mettre non en avant de lui mais par côté ou bien en arrière.

Schéma 2. — Pour éviter la fuite du haut du tronc en arrière, que provoque la déflexion forcée de la tête, l'aide, qui soutient celle-ci, immobilise aussi les épaules avec son coude gauche, en les maintenant en avant suivant la poussée représentée par la flèche *F*. Mais alors le patient cherche encore à échapper à la douleur, que provoquent la déflexion forcée de la tête et l'immobilisation des épaules, en fuyant par en bas : le bassin *D* glisse en avant (*D'*). Dans cette manœuvre le résultat est également défectueux et, en définitive, est le même que dans le schéma 1.

Schéma 3. — Pour éviter la fuite du tronc par en bas et en avant, le patient est assis sur une chaise spéciale de Brünings, dont le siège fortement incurvé empêche le bassin de glisser en avant. D'autre part l'aide immobilise les épaules en pressant sur elles suivant la direction de la flèche *F*. Dans cette position le tronc du patient ne peut donc plus fuir ni *par en haut*, ni *par en bas*. Mais la forte gêne que l'opéré éprouve alors pendant la déflexion forcée de la tête est plus désagréable encore dans cette sorte d'enclavement de tout son corps. Ne pouvant se soulager en fuyant ni par en haut, ni par en bas, il cherche à diminuer la douleur, qu'il ressent dans l'articulation occipito-atloïdienne trop étendue par la déflexion forcée de la tête, en faisant jouer la *flexibilité* de la colonne vertébrale. Sous la double influence de la déflexion de la tête et de la poussée des épaules en avant il cambre sa région lombaire et exagère la convexité que la colonne cervicale forme naturellement. La cambrure lombaire ne gêne en rien pour introduire le bronchoscope et soulage au contraire le patient. Mais l'exagération de la convexité de la colonne cervicale (trait plein *C'*), a pour résultat: 1° de faire une saillie contre laquelle vient buter l'extrémité de l'instrument; 2° de refouler le larynx *L* en avant, sous la base de la langue, et de rendre difficile et parfois impossible l'introduction du tube dans cet organe. Pour éviter cela Brünings dit qu'il faut se contenter d'une déflexion forcée *minima* de la tête. Malheureusement c'est là un point difficile à trouver sur certains sujets. Aussi cet auteur a-t-il imaginé de se servir alors d'un appareil propulseur du larynx en arrière, qui, refoulant cet organe suivant la direction de la flèche *P*, oblige cet organe à se dégager de dessous la base de la langue et à présenter son orifice à l'extrémité du bronchoscope, qui le cherchait vainement.

Grâce à ces diverses manœuvres la bronchoscopie finit par être possible dans la plupart des cas, mais elle reste très pénible pour le patient et de plus la position de l'opérateur (*O*), qui doit être plus haut placé que l'opéré et qui le domine verticalement, est loin d'être commode, non seulement pour faire un simple examen, mais surtout pour exécuter les manipulations que l'on peut avoir à faire à travers le tube endoscopique.

Schéma 4. — Ce schéma montre le passage de la position assise naturelle, tronc vertical, à ma position assise, *bassin et tronc fortement inclinés en avant.* Dans la position fortement inclinée en avant l'axe buccal *A"B'* est resté horizontal et parallèle à *AB* qui représente ce même axe dans la position assise normale. L'angle *A B D* s'est ouvert en *A"B'D'*, de façon suffisante pour permettre l'introduction du tube bronchoscopique *T*. Cette ouverture, au lieu de se faire par en haut, par le redressement du côté supérieur (*A B*) comme dans la position classique, s'est faite par en bas, par écartement du côté inférieur et postérieur *B D* (colonne vertébrale). Cette ouverture se fait ainsi *naturellement, sans effort, sans douleur*, et *sans le secours d'aucun aide*. Dans cette position le patient n'est plus emprisonné entre la chaise de Brünings et les bras de l'aide, qui lui enserre la tête en même temps qu'il refoule

ses épaules en avant : tout son corps est libre. En *inclinant* le tronc en avant et en *tendant* aussi le cou en avant il *tend* la colonne vertébrale et réduit au minimum la convexité naturelle de la colonne cervicale au lieu de l'exagérer : par suite, le larynx n'est pas refoulé en avant sous la base de la langue. L'introduction du tube endoscopique devient ainsi plus facile et moins pénible. L'opérateur trouve, en outre, une position plus commode pour lui-même, car, au lieu d'être obligé de dominer le patient et de se placer à côté ou en arrière de lui, il reste en face de l'opéré : il voit ainsi plus facilement au fond du tube et se trouve plus à l'aise pour exécuter les diverses manipulations qu'il peut avoir à faire à travers le bronchoscope ou l'œsophagoscope.

La bronchoscopie et l'œsophagoscopie peuvent être faites sous simple anesthésie locale ou bien sous anesthésie générale. Dans le premier cas, le patient peut être assis sur une chaise, dans le second cas, il doit être couché sur une table.

Bronchoscopie ou œsophagoscopie en position assise

Position assise, tronc vertical, tête fortement défléchie :

1° Le redressement de l'angle bucco-pharyngien se fait, *avec peine*, par *en haut* en défléchissant fortement la tête en arrière.

2° Un aide est nécessaire pour défléchir la tête et refouler les épaules, qui ont tendance à fuir en arrière.

3° La chaise de Brünings est utile pour empêcher le bassin de glisser en avant, lorsque les épaules sont retenues par l'aide.

4° La déflexion de la tête provoque un tassement des articulations vertébrales, qui se traduit par une exagération de la courbure antérieure de la colonne cervicale.

5° L'exagération de la courbure de la colonne cervicale pousse le larynx en avant et le porte à se cacher sous la base de la langue. On est alors obligé de se servir du contre-presseur de Brünings pour refouler le larynx en arrière.

6° Le tube endoscopique est introduit verticalement : pour cela l'opérateur doit dominer le patient et se placer à côté ou en arrière de lui.

7° Dans cette position, l'examen et surtout les manœuvres endoscopiques sont mal aisées.

Position assise, tronc fortement incliné en avant, tête en simple extension :

1° Le redressement de l'angle bucco-pharyngien se fait *naturellement* par *en bas*, en portant le bassin en arrière, en inclinant le tronc et en tendant la tête en avant.

2° Nul aide n'est nécessaire : les épaules se portent en avant et ne peuvent fuir en arrière.

3° La chaise de Brünings est inutile : le bassin ne peut glisser en avant.

4° En inclinant le tronc et en tendant le cou en avant, la courbure naturelle de la colonne cervicale tend à disparaître au lieu de s'accentuer.

5° Si le larynx fuit en avant sous la base de la langue, c'est que le patient au lieu de tendre simplement sa tête, la défléchit et incurve la colonne cervicale : il suffit de lui faire très légèrement le gros dos pour remédier à cet inconvénient.

6° L'opérateur reste toujours en avant du patient, le dominant légèrement : le tube endoscopique s'introduit d'avant en arrière, de façon plus commode pour le patient et l'opérateur.

7° Dans cette position, l'examen est moins pénible pour le patient, facile pour l'opérateur, les manœuvres endoscopiques se font avec plus de commodité et plus de sûreté.

Bronchoscopie ou œsophagoscopie en position couchée

Lorsqu'on veut endoscoper un patient sous anesthésie générale, il faut le mettre en position couchée. On peut faire la bronchoscopie et l'œsophagoscopie : 1° en position couchée sur le dos, le corps étendu et la tête en déflexion à l'extrémité de la table ; 2° en position couchée latérale droite ou gauche, le corps étendu, la tête tenue en déflexion ; 3° en position couchée latérale, *le tronc fortement incliné en avant et la tête en extension.*

(*a*) *La position couchée sur le dos, corps étendu sur la table et tête en déflexion* me paraît être la moins favorable. Dans cette position le corps est rectiligne, immobilisé, et ne peut subir aucun des mouvements qui peuvent faciliter l'introduction du tube endoscopique. Le degré de déflexion de la tête *suffisant* et *nécessaire* pour redresser l'angle bucco-pharyngien est difficile à apprécier : en général, il est exagéré et cela gêne l'introduction du tube au lieu de la faciliter ; d'autre part, dans cette position, le poids de la langue tend à faire tomber cet organe au-devant du larynx et oblige souvent à se servir d'une pince-langue et d'un abaisse-langue.

(*b*) Dans la *position couchée latérale, corps étendu, tête défléchie,* les difficultés sont les mêmes que dans la position assise ordinaire, le corps droit et la tête défléchie. Dans cette position, la déflexion forcée de la tête peut provoquer les mêmes ennuis: 1° fuite des épaules en arrière, ou bien, 2° fuite du bassin en avant, et 3° exagération de la courbure de la colonne cervicale, avec poussée du larynx sous la base de la langue.

(*c*) *La position couchée latérale, le tronc étant fortement incliné en avant et la tête en extension* donne les meilleurs résultats, comme dans la position assise. Il faut mettre le patient dans une position qui rappelle la position dite ' couchée en chien de fusil '. Toutefois, tandis que, dans la position couchée en chien de fusil, la tête est fléchie en avant, sur la poitrine, la tête doit, au contraire, être un peu relevée et mise extension légère, mais non en déflexion sur le dos.

Les membres inférieurs sont fléchis et ramenés vers l'abdomen. Le bassin est tiré en arrière vers le bord correspondant de la table. Les épaules sont poussées en avant, vers le bord opposé. La tête, attirée à l'extrémité de la table, est maintenue en extension par un aide. Cette position ressemble en tous points à la position assise, avec le tronc fortement incliné en avant. Comme dans celle-ci l'ouverture de l'angle bucco-pharyngien se fait par le *côté vertébral* de cet angle et non par le *côté tête.* Les avantages sont les mêmes.

Dr. WILLIAM HILL (London), showed a normal set of his modified endoscopes for the pharynx, larynx, bronchi, gullet, and stomach, and drew special attention to the value of the slotted laryngoscope as a guide for the passage of bronchoscopes and of gum elastic tracheal catheters for anæsthesia. He showed a special modification of his own of the latter, in which a rubber shoulder, fitted three inches from the distal end, rested on the ventricular bands and successfully prevented blood from entering the air passages during sanguinary operations on the nose, jaws and throat. This instrument was recommended as a cheap, efficient and safe substitute for Kuhn's peroral intratracheal anæsthetic apparatus. He had put his apparatus to a severe test and no blood

entered the air passages, even although the mouth and pharynx were full for many minutes.

He was astonished at Dr. Chevalier Jackson's pronouncement that neither local nor general anæsthesia was necessary for laryngoscopy and bronchoscopy in children. He would be very pleased to know how Dr. Jackson managed to abolish the laryngeal cough and spasm without the use of cocaine. Was it a case of hypnotic suggestion, or of superior technique? and could Dr. Jackson's assistants carry it out with equal facility? They had evidently something to learn here and he was anxious for instruction.

Dr. Hill demonstrated the forward position of the patient which he now employed; this he thought originated with Mosher and not with Johnson.

Dr. H. MARSCHIK (Wien) hat gute Erfolge mit zwei Mittelstellungen gehabt: 1. halb sitzend, halb liegend, bei ängstlichen und älteren, fetten Leuten; 2. bei Ansammlung von Schleim, der nicht mehr schnell genug aspiriert werden kann, Uebergang in eine der Mouret'schen ähnliche Stellung, bis der Oberkörper in horizontaler Lage ist, bei rückgebeugtem Kopf. Gastroskopie ist an der Klinik Chiari in Wien seit 1910 geübt mit dem Instrument von Foramiki, Chirurg in Wien, welches ähnlich dem London'schen Instrument ist. Die Erfolge sind sehr befriedigend. Referent erwähnt einen Fall von Tracheostenose durch peribronchiale Symptome, der wegen Membranbildung für Diphtherie gehalten wurde. Bei dem tracheotomierten Kind wurden mit der unteren Bronchoskopie massenhaft Membranen entfernt, aber wegen anscheinender Ausfüllung der Lunge damit die Operation abgebrochen. Exitus. Bei der Sektion war keine Spur mehr von den Membranen zu sehen. Das bronchoskopische Bild war vielleicht gleichzeitig durch Oedem verursacht. Aber ein ähnlicher Fall zeigt, dass endotracheales oder bronchiales Sekret und Membranen leicht postmortal verschwinden können.

Mr. HERBERT TILLEY (London), showed a large green pea which he had removed the preceding evening from the right bronchus of a man, aged 64 years, who had inhaled it two days previously.

He cocainized the larynx and upper region of the trachea, passed the tube into the right bronchus and lightly pressed the end of it against the pea. He then made a piston of some moist wool mounted on a long probe, passed this down to the foreign body and by a sudden retraction of the wool, sucked the pea into the tube and removed it.

The speaker failed to understand how timid women and children could be carefully examined without general narcosis. It was his custom to administer atropine and morphia an hour previous to the operation, and this had the double effect of quieting the reflexes and minimizing the secretions. Under general narcosis skilfully induced and maintained, one was never worried as to the mental condition of the patient and one knew one was giving no pain or discomfort.

Dr. HARRIS P. MOSHER (Boston, U.S.A.). For a number of years I have used the open speculum with great satisfaction. It had the drawback of being made in several sizes, but during the past year it has been improved and rendered adjustable, so that now one instrument is sufficient for all cases.

Ballooning of the œsophagus under strong pressure has proved in my hands one of the greatest helps with which I am familiar. No other method of diagnosing œsophageal pouches can rival it. No matter for what purpose the examination is undertaken, ballooning will always define the lumen of the œsophagus.

Within the last year it has been found, that in certain cases of cancer of the œsophagus repeated curettings under general anæsthesia were tolerated and that they restored the patient's ability to swallow soft solids, and very materially prolonged life.

Dr. A. LOGAN TURNER (Edinburgh) said that he wished to join in the discussion in order to offer his congratulations to Professor Killian upon the introduction of his suspension laryngoscopy. His own experience with the method was not yet large, but it was sufficient to demonstrate to him its great usefulness.

There was no better method of examining the larynx in young children, and both in children and in adults it was most serviceable for operative purposes. Again, in the diagnosis of post-cricoid carcinoma he had been able to satisfy himself of the superiority of the suspension method over the œsophageal tube and in two cases recently in which a coin had been lodged in the mouth of the œsophagus, it was readily seen and extracted without the necessity of introducing a tube.

It was further a most useful way to demonstrate to a class anatomical and pathological conditions of the larynx and hypo-pharynx and he thought that it might be usefully employed for the purpose of photographing the movements of the vocal cords.

Mr. E. B. WAGGETT (London). One point raised for discussion by Professor Chevalier Jackson, was to the effect that endoscopic manipulations should be retained in the hands of a few surgeons. It would be misleading if this were allowed to pass without comment; every young laryngologist should consider it his bounden duty to make himself expert in this procedure, now that the methods have arrived at a high degree of perfection.

Dr. R. H. SKILLERN (Philadelphia). I wish to mention particularly the value of the tracheo-bronchoscope as a diagnostic means. We have used this extensively in our clinic at the Medico-Chirurgical College in cases of obscure cough, dyspnœa, dysphagia, and hæmoptysis with the utmost satisfaction and in many instances it has immediately cleared

up an obscure diagnosis. This endoscopic method should be a routine procedure in every clinic devoted to the treatment of diseases of the nose and throat.

Replies

Professor KILLIAN (Berlin). Es ist mir leider nicht möglich, ein vollständiges Schlusswort zu unserer langen und interessanten Diskussion zu geben, weil ich nicht alle Redner hinreichend verstanden habe.

Die grosse Menge der neuen Instrumente, welche demonstriert wurden, zeigt, mit welchem Eifer an der Verbesserung der Methoden gearbeitet wird. Sie erhalten bei den verschiedenen Nationen und in den Händen verschiedener Aerzte ein durchaus individuelles Gepräge. So weit ich es im Augenblick beurteilen kann, scheint mir an den neuen Instrumenten vieles sehr beachtenswert, insbesondere an den Oesophago- und Gastroskopen. Der Apparat von Lewisohn erinnert an eine alte Konstruktion von Nitze-Leiter in Wien aus der Zeit von v. Mikulicz. Man wird mit dem Instrument zwar Diagnosen stellen, aber kaum operativ vorgehen können.

Ueber Krankheitsfälle wurde, von den Fremdkörpern abgesehen, wenig gesprochen. Beim Oesophaguskarzinom wurde von Mosher empfohlen, Auskratzungen zu machen. Ich bezweifele nicht die guten Erfolge, die Mosher damit hatte. Ich muss aber zugleich zu bedenken geben, dass wir leicht Schaden mit den Auskratzungen stiften können. Von den Blutungen abgesehen, sind es die Infektionen, die wir fürchten müssen. Die Patienten können leicht Mediastinitis und Pleuritis bekommen und daran rasch zu Grunde gehen.

Sehr gefreut habe ich mich über die verschiedenen Mitteilungen, welche wir über die Schwebelaryngoskopie erhalten haben. Es ergibt sich daraus, dass diese Methode mehr und mehr Anklang findet und auch in den Händen anderer gutes leistet. Eine Demonstration meines neuesten Instrumentes und seiner Anwendung am Lebenden hoffe ich Ihnen morgen geben zu können.

Zu dem, was Chevalier Jackson über Auge und Hand gesagt hat, möchte ich noch hinzufügen, dass der Arzt vor allem auch sich selber in der Gewalt haben muss. Wenn er auf Schwierigkeiten stösst, so muss er nicht *um jeden Preis* direkt und von innen vorgehen wollen. Es ist viel besser, im rechten Moment den inneren Weg aufzugeben und den äusseren zu wählen, weil er sicherer ist. Insbesondere warne ich nochmals davor, bei kleinen Kindern, ausser in den ersten Stunden, gequollene Bohnen mit der oberen Bronchoskopie entfernen zu wollen. Es ist dies so eminent gefährlich, dass stets die Tracheotomie ausgeführt werden sollte. Die Statistik kommender Jahre würde sich danach viel günstiger gestalten. Ich selber habe leider einen Bohnentodesfall zu beklagen, und wenn er sich auch schon vor einigen Jahren ereignete, so kann ich ihn doch gar nicht aus der Erinnerung verlieren. Ich mache mir in diesem Falle den Vorwurf, den unteren Weg zu spät eingeschlagen zu haben.

Dr. CHEVALIER JACKSON (Pittsburg, Pa., U.S.A.). As I am unfortunate in not understanding any language other than English, I am unable to discuss the remarks of a number of the speakers.

Dr. Lewisohn referred to the dangers of the straight tube, and I quite agree with him. The straight tube is dangerous unless passed very carefully and skilfully and under guidance of the eye. On the other hand, I believe we should not consider Dr. Lewisohn's own instrument as totally free from danger. It is perfectly safe when passed by Dr. Lewisohn, or by any other careful man; but the absolutely safe œsophagoscope will never be devised, if by safe we mean one that can be passed with perfect safety without either skill or care. I saw Dr. Lewisohn introduce his œsophagoscope; it went down promptly and the view I got was excellent. I consider it a good instrument that will probably assist largely in our study of œsophageal conditions, and I think that all such efforts should be encouraged.

I heartily endorse Professor Kyle's preference for local anæsthesia in the instance referred to. The case he mentions is a beautiful demonstration of careful work and is a contrast to the violent removal that one is tempted to effect when the forceps grasp the foreign body. Skill such as possessed by Dr. Kyle renders possible the removal of these large bodies for which many authorities consider external œsophagotomy is required.

I was unable to understand the remarks of Professor Mouret, but the position of the patient as demonstrated is, so far as the head and neck are concerned, precisely that recommended by Professor Kirstein, and as depicted in all the instrument catalogues nearly twenty years ago. I think Professor Mouret's use of the back of a chair is an excellent suggestion, as it helps to keep the patient in the proper position.

Dr. Mosher said he did not know who was the first to use the open speculum. It was Mosher himself. It also was he who first demonstrated the advantages of flexion of the neck to relax the anterior muscles. Dr. Johnston gives Dr. Mosher full credit for this, but Johnston places the patient on the back and does not turn the head to the side, though obtaining the fullest advantage of the relaxation of the neck muscles, as first demonstrated by Mosher, without using the rotation of the speculum to open up the œsophagus. I quite agree with Dr. Mosher as to the advantages of ballooning the œsophagus. He demonstrated them to me six or eight years ago at the Massachusetts General Hospital.

I quite agree with Mr. Waggett that laryngologists should use endoscopic instruments constantly; if they do so, of course my remarks in regard to the occasional operator will not apply. That is just the point that I wished to make, and that I thought I made, clear in my paper. I gathered from the answers to my circular letters of inquiry that those who were doing direct work constantly were getting excellent results, while those who only did it rarely, and were very poorly equipped for the

work as to instrumentarium, assistants, and nurses, were not getting equally good results.

Dr. Skillern is doing excellent work, and I regret that he did not express his opinions more fully. He is, himself, so skilful a bronchoscopist that I feel sure he does not realize the difficulties that the occasional operator encounters.

I congratulate Dr. Tilley on the beautiful work in the case of the man who had aspirated the pea. It emphasizes, I believe, one of the points that I tried to make in my paper in regard to the unnecessary tracheotomies that are done for bronchoscopy; these could be dispensed with if the bronchoscopist would only try from above. I feel that later it will be the same in regard to bronchoscopy without anæsthesia; those who have criticized it most this morning have never tried it.

Dr. London's excellent work on gastroscopy came to my attention after my report was written. I have seen his instrument demonstrated, and it gives an excellent view of the interior of the stomach.

In reply to Dr. Hill, I beg to refer to my paper, where the reasons for strictly aseptic technique are given as 'essential in a procedure which necessarily frequently comes in contact with tuberculosis, diphtheria, lues, and other infectious diseases'. Of course, I always wear a mask as protection not only to the patient, but to myself. Large protective glasses are also worn, and two sets of these are always at hand so that the nurse can put a cleaned pair on me when those worn become soiled.

I fear my position in regard to anæsthesia has been misunderstood. I have never said that I did not use general anæsthesia at all. There are certain cases in which I think it is advisable and always use it, as mentioned in my paper. I did not say that there is no cough when working without anæsthesia, for there is cough, though usually it subsides or lessens materially in a short time. In adults, for bronchoscopy I sometimes use local anæsthesia, rarely general, and occasionally none at all. Just before leaving the United States, I removed from a woman of 48 years of age a collar button which had been in her lung for twenty-five years. Her condition was profoundly septic from the foreign body abscess having burst into the pleura, and being almost moribund, no anæsthetic, general or local, was to be thought of. She told me a number of times afterwards that there was no pain, though there was a disagreeable choking sensation.

The psychic element has absolutely nothing whatever to do with work on children under six years of age without anæsthesia. We do not ask the child's consent. We have an organization of from four to six people, and we go ahead and do the endoscopy. It is team work. That it is not a severe ordeal is demonstrated by the fact that, as mentioned in my paper, a number of my patients have been the children of physicians, and the parent-physician was, in each case, present at the operation. Each and every one of them expressed his gratification that no anæsthetic was used. It seems to me that this is the supreme test, and especially so

as the physicians did not realize, as we do, that bronchoscopy and œsophagoscopy introduce into anæsthesia many elements of danger that do not pertain to anæsthesia in other kinds of work. To those who are sceptical, I would suggest that in the next foreign body case in a child under six years of age they try it without anæsthesia, and when a case comes along that also requires a tonsillectomy, I would suggest that the tonsillectomy be done also without anæsthesia, as is done everywhere in Germany, I believe. Then ask the patient which of the two ordeals was the worse. You will find in every instance the answer will be 'tonsillectomy'.

SECTION XV

RHINOLOGY AND LARYNGOLOGY

INDEPENDENT PAPER

QUE DEVIENNENT LES SINUS OPÉRÉS?

Par M. le Professeur E. J. MOURE, de Bordeaux

Les opérations faites sur les cavités accessoires de la face ont, depuis ces dernières années, passé par des phases successives, particulièrement intéressantes au point de vue de leur évolution. C'est ainsi que la cure radicale des suppurations de l'antre maxillaire a trouvé des partisans acharnés et convaincus de l'opération endo-nasale (soit par le méat moyen, soit par l'inférieur) et, en France surtout, le drainage de l'antre par les voies naturelles agrandies jouit encore d'une certaine faveur. Quelques praticiens (Mahu, Vacher) ont bien ajouté à l'ouverture simple le curettage plus ou moins incomplet de l'antre d'Highmore, fait avec des curettes coudées, mais on conviendra que par cette voie il est difficile d'aller dans tous les recoins, les recessus et les anfractuosités d'une cavité souvent très spacieuse, anguleuse et même cloisonnée.

L'opération par la fosse canine (Caldwell-Luc), plus ou moins modifiée dans ses détails par ses auteurs et par bon nombre de praticiens, reste encore et quand même la méthode de choix, au moins pour les cas rebelles de sinusites maxillaires.

La suite de ma communication fournira, je crois, un argument péremptoire aux partisans de la cure radicale que l'anesthésie locale à la cocaïne a rendu beaucoup plus simple et bien plus facile à pratiquer.

Pour ce qui a trait aux suppurations de l'antre frontal, je ne m'occuperai pas des procédés qui consistent à les traiter et même à les guérir parfois, en agissant par les voies naturelles. Je suis convaincu que ces méthodes ont donné des résultats encourageants, définitifs au moins momentanément, entre les mains de quelques praticiens expérimentés. J'ai déjà dit par ailleurs pourquoi je pensais que cette manière de faire ne pouvait pas être généralisée. D'autre part, de l'avis même de ceux qui ont préconisé la mise en pratique de ce mode de traitement, il existe des cas dans lesquels il faut, de toute nécessité, recourir à l'opération par la voie externe et, cette fois encore, les spécialistes ne sont pas d'accord sur le modus faciendi.

Beaucoup estiment que, pour bien nettoyer une cavité frontale, il est indispensable de la mettre à nu dans sa totalité, en détruisant toute la paroi antérieure (procédé Kuhnt) dans le but, avait dit le promoteur

de la méthode, d'accoler la peau à la paroi interne du sinus et d'obtenir ainsi une occlusion complète de la cavité opérée. Killian, surenchérissant encore, pour mieux obtenir cet accolement en apparence indispensable, avait conseillé l'opération dont tout le monde connaît la technique, que je n'ai pas à rappeler ici.

En réalité, toutes ces interventions ont été imaginées et exécutées dans le but de supprimer le plus complètement possible l'antre frontal. Or, nous savons, d'une façon tout à fait certaine : 1° que cette suppression idéale est irréalisable dans bien des cas ; 2° que malgré la persistance de certains soi-disant points morts, nos malades parviennent à guérir complètement et définitivement ; 3° que le seul point véritablement important, mis plus tard en évidence, était la suppression des lésions de l'ethmoïde, pendant trop longtemps négligées. De telle sorte que des opérations frontales sérieusement et complètement exécutées aboutissaient à diminuer la suppuration et non à la tarir, les cellules ethmoïdales infectées continuant à déverser du pus dans le canal infundibulaire.

La plupart des auteurs ont également pensé qu'il était indispensable d'assurer un drainage fronto-nasal constant en élargissant au maximum le canal qui fait communiquer le sinus du front avec l'intérieur des fosses nasales. De ce drainage dépendait, croyait-on, la guérison définitive du malade.

Je suis bien convaincu, depuis déjà longtemps, que ces diverses conceptions étaient et sont encore plus théoriques que cliniques. Pendant de longues années, j'ai eu des succès durables en pratiquant la cure radicale simple, réduite au minimum comme traumatisme extérieur et, comme beaucoup de mes confrères, c'est pendant la période où j'ai voulu ouvrir largement les cavités frontales et ethmoïdales que j'ai connu les insuccès, les déboires et même les accidents. Aussi n'ai-je pas tardé à reprendre les procédés du début, c'est-à-dire l'opération idéale d'Ogston-Luc à laquelle j'ai ajouté, il est vrai, lorsque c'était nécessaire, l'évidement de l'ethmoïde antérieur en général, et postérieur, plus rarement.

Il m'est absolument démontré, à l'heure actuelle, par une expérience déjà longue, que si le curettage de la cavité frontale est bien exécuté, s'il est complet, c'est-à-dire si toute la muqueuse qui la recouvre est enlevée, même si ce nettoyage est fait par un orifice de dimensions restreintes, si les cellules diverticulaires (en général rétro-nasales) derrière les os propres, sont ouvertes et curettées avec soin, enfin si l'ethmoïde est désinfecté d'une manière consciencieuse, le drainage fronto-nasal n'a nullement besoin d'être aussi large et aussi persistant qu'on a bien voulu le dire.

Il suffit, en effet, de savoir ce que deviennent les cavités ainsi évidées et asepsiées pour comprendre l'importance du fait que je viens d'avancer. Ayant eu l'occasion d'ouvrir à nouveau des cavités frontales et maxillaires opérées une première fois, soit parce que j'avais laissé des cellules diverticulaires ou aberrantes qui suppuraient encore, soit parce que j'avais fait un curettage ethmoïdal incomplet, je n'ai pas eu de peine à m'apercevoir que des antres, très spacieux au moment de l'opération,

n'existaient pour ainsi dire plus lorsque j'intervenais à nouveau. Toutes les cavités étaient comblées par une sorte de tissu fibreux, les parois osseuses elles-mêmes s'étaient rapprochées l'une de l'autre et le sinus avait en quelque sorte disparu. Il ne restait plus, en réalité, que les cellules suppurantes, à la recherche desquelles il me fallait aller pour guérir définitivement mes malades.

Ce fait de l'oblitération cicatricielle des sinus attira mon attention et de là à conclure qu'il était tout à fait inutile de laisser un canal naso-frontal largement ouvert, puisqu'il ne pouvait plus drainer un recessus non existant, il n'y avait pas loin.

D'autre part, si les sinus opérés se ferment et se comblent, pourquoi faire des opérations aussi étendues, aussi déformantes que celles de Kuhnt ou de Killian ? N'est-il pas dès lors évident que, pour opérer et guérir une sinusite, il suffit d'avoir un orifice convenable pour bien voir dans l'intérieur, afin de pouvoir les curetter et enlever toute la muqueuse infectée ou non, condition essentielle pour obtenir l'oblitération post-opératoire ? C'est pour ce motif que les opérations de Caldwell-Luc, pour le maxillaire, et d'Ogston-Luc pour le frontal, sont et restent toujours, à mon sens, les opérations idéales lorsque ces cavités suppurantes nécessitent une opération par voie externe.

Étant donné que les cavités sinusiennes opérées par l'extérieur se comblent et sont à jamais supprimées, non seulement on obtient la guérison de la sinusite, mais on débarrasse également le malade de la préoccupation d'une récidive possible. Au contraire, dans les cas où les sinusites maxillaires ou frontales ont été traitées par les voies naturelles et même guéries par ces procédés, on doit toujours craindre de voir ces cavités suppurer à nouveau sous l'influence des mêmes causes qui ont déjà occasionné leur infection une première fois et à diverses reprises (coryza, grippe), etc.

Dans ces cas, les antres Highmoriens et frontaux ne s'oblitérant pas, ils conservent la forme et la configuration qu'ils avaient pendant qu'ils suppuraient. Le fait peut être vérifié, d'une part à la diaphanoscopie, qui démontre généralement le retour de la transparence normale dans ces cas, tandis que chez les opérés de cure radicale le sinus, quoique guéri, reste définitivement opaque, car il n'existe plus ; d'autre part, un stylet, ou un trocart, pénètre encore aisément et se promène dans les sinus maxillaires ouverts et traités par les voies naturelles, tandis qu'il est impossible de le faire pénétrer dans les antres traités et curettés par voie externe.

La conclusion pratique est facile à tirer.

Ainsi s'expliquent les succès que donnent les méthodes sanglantes externes, dites radicales. Toutefois pour traiter les sinusites frontales chroniques, on doit poser un principe : qu'il faut avant tout nettoyer les cavités ethmoïdales par les voies naturelles. Cet évidement de l'ethmoïde est d'une exécution simple et exempte de danger. Elle doit être le stade préliminaire de l'antrotomie par voie externe. Pour faire cette dernière, on ne fera au frontal que l'ouverture strictement nécessaire

pour bien voir sa cavité avec tous ses diverticules s'il en existe. On fera, par cet orifice esthétique, un curettage parfait et bien complet de tout le sinus dont toute la muqueuse devra être enlevée. J'ai, pour ma part, l'habitude d'écouvillonner l'intérieur avec un petit tampon de ouate monté sur un stylet fin, imprégné d'une solution de chlorure de zinc au $\frac{1}{10}^{e}$. Le tampon doit être assez petit pour s'insinuer jusqu'au fond de toutes les rainures et bien détacher les parties de la muqueuse encore adhérente. Une série de petites curettes permet de compléter le nettoyage de l'antre frontal.

Ceci fait, bien explorer l'infundibulum du côté de l'unguis, derrière les os propres du nez, où existent assez souvent des cellules diverticulaires, agrandir ensuite le canal naso-frontal jusque dans la cavité du nez.

On peut terminer par un bourrage du sinus à l'aide d'une lanière de gaze iodoformée ou vioformée inodore d'une largeur de 2 à $2\frac{1}{2}$ cent., dont l'un des bouts est introduit jusque dans la fosse nasale, par laquelle on le sortira 3, 4 ou 5 jours plus tard ; l'autre extrémité de la gaze sort par l'angle interne de l'incision cutanée, entre deux points de suture.

Cette manière de faire permet de s'assurer que toute la gaze a été enlevée, elle donne aussi la certitude d'avoir, pendant toute la période de cicatrisation, 5 à 6 semaines au moins, un drainage constant. D'après ce qui précède, on n'a pas à se préoccuper, ultérieurement, de l'existence de ce canal artificiel dont le sinus inexistant n'a plus aucun besoin.

J'avais déjà fait ces réflexions et jeté les bases de mon travail [1] lorsque parut, dans les *Archiv für Laryngologie*, janvier 1913 (Bd. xxvii, H. 1. S. 137), et plus récemment encore dans les *Archives internationales de Laryngologie*, n° 2, mars–avril 1913, un article du docteur Ssamoylenko démontrant, d'une façon expérimentale, les idées que j'avais sur l'oblitération post-opératoire des cavités maxillaires et frontales. C'est avec le plus grand plaisir que j'ai lu ce travail original, fait dans la clinique du docteur Simanowski, de Saint-Pétersbourg, dans lequel l'auteur expose les résultats d'expériences exécutées sur des chats (16), et sur des chiens (8).

L'auteur, sous la narcose chloroformique, ouvrait l'un des sinus, qu'il nettoyait et curettait avec beaucoup de soins, enlevant toute la muqueuse à la cuiller tranchante, laissant le sinus opposé intact, aux fins de contrôle. Il sacrifiait ensuite ces animaux, tantôt 14 jours, puis 4 semaines, 1 mois, 3 mois, 5 mois, $6\frac{1}{2}$ mois après l'opération. Il put ainsi constater que, dès les premiers jours qui suivaient l'intervention, le périoste avait augmenté de volume, puis à mesure qu'on s'éloignait de l'époque de l'opération apparaissait un tissu fibreux cicatriciel qui, peu à peu, comblait l'intérieur de la cavité sinusienne opérée.

Ce tissu de nouvelle formation, examiné au microscope, à des grossissements variés, démontra à l'auteur que le sinus s'oblitérait par reproduction d'os nouveau et par une sorte de tissu fibreux. L'oblitération était complète en l'espace de 6 mois, quelquefois même plus tôt.

[1] C'est, en effet, en 1912, avant l'apparition du mémoire de Ssamoylenko, que j'ai adressé à Londres le titre de cette communication.

D'où l'auteur conclut encore que, la structure de l'os étant identique chez l'homme et chez l'animal, une oblitération des sinus frontaux était également possible de la même manière qu'elle se produisait sur les animaux. Cet auteur démontrait, en outre, d'une façon péremptoire, que ce n'était point la muqueuse nasale qui pénétrait dans le sinus frontal pour l'oblitérer, fait impossible, mais bien un tissu de nouvelle formation et il ajoutait encore, en manière de conclusion (n° 7), que dans les opérations de sinus frontaux chez l'homme, il fallait veiller à l'enlèvement total de la muqueuse et au côté cosmétique de l'opération, sans qu'on soit obligé d'avoir recours à des mesures spéciales pour produire les conditions favorables à l'oblitération du sinus.

Cette démonstration expérimentale de la suppression des cavités opérées contient un enseignement extrêmement pratique ; elle prouve, en effet, que pour guérir un sinus maxillaire ou frontal fongueux et chroniquement infecté, on doit en faire le curettage complet.

C'est donc là, je le répète, un fait d'une importance capitale, puisqu'il permet, en réduisant au minimum l'ouverture externe, d'effectuer des opérations qui ne doivent être ni déformantes, ni mutilantes. De cette manière on diminue, dans des proportions considérables, la gravité des actes opératoires que beaucoup de praticiens hésitent à pratiquer, à cause des conséquences néfastes qu'ils avaient eu quelquefois à déplorer.

Ce que je viens de dire pour les sinusites frontales, que certains opérateurs n'abordent qu'avec une certaine crainte, je puis le répéter pour les sinusites maxillaires.

En effet, si l'on décortique toute la muqueuse Highmorienne, on est tout étonné, quelques mois plus tard (5 à 6) de ne plus pouvoir pénétrer dans le sinus qui, au moment de l'opération, avait été trouvé très large et très spacieux. Ici, encore, il s'est fait le même travail réparateur que dans les sinus frontaux : la cavité a disparu, comblée en partie par le resserrement de l'os et aussi par un tissu fibreux de nouvelle formation.

Ainsi s'expliquent du reste le peu de déformations jugales que laissent même des ouvertures très larges faites au niveau de la paroi canine.

Si l'on songe également à ce que deviennent les malades auxquels on a enlevé tout ou partie du maxillaire supérieur, si l'on se rend compte de la réparation véritablement extraordinaire qui se produit au niveau d'une brèche osseuse, pourtant très large et très considérable au début, on ne sera pas étonné que la nature cherche à combler des cavités qui ont été dépouillées de leur revêtement intérieur pendant les opérations désignées à juste titre sous le nom de ' cures radicales '.

Cette démonstration, à la fois clinique et expérimentale de l'oblitération post-opératoire des sinus réduit encore à néant les procédés qui consistent à supprimer les cavités frontales en les comblant avec des produits plus ou moins aseptiques (pâte de Mosetig-Moorhof, plombages variés, etc.).

J'engage donc mes collègues à méditer ce fait, dont l'importance pratique ne saurait leur échapper.

DISCUSSION

Dr. Maurice (Paris). Nous sommes d'avis, comme le prof. Moure, que les opérations de Caldwell-Luc et d'Ogston-Luc sont idéales. Nous ne croyons pas que le *drainage prime le curettage*, mais nous ne pensons pas non plus, même avec les cures radicales, que le malade est débarrassé de la préoccupation d'une récidive. Nous pouvons citer une récidive chez une malade un an et demi après un Caldwell-Luc fait par nous : le sinus était comblé en partie et des fongosités nouvelles avaient apparu ; celles-ci disparurent sous l'influence d'enfumage iodé. Nous sommes d'avis que la cavité frontale s'obstrue presque toujours après l'opération, mais il n'en est pas de même (30 pour cent environ) pour le sinus maxillaire.

C'est le curettage de toute la muqueuse qui favorise l'obstruction, mais il y a des cas où vraiment il serait abusif d'agir ainsi, lorsqu'en particulier il n'existe des fongosités qu'au niveau de l'ostium ou du plancher du sinus maxillaire.

Sir Robert Woods (Dublin) expressed his general agreement with Dr. Moure's views. He was of opinion that the most satisfactory method for chronic cases was the external one. When the mucous lining was degenerated mere drainage was not enough for cure, for it was notorious that the drainage in many chronic cases was already perfectly free. The principle of the external operation (say on the frontal sinus) was of the simplest kind. It consisted in complete removal of the degenerated lining membrane. This substituted a raw granulating surface for an epithelial one, and virtually converted the epithelial-lined cavity into an abscess. Fusion of the growing walls thus took place, and obliteration was the result. Therefore, the answer to the question as to how much bone should be removed from the front wall of the sinus was—just as much as and no more than enabled the operator to be quite sure that he had complete access to every portion of the sinus, so as to be certain that no epithelium was left.

He had operated on more than one hundred cases of frontal sinusitis with completely satisfactory results, and without fatality. He therefore hesitated to try the endonasal route, and thought it likely that it would never supplant the methods of obliteration.

Dr. H. Marschik (Vienna). In Chiari's clinic in Vienna we have had some cases of recurrence after radical operation on the frontal and maxillary sinus, caused by leaving a recess lined with mucous membrane in the zygomatic bone, which became closed by the very quickly contracting scar tissue before the recess had filled up. In the radical treatment of these sinuses we are therefore coming to adopt as thorough a technique as possible, so that we like to take away a large part of the anterior and also of the inner bony wall.

Dr. Trétrôp (Anvers, Belgique). Il y a treize ou quatorze ans un oto-rhino-laryngologiste belge, feu le Dr de Greift, d'Anvers, prétendait qu'on n'obtenait la guérison des sinusites qu'à la condition de supprimer complètement la muqueuse, mais toute la muqueuse par le curettage. Il opérait, également comme M. Moure, par voie externe. La communication prématurée du Dr de Greift au Congrès annuel de la Société belge à Bruxelles n'eut pas le succès qu'elle méritait. Les observations du Dr Moure semblent confirmer que notre collègue belge avait raison.

Dr. Luc (Paris). Je suis heureux d'apporter ici le résultat de mon expérience personnelle à l'appui de celle du professeur Moure ; mais il importe d'insister sur la nécessité d'un curettage complet du foyer pour que se produise son comblement par du tissu fibreux, et j'ai la conviction que les prétendues *récidives* invoquées contre les opérations par voie externe étaient simplement le résultat d'une désinfection incomplète. Le professeur Moure nous a fait remarquer que ce comblement fibreux nous dispense de pratiquer des brèches osseuses très étendues. J'ajouterai qu'il nous dispense également de compliquer l'opération par la taille de lambeaux destinés à amorcer l'épidermisation de la surface interne du foyer.

Enfin le plus grand avantage des méthodes d'ouverture large par voie externe me paraît consister dans la possibilité qui en résulte pour nous de *visiter* l'intérieur de la cavité suppurante.

Cette année même, j'ai eu l'occasion d'opérer et de guérir par ma méthode deux malades dont la suppuration maxillaire avait résisté à un certain nombre de lavages. Or à l'ouverture des deux foyers, je trouvai, dans l'un, un séquestre mobile, dans l'autre, un petit projectile dont la pénétration remontait à 18 ans. Je n'ai pas besoin de vous faire remarquer que, ni dans le premier ni dans le second cas, l'opération par voie nasale n'aurait permis de découvrir et de supprimer la cause de la persistance de la suppuration.

Dr. Halle (Berlin). Es geht nicht an, prinzipiell bei Erkrankungen der Nebenhöhlen eine radikale Operation zu machen. Es gibt selbstverständlich eine grosse Reihe von Fällen, wo nur die externe Operation in Frage kommen kann, z. B. bei Fremdkörpern, Komplikationen u. dgl. In anderen Fällen kann die externe Operation nur dann prinzipiell angeraten werden, wenn man überzeugt ist, dass die chronisch erkrankte Schleimhaut nicht rückbildungsfähig ist. Das ist sie aber sicher. Beweise dafür gibt die tägliche Erfahrung. So *müssen* wir, auch in Fällen von chronischer Eiterung, zuerst die konservative Behandlung versuchen (Eröffnung der Höhle vom unteren Nasengang mit irgend einer Methode, Spülung etc.). Führt sie nicht zum Ziel, so kommt die externe Operation in Frage. Hier ist die Methode nach Luc-Caldwell, ihre Erweiterung durch Denker, oder endlich die Methode nach Canfield-Sturmann am Platze. Letztere ist nichts weiter als eine Denker'sche

Operation von der Nase aus. Schnitt am vorderen Ansatz der unteren Muschel entlang der Apertura piriformis, Freilegen des Os maxillare, breite Eröffnung der Höhle, zum Schluss Abwärtsklappen der Schleimhaut des unteren Nasenganges in die auskurettierte Höhle. Operation unter lokaler Anästhesie. Hiermit habe ich die besten Erfolge.

Dasselbe gilt von der Operation der Stirnhöhle. Auch hier gibt es Fälle, wo die Indikation zu einem externen Eingriff strikt gegeben ist. Aber es gibt viele Fälle von chronischer Stirnhöhleneiterung, die intranasal geheilt werden können. Auch refusieren viele Patienten die äussere Operation aus Furcht vor der oft sehr unangenehmen Narbe. Ich erinnere an meine intranasale Operation und führe aus, dass man die ganze untere Wand der Stirnhöhle entfernen und von der oft riesigen Oeffnung die Höhle behandeln oder, exzessiv grosse Höhlen ausgenommen, mit biegsamen Kuretten die Schleimhaut entfernen kann. Ritter hat Dilatatoren zur Erweiterung der Stirnhöhle angegeben. Kann man diese einführen, so gelingt die intranasale Eröffnung der Höhle mit dünner birnförmiger Fraise ohne jede Gefahr, wenn man medial nicht über den Ansatz der mittleren Muschel hinausgeht. Von 28 Fällen, die ich nach dieser Methode behandelt habe, und die zum Teil 20 Jahre krank waren, musste einer von aussen operiert werden, ein anderer sezernierte noch stark. Die übrigen wurden geheilt.

Dr. Guisez (Paris). L'idée que le tissu fibreux comble les cavités des sinus dans le processus de guérison semble être tout à fait exacte. Dès 1901 nous avions pu faire cette constatation en réopérant un malade atteint de pansinusite. Les cavités frontales étaient remplies de tissu fibreux. De nos constatations ultérieures il résulte que ce tissu fibreux n'est pas constant dans les sinusites maxillaires opérées par le procédé de Caldwell-Luc. Mais il paraît nécessaire dans la vraie guérison des sinusites frontales. Aussi pour le favoriser nous laissons la plaie légèrement ouverte à son angle tout interne pendant les huit premiers jours et nous touchons à la teinture d'iode son intérieur, pratique qui nous a paru beaucoup faciliter la production du tissu fibreux. Mais pour sa production il est indispensable que le curettage soit complet, et pour cela une brèche horizontale suffisamment longue pour qu'on puisse atteindre les angles du sinus est indispensable. A la condition de maintenir les saillies osseuses il ne persiste dans la suite aucune déformation chez nos opérés.

Un large drainage est indispensable du côté du nez pendant toute la période où le tissu fibreux se forme, et la seule façon de l'obtenir est de réséquer largement l'ethmoïde, ce qui nous paraît insuffisant dans l'opération d'Ogston-Luc. Nous l'obtenons en réséquant une partie de l'os propre du nez et en attaquant l'ethmoïde horizontal d'avant en arrière.

Mr. Herbert Tilley (London). It is possible to cure some cases of chronic frontal sinus suppuration by freeing the lower end of the fronto-nasal canal, and particularly the cell on its outer wall which Mosher has

carefully described. Having thus established free drainage, the sinus may be cleansed, dried, and the fungosities of the mucous membrane destroyed by injections of nitrate of silver, 60 to 80 grains to the ounce. The débris can be removed in forty-eight hours and the sinus then irrigated daily for a week or ten days with a pint of warm water to which a drachm of tincture of iodine has been added.

Complete obliteration of the sinus is the only means of absolutely preventing any future infection. Failure after external operation is due in most cases to infection by means of orbito-ethmoidal cells. Only by an external operation can one safely deal with this disposition of cells.

In 1903 the speaker, in a communication read before the Odontological Society of London, described the results of curettage of the antrum and exhibited a diagram which illustrated the filling of the sinus by a plug of fibrous tissue.

He thinks that it is wiser to curette only diseased mucous membrane and to leave the normal mucous membrane, which will tend to spread over and cover those parts which have been denuded by the operation.

If scar tissue only remains suppuration will occur after mild infections, because granulations cover the fibrous plug and leucocytic exudation must take place.

Professor KUBO (Japan). Ob man die Nebenhöhlen von aussen oder innen operieren muss, hängt von der Indikationsstellung ab. Gewisse Fälle heilen durch Ausspülung. Im Nichtfalle muss man an eine starke Veränderung der Höhle denken, manchmal cystisch, eitercystisch oder mit Polypen begleitet, was radikale Entfernung der Schleimhaut erfordert. Ich operiere Kieferhöhle nach Luc und Stirnhöhle nach Killian von aussen mit totaler Ausrottung der Schleimhaut. Es kommt manchmal vor, dass eine makroskopisch fast gesund oder verdickt aussehende Schleimhaut zeitweise mit Eitercysten versehen ist.

Durch Reoperation von einmal operierten Sinusitis maxillaris sieht man, dass die Wiederherstellung der Kieferhöhlenauskleidung durch zwei Vorgänge vor sich geht: (1) Durch Hineinwachsen und Wucherung (zeitweise mit Verknöcherung verbunden) des Weichteils der vorderen Wand. Diesen Vorgang konnte ich durch X-strahlenphotographie bei einer Reihe von Fällen feststellen. (2) Durch Hineinwachsen des Epithels von den Oeffnungen im mittleren und unteren Nasengang.

Nach der Kieferhöhlenoperation muss das Fenster für eine Zeitlang als Drainage offen bleiben, sonst kommt die Infektion der Blutkoagula oder Blutgerinselmasse in die Höhle. Dafür halte ich für notwendig, dass die Schleimhautlappen die Knochenränder des Fensters möglichst gut decken, was durch einen schrägen Kreuzschnitt der Schleimhaut gut erzielt wird. Die 4 Lappen werden nach 4 Richtungen umgeschlagen, um die Ränder des Fensters zu decken. Die Gazestreifen werden nur so viel tamponiert, dass sie die Schleimhautlappen an den Fensterrändern

halten. Gazestreifen werden nach 3 Tagen vom Mund aus entfernt. Sekundäre Nahtanlegung wird gleich nachher vorgenommen.

Professeur JACQUES (Nancy). La condition indispensable à une cure véritablement radicale de sinusite chronique purulente est — après ablation aussi complète que possible de la muqueuse dégénérée (éventuellement des séquestres) — d'assurer un drainage large, direct, permanent des sécrétions post-opératoires. La suppression immédiate, opératoire, de la cavité n'est nullement indispensable. C'est un principe que j'ai défendu depuis dix ans à propos de mon procédé de cure radicale de la sinusite frontale, procédé visant à éviter toute déformation extérieure. Pour le réaliser j'attaque le sinus par la région supéro-interne de l'orbite ; puis les dimensions et la configuration de la cavité étant repérées, je supprime tout le plancher du sinus, et, par cette large brèche inférieure, une exploration minutieuse et un nettoyage complet de la portion ascendante du sinus est presque toujours réalisable avec l'éclairage frontal. L'esthétique du visage est ainsi respectée. La cavité sinusienne est réduite immédiatement de moitié. Ce qui en reste affecte la forme d'un cône creux, ouvert en bas, vers l'ethmoïde. Le deuxième temps de l'opération consiste dans une résection bien complète du labyrinthe ethmoïdal entre la lame papyracée en dehors et le cornet supérieur en dedans. Ainsi se trouve maintenue la graisse orbitaire et se trouvent respectées les gaines lymphatiques des filets olfactifs, voie possible d'infection pour l'espace céphalo-rachidien. La cavité frontale désinfectée et réduite de volume est assurée d'un drainage large et direct pendant un temps largement suffisant à son comblement progressif, à son oblitération fibreuse. Cette oblitération secondaire garantit tout aussi sûrement contre la récidive qu'une suppression opératoire immédiate. Elle respecte en outre totalement l'esthétique de la face ; car la dépression résultant de la trépanation frontale et de la résection de l'apophyse montante et de l'os nasal est tout à fait inappréciable après quelques mois.

Mr. E. B. WAGGETT (London). With regard to curettage of the antrum the speaker asked Dr. Moure to describe the immediate convalescence after this operation. The radical antral operation, as generally conducted in this country (opening through the canine fossa, followed by removal of a large portion of the antro-nasal wall below the inferior turbinate, and retention of the mucous membrane with the exception of polypi and gross hypertrophies), was a minor operation, taking some five or ten minutes to perform. The immediate convalescence was extremely simple and the patient was practically well in a week. In the speaker's experience recurrence, though theoretically possible, never occurred where a large antro-nasal opening had been made ; he therefore wished to know if the convalescence after extensive curettage was equally simple.

Dr. Donelan (London) desired to express his agreement with what had been so well said by Mr. Waggett. He had published in 1909 (*Lancet*) a series of 103 cases, in which a large part of the naso-antral wall had been removed together with the middle third of the inferior turbinal. He had introduced some rectangular chisels and an angular perforator for facilitating this procedure, and had since operated on a further series of more than 200 cases. In only 6 cases of the 300 had it been found necessary to perform the Caldwell-Luc operation. He entirely agreed with Dr. Moure that the antrum often became obliterated, but in some of his cases in which he had been able to follow the subsequent history he found this happened quite as readily when only a portion of the wall had been removed. In an adult man, with an antrum of normal size, a piece of the wall as large as a shilling could be removed in a few seconds, and this gave adequate drainage and access for further treatment.

Reply

Professeur Moure (Bordeaux). En résumé, les rhinologistes restent toujours divisés en deux camps : ceux qui veulent traiter les sinusites par la voie endonasale et les partisans de la cure par l'extérieur. Il est bien entendu que nous éliminons de cette discussion les sinusites que peuvent guérir des moyens simples (ablation de dents, par injections, etc.) pour nous occuper seulement des sinusites réellement chroniques. Or, j'estime toujours que la cure dite radicale peut seule nous donner un résultat définitif, et il faut, comme je l'ai déjà indiqué, enlever toute la muqueuse. En effet, cette dernière, saine en apparence, est presque toujours malade. Je suis à ce sujet d'accord avec le professeur Killian. Je conclurai en disant que nous avons deux procédés : l'un qui peut guérir par les voies naturelles, laissant les malades exposés à des récidives ; l'autre radical, parce qu'il supprime la cause suppurante. Le procédé de Desault, autrefois employé par les chirurgiens généraux, arrivait au même résultat.

SECTION XV

RHINOLOGY AND LARYNGOLOGY

INDEPENDENT PAPER

DE CERTAINS PROGRÈS DANS LA THÉRAPEUTIQUE DES AFFECTIONS DE L'ŒSOPHAGE, DE LA TRACHÉE ET DES BRONCHES, ET EN PARTICULIER DE LA RADIUMTHÉRAPIE DU CANCER DE L'ŒSOPHAGE

Par M. le Dr. J. GUISEZ, de Paris

Dans cette communication nous ne reviendrons pas sur la technique de la broncho-œsophagoscopie, dont tous les détails ont été discutés magistralement par les rapporteurs, MM. les Drs Killian et Chevalier Jackson, ni sur la question des corps étrangers, mais nous aurons en vue certains points de notre pratique concernant la pathologie de l'œsophage, de la trachée et des bronches, et en particulier la thérapeutique endoscopique de quelques affections de ces conduits.

1° *Œsophage.* — Dans plusieurs travaux parus antérieurement et depuis 1908 nous avons insisté sur une forme de sténose de l'œsophage peu connue, mais cependant assez fréquente, puisque nous avons pu en réunir cent cinquante cas ; nous voulons parler des *sténoses inflammatoires* de ce conduit. L'œsophage est susceptible en effet de se rétrécir par un processus inflammatoire simple, donnant lieu à des affections à évolution lente progressive à forme souvent grave. Ces rétrécissements inflammatoires ont deux sièges d'élection principaux : l'origine et la terminaison ; c'est-à-dire les portions exactement canaliculaires de ce conduit.

On sait en effet que la moyenne partie de cet organe doit être regardée *comme une véritable cavité* ; seules les deux ou trois centimètres de la portion cervicale qui font suite à la bouche de l'œsophage et la portion terminale ou *cardiaque*, c'est-à-dire celle qui du niveau de l'hiatus diaphragmatique s'étend jusqu'à l'estomac sont capables de se rétrécir par l'œsophagite chronique, tout comme l'urètre se rétrécit dans l'urétrite chronique. La cause intime de cette œsophagite est la suivante : le spasme était à l'origine d'un grand nombre des cas que nous avons suivis. Le spasme crée une stase momentanée tout d'abord, puis de plus en plus durable, amenant à sa suite de l'inflammation des parois œsophagiennes. L'œsophagite chronique amène petit à petit une altération, un épaississement des parois œsophagiennes et les sténoses inflammatoires de l'œsophage

se présentent bientôt sous deux formes : la *forme hypertrophiante* avec néoproductions inflammatoires simulant souvent le cancer (*pseudo-cancers*) et la *forme fibro-cicatricielle*. Dans ce dernier cas, le plus fréquent, on a affaire à une sténose fibreuse dure, se laissant difficilement franchir à la bougie filiforme qui cherche à la cathétériser. Elle affecte tous les caractères d'un rétrécissement cicatriciel.[1]

Ces sténoses sont prises la plupart du temps pour des spasmes à forme grave ; elles constituent de véritables rétrécissements organiques qui évoluent progressivement jusqu'à la fermeture complète de l'œsophage. Elles sont cause, si elles siègent à l'origine, de *diverticules de l'hypopharynx* (diverticules *par pulsion*), et si elles sont au niveau du cardia, elles déterminent les *grandes dilatations* dites autrefois *idiopathiques de l'œsophage*.

L'alimentation difficile par intermittence au début, pendant la période spasmodique devient de plus en plus malaisée aussi bien pour les aliments solides que pour les liquides, jusqu'à devenir tout à fait impossible : le pronostic de cette affection est donc particulièrement grave.

La dilatation progressive faite avec des bougies olivaires tout d'abord sous le contrôle de l'œsophagoscope ; l'*usage de la bougie à demeure* pour faire le trajet dans les cas difficiles et au début, l'électrolyse circulaire combattent et guérissent ces sténoses. Les néodilatations pathologiques, dilatations, diverticules se trouvent du même coup heureusement influencés et disparaissent peu à peu au fur et à mesure que la cause productrice se guérit. Nous suivons depuis plusieurs années des malades dont le diverticule ou la grande dilatation œsophagienne a diminué dans des proportions considérables depuis le début du traitement.

Nous avons, dans un travail paru tout récemment, rapporté quatre cas de *sténoses congénitales* de l'œsophage.[2] Le premier concernait un jeune homme de dix-huit ans qui depuis son enfance n'avait jamais pu prendre que du lait ; il était cependant arrivé à l'âge adulte, mais avec un développement incomplet et des formes efféminées. A ce sujet on peut se demander si cette anomalie de développement était due à l'alimentation exclusivement lactée ou était liée à la même cause qui avait également empêché l'œsophage de se développer complètement.

L'examen œsophagoscopique nous a montré qu'il existait une valvule au niveau de l'hiatus diaphragmatique, qui obstruait à peu près complètement l'œsophage, ne laissant qu'un *tout petit orifice gros comme une tête d'épingle* rejeté vers la droite. Il fut possible de dilater cet orifice et par l'œsophagotomie de sectionner cette valvule pour donner à ce malade une alimentation normale.

Dans le deuxième cas il s'agissait d'un jeune enfant de 10 ans qui lui non plus n'avait jamais pu prendre que du lait et de l'eau ; tout autre aliment était immédiatement rejeté. Nous fîmes au point de vue œsophagoscopique les mêmes constatations que dans le cas précédent, quoique l'orifice reliquat de la lumière œsophagienne fut un peu plus large. Ici encore, formes efféminées et arrêt du développement des organes génitaux.

[1] Voir *Rapport du Congrès de Chirurgie*, octobre 1912.
[2] Voir *Presse médicale*, avril 1913.

Le traitement fut le même et l'alimentation revint à la normale assez rapidement. Dans les deux dernières observations la sténose était incomplète.

En janvier dernier, le Dr Legendre, de Paris, nous a adressé un jeune homme de 33 ans, qui depuis son enfance a toujours souffert de troubles de la déglutition. Il traversait fréquemment des périodes où toute alimentation était impossible et où les aliments étaient invariablement rejetés. Ces troubles remontent à la toute enfance, mais toujours les crises étaient suivies de périodes où l'alimentation se faisait de façon à peu près normale. Malgré cela le malade a pu grandir, se développer et faire ses études de droit. Les traitements les plus divers ont été essayés et le diagnostic qu'on donnait le plus communément à son affection était ' spasme de l'œsophage '.

Il existait ici encore un peu au-dessus de l'orifice diaphragmatique une sorte de valvule ne laissant de la lumière qu'un orifice rejeté vers la droite ; orifice ne présentant également que quelques millimètres de large. Les bords en étaient granuleux, rouges, saignant facilement et sur son côté gauche avait poussé un polype pédiculé, jouant sans doute dans la genèse des crises dysphagiques un rôle par l'obturation mécanique de l'orifice restant de la lumière œsophagienne.

Enfin, chez une jeune fille de 17 ans, l'aspect valvulaire mais incomplet de la sténose était également le même et le siège encore dans la région du cardia.

Comme on le voit, certains caractères se retrouvent toujours dans ces différents cas. D'abord les troubles, remontant à la toute enfance, existaient déjà pendant l'allaitement, c'est ce qui permet de dire qu'*il s'agissait de rétrécissements congénitaux ; l'aspect valvulaire* était toujours le même (comme dans tous les rétrécissements congénitaux quelqu'en soit le siège (urètre, rectum)), le siège toujours identique au niveau de la région cardiaque de ce conduit. Le traitement basé tout entier sur les constatations œsophagoscopiques fut très simple et ces malades doivent leur guérison à nos constatations endoscopiques.

Le *cancer de l'œsophage* semble avoir bénéficié, à la suite d'un diagnostic précis par l'œsophagoscopie, d'un traitement local direct par les applications de radium. Depuis six ans nous avons soumis tout une série de malades à cette thérapeutique et nous désirons vous rapporter ici quelques cas où l'effet semble avoir été non seulement palliatif mais curatif.

Le premier cas concernait un cancer de l'œsophage à forme particulièrement grave, qui semble non seulement amélioré mais guéri par les applications de radium. La déglutition, impossible auparavant, est redevenue normale bien que l'amélioration ait été longue à se manifester. Actuellement, c'est-à-dire $2\frac{1}{2}$ ans après la fin du traitement, la santé du malade semble absolument normale ; il ne présente plus aucun symptôme rappelant son affection œsophagienne.

M. D., 52 ans, est présenté au Dr Labourré, d'Amiens, envoyé par le Dr Boury, de Ham, en mars 1911, pour examen œsophagoscopique, avec l'anamnèse suivante : son père est mort de cancer de l'œsophage ; sa

mère de cancer de l'estomac. Lui même avale avec difficulté croissante, depuis 6 mois, il a maigri de 25 livres.

Le Dr Labourré pratique l'œsophagoscopie sous anesthésie locale, le 28 mars, avec un tube de Guisez de 13 mm. de diamètre, on pénètre jusqu'à la bouche œsophagienne où on est arrêté un quart d'heure. A 19 cm. de l'arcade, la muqueuse présente déjà un changement de coloration (petit piqueté hémorragique). A 20 cm. la paroi est épaissie, raidie, immobilisée ; la paroi présente un relief assez considérable, étalé sur la moitié gauche de l'œsophage, au niveau du rétrécissement aortique ; les troubles de déglutition s'expliquent à la fois par le volume de la tumeur, son siège en un point rétréci naturellement, et le spasme secondaire de la bouche œsophagienne.

Le malade est revu six semaines plus tard, la tumeur a encore progressé, la déglutition est plus pénible encore, le poids est tombé de 102 à 85 kg. Le Dr Labourré conseille de recourir à une gastrostomie, mais d'essayer auparavant des applications de radium à Paris chez le Dr Guisez, qui confirme le diagnostic de cancer.

L'examen œsophagoscopique nous fit constater au tiers moyen de l'œsophage un épithélioma à forme infiltrante et bourgeonnante, à forme circulaire ; sténose à peu près complète. L'*examen histologique*, fait par le Dr Deglos (février 1911), nous a donné comme résultat : *épithélioma tubulé.*

Applications quotidiennes de 0,05 gr. de radium. Huit séances de quatre ou cinq heures consécutives. La déglutition est alors plus facile. Le Dr Labourré revoit le malade en mai pour la troisième fois et constate que la tumeur est lisse et affaissée, pâlie, ne saigne plus, est souple ainsi que la région avoisinante. Il y a vraiment amélioration considérable.

En mai, nouvelle application de radium, 0,075 gr. pendant 5 heures, cinq jours consécutifs. En juillet, nouvelle application de 0,10 gr. pendant cinq jours. Le malade avale mieux, mais ne retrouve pas l'appétit, il maigrit encore de 82 kg., il fléchit à 77.

Le Dr Labourré revoit le malade en octobre, la déglutition n'est pas satisfaisante, il demande à l'œsophagoscoper, le malade refuse. Quelques jours plus tard, il fait appeler le Dr Boury parce qu'il ne peut plus avaler, même de l'eau. Le Dr Boury arrive péniblement à cathétériser son œsophage avec une bougie de très faible diamètre. L'obstacle siège environ à 20 cm. de l'arcade dentaire, mais non sur les extrémités de l'œsophage ; il ne s'agit donc pas de spasme. Deux hypothèses se présentent à l'esprit : ou bien il y a récidive de la tumeur, ou bien transformation fibreuse et rétrécissement par action excessive du radium. Le diagnostic différentiel n'est pas établi, car le malade se refuse toujours à un nouvel examen. L'évolution ultérieure devait seule trancher la question en faveur d'un rétrécissement fibreux.

La dilatation œsophagienne est continuée à l'aide de bougies olivaires œsophagiennes. L'amélioration va croissant, au point qu'en six mois il retrouve à peu près son poids normal à 3 ou 4 kg. près. C'était en mars 1912, plus d'un an après le premier examen œsophagoscopique.

Depuis lors, l'amélioration a persisté, le poids s'est maintenu, le teint du sujet n'est plus blafard mais blanc clair, rosé aux joues. L'appétit est excellent. Aucun dégoût pour aucun aliment. Le pain, la viande, légumes sont avalés pourvu qu'ils soient suffisamment réduits en purée soit par l'apprêt culinaire, soit par une mastication suffisante. Le sujet est gai, plein d'entrain, et vaqué à ses occupations habituelles.

En avril 1913 (exactement le 17) l'ex-malade, revu par les docteurs Boury et Labourré, est en aussi bon état que six ans auparavant, bien avant le début de son cancer.

Dans ce cas particulier, la déglutition, d'abord meilleure est devenue plus pénible ; nous avons démontré la transformation en fibromes des cancers améliorés ou guéris. La réaction fibreuse a peut-être été excessive par un excès d'irradiation. Quoiqu'il en soit, fin 1911, on pouvait encore craindre une issue fatale, car l'état général ne cessait d'empirer.

Or, nous savons que tout cancéreux est profondément intoxique et peut-être la destruction des cellules cancéreuses par le radium ne fait-elle qu'ajouter encore à cette intoxication. De plus, le rétrécissement œsophagien fibreux secondaire de guérison augmente encore la cachexie alimentaire. Le cathétérisme de l'œsophage, en rétablissant la perméabilité, a donc été heureux.

Quant aux injections 'd'antiseptic-oil' (des Drs Luchini et Peretti), il semble qu'elles aient été ici d'un heureux effet, en luttant contre l'intoxication de l'organisme. On leur doit déjà la guérison de quelques cas de cancers superficiels, cutanés, et même de cancers intestinaux.

Il est bien évident que le traitement du cancer n'est pas univoque, mais extrêmement divers. Si le radium présente vraisemblablement le maximum d'action destructive, que nous puissions diriger contre les cellules cancéreuses elles-mêmes, tous les agents physiques, toutes les méthodes antiseptiques, tous les sérums destinés à améliorer le fonctionnement des glandes digestives et endocrines des poumons et de la peau, sont capables d'aider le médecin à triompher du cancer.

Ce cas représente donc un cas curieux de guérison intégrale d'un cancer œsophagien par le radium : le cathétérisme. Les injections d'huiles essentielles qui ont été faites entre temps semblent avoir agi surtout sur l'état général comme remontant.

Dans plusieurs publications antérieures[1] nous avons relaté les résultats encourageants obtenus par ce traitement tout spécial chez tout une série de malades :

Le premier cas concerne un malade chez lequel nous avons eu la chance de faire le diagnostic œsophagoscopique, confirmé par l'examen histologique, tout à fait au début, semble-t-il, de son épithélioma. Il s'agissait d'un malade, qui nous fut amené par le Dr Lemoult, de Paris, et qui vint nous consulter, au mois de novembre 1911, pour de la gêne de la déglutition, gêne qui s'était manifestée depuis six mois, et qui actuellement était tellement accusée que le malade ne pouvait plus avaler que les aliments liquides ; certains jours même, toute alimentation devenait impossible. Il vint nous demander un examen œsophagoscopique, lequel fut fait le 26 novembre 1911, sous cocaïne.

A quelques centimètres au-dessus du cardia, on remarque, sur la paroi postérieure de la muqueuse œsophagienne, une sorte de zone rouge, de la grandeur à peu près d'une pièce de cinq francs. En examinant de plus près, on voit qu'il existe, vers le centre de cette zone, une sorte d'ulcération, grande à peu près comme une pièce d'un franc, dont les bords sont bourgeonnants. Cette ulcération n'est pas très profonde,

[1] Voir *Bulletin et Mémoires de la Société médicale des Hôpitaux*, 2 avril 1910, en collaboration avec le Dr Barcat, 29 mars 1911, et *Gazette des Hôpitaux*, 21 mai 1912.

mais la paroi, sur laquelle elle repose, est comme cartonnée et figée. Si on la touche avec un porte-coton, elle saigne assez abondamment. Ces caractères nous faisant craindre l'épithélioma, nous en prenons un fragment pour faire un examen histologique. Celui-ci, fait par notre collègue M. Bauer (laboratoire Corrion-Hallion), donne les résultats suivants :

' Le fragment examiné contient du tissu réticulé riche en cellules lymphoïdes et des glandes en tubes à épithélium calciforme, avec quelques îlots, très restreints d'épithélium pavimenteux stratifié. L'épithélium calciforme du revêtement est constitué par des cellules remarquablement hautes : il tapisse de petites cryptes anfractueuses séparées par des papilles très irrégulières, dentelées. L'irrégularité de cette surface, le développement considérable des cellules épithéliales, sont en faveur du diagnostic *épithélioma à cellules cylindriques*.'

Traitement. — Applications de radium ; quantité variant de 2 à 5 centigrammes à deux jours d'intervalle.

Comme effet, au bout de la cinquième séance, il remarque que les brûlures ou aigreurs œsophagiennes, dont il se plaignait, ont complètement disparu.

Nouvel examen œsophagoscopique le 18 décembre 1911 ; l'œsophagite intense, qui existait dans les deux tiers inférieurs de la muqueuse œsophagienne, a complètement disparu. Les lésions, que nous avions remarquées lors de notre premier examen, sont modifiées.

(*a*) L'ulcération a partiellement disparu, elle est remplacée par une surface lisse, rouge. A la partie inférieure, il existe encore deux ou trois bourgeons d'apparence un peu suspecte ; ceux-ci saignent au contact du porte-coton, tandis que le reste de la tumeur ne donne plus d'hémorragie (lors de notre premier examen l'hémorragie était très abondante par le simple contact du porte-coton).

(*b*) En outre, la perméabilité œsophagienne est beaucoup augmentée, puisque nous pouvons franchir le cardia avec un tube de 11 mm. et pénétrer facilement dans l'estomac.

Une nouvelle œsophagoscopie nous montre la disparition de tout bourgeonnement (avril 1912). On voit une surface cicatricielle grisâtre au niveau même de l'ulcération.

Dans un autre cas, il s'agissait d'un malade soigné en mai 1912. L'œsophagoscopie nous montra qu'il y avait là une masse infiltrante. Le malade, âgé de cinquante-trois ans, amené par le Dr Lian, chef de clinique à la Faculté de Médecine, vint nous consulter pour des troubles de dysphagie. Ce malade présentait, à l'œsophagoscope, une masse épithéliomateuse faisant corps avec la paroi postérieure de l'œsophage à 8 cm. de l'origine de ce conduit.

L'examen d'un fragment enlevé, fait par le Dr Deglos, nous donne comme résultat net : épithélioma pavimenteux, à forme infiltrante, avec masses néoformées, présentant les caractères de l'*épithélioma pavimenteux lobulé*.

A la suite de deux séries d'applications de radium, la déglutition redevient normale. Il peut reprendre son métier de plombier.

Nouvel examen œsophagoscopique en octobre 1912. Nous voyons qu'il existe encore une masse faisant corps avec la paroi de l'œsophage, au point même où siègeait la tumeur, que nous avons observée la première fois ; mais cette masse a diminué certainement de volume ; elle est lisse, ne présente pas de bourgeonnement, ne saigne pas au contact. Nous en prenons un fragment, pour l'examen histologique, lequel, fait par le Dr Deglos, donne le résultat suivant :

' Il semble que la tumeur histologiquement ait changé de nature, dans

ce cas particulier, et que d'épithéliomateuse elle est devenue véritablement inflammatoire.'

Nous venons de revoir ce malade tout récemment (juillet 1913, 1½ ans après le début du traitement). Il existe au niveau où siégeait la tumeur une sorte d'infiltration et d'induration de la paroi ; mais plus d'apparence de tumeur proprement dite. Le tube œsophagoscopique de 11 mm. descend sans aucune difficulté jusqu'à l'estomac. Nous l'avons présenté à la Société des Chirurgiens de Paris, le 14 février 1913.

On connaît les bons résultats obtenus par la radiumthérapie dans le cas de cancer de la peau, et c'est même, peut-on dire, dans cette localisation qu'a triomphé le radium. Il semble que ce médicament ait une action de prédilection dans les cancers à marche lente, le cancroïde cutané et les autres tumeurs analogues superficielles.

Il est une variété de cancer viscéral qui présente avec ces derniers les plus grandes analogies, au double point de vue de sa structure et de son évolution : c'est le *cancer de l'œsophage*. L'œsophage est, en effet, recouvert d'une muqueuse ectodermique, et, lorsque le cancer vient à se développer sur elle, il semble présenter, par lui-même, une évolution toute particulière, sans tendance à la généralisation. Et n'étaient sa situation sur un point du tube digestif, et les troubles d'inanition qui en résultent bientôt, il présenterait une *évolution tout à fait lente.*

On connaît, d'autre part, les faits d'assez longue survie que donne la gastrostomie, chez de pareils malades, lorsqu'elle est faite au début ; si bien que, dans ces cas, on est quelquefois amené à penser qu'il ne s'agissait point d'un cancer de l'œsophage. A noter, aussi, qu'il s'agit d'une tumeur qui a très peu de tendance à la généralisation. On voit exceptionnellement des malades succomber à la généralisation, dans le cancer de l'œsophage, même après la gastrostomie qui seule pourrait la permettre.

Toutes ces raisons combinées faisaient espérer que le radium devait être efficace dans cette forme toute particulière du cancer.

Nous avons, systématiquement, traité par le radium, actuellement, 60 *malades*, en *choisissant nos cas parmi* les plus favorables et l'appliquant seulement chez ceux où on pouvait encore introduire la sonde porte-radium, au sein même du rétrécissement cancéreux. L'*effet palliatif* a été évident chez tous ; dans un certain nombre, environ un tiers des cas, l'amélioration était telle que l'alimentation est redevenue normale, pendant de longs mois. D'autres, enfin, évidemment le plus petit nombre, présentent, actuellement, toutes les apparences de la guérison.

Existe-il des formes d'épithélioma œsophagien qui sont plus nettement influencées par le radium ? Il nous a paru que, dans les formes peu bourgeonnantes, plutôt infiltrantes, dans le squirrhe, par exemple, l'action semble plus efficace et plus durable. Toutefois, il n'y a pas de règle précise, car dans le cas qui fait l'objet de cette communication, la forme semblait assez envahissante, indurée, faisant corps avec une grande partie de la paroi œsophagienne.

Au point de vue technique nous nous sommes servi pour porter le radium au sein même du rétrécissement de l'œsophage du dispositif suivant : le tube porte-radium est glissé dans une sonde en gomme, la situation exacte du rétrécissement a été bien repairée ; il est facile dès lors de faire que le radium soit exactement au niveau de la tumeur. Dans les cas difficiles nous préférons pour placer le radium le contrôle œsophagoscopique, mais généralement il est facile de faire la chose directement sous le doigt.

Comme quantité de radium 0,05 à 0,10 gr. de radium à activité de 2,000,000 sont indispensables, sinon l'action est à peu près nulle ou de courte durée.

Les séances d'application doivent être les plus longues possible. Généralement les malades supportent 4 ou 5 heures facilement ; un minimum total de 50 heures d'application est nécessaire. Il semble donc et d'après les quelques cas typiques que nous avons cité plus haut que l'on ait dans le radium dans un certain nombre de cas un remède efficace contre cette affection particulièrement désespérante qu'est le *cancer de l'œsophage.*

Enfin comme fait très rare et qui constitue une véritable trouvaille œsophagoscopique nous désirerons citer celui d'un *angiome du cardia* pris pour un cancer chez un homme âgé, et cette tumeur a guéri complètement tout comme les angiomes de la peau par quelques applications locales de radium.

Un confrère du Centre vient le 23 décembre 1911 nous demander de faire un examen œsophagoscopique, amené par le Dr Amblard, pour des phénomènes de dysphagie, qui ont présenté un caractère tout à fait particulier. Il y a un peu plus d'un an le malade s'est aperçu, en mangeant des aliments mal mastiqués, en particulier du pain, de la viande, qu'il éprouvait une certaine difficulté à les avaler. Les phénomènes dysphagiques ont été en augmentant progressivement, pendant trois mois, puis, brusquement, tout a cessé et est rentré dans l'ordre ; si bien que, pendant les 4 mois qui ont suivi, le malade ne ressentait plus aucune espèce de trouble dysphagique.

Reprise de symptômes analogues, au mois de septembre dernier, après une période de surmenage ; ils ont été constants, depuis trois mois, avec caractère d'augmentation progressive ; si bien qu'actuellement le malade ne peut plus avaler que des aliments liquides et se nourrit de bouillon, de lait. Il a remarqué toutefois que le lait passait beaucoup plus difficilement que les autres aliments liquides, et donnait des aigreurs. Il y a un amaigrissement de 25 livres. Pas de vomissements, quelques régurgitations, pas d'hématémèse ; facies très maigri, les lèvres pâles sont décolorées.

Examen œsophagoscopique, le 23, sous cocaïne, en position couchée : introduction du tube facile. Œsophagite dans les $\frac{2}{3}$ supérieurs de l'œsophage ; la muqueuse est rouge et enflammée. Dilatation moyenne de $\frac{1}{3}$ inférieur de l'œsophage (1 litre), pas de débris alimentaires, un peu de mucus dans la poche ; cardia fermé par contracture simple, rien de particulier à première vue. Après cocaïnisation, il s'entr'ouvre assez facilement et, en le déplissant, nous voyons sur la lèvre postérieure de l'œsophage une sorte de tumeur brunâtre, de forme contournée, ressemblant à une sangsue gorgée de sang. En appuyant avec notre tube sur la partie antérieure du cardia, nous arrivons à la limite antérieure de cette tumeur,

qui mesure 4 cm. de longueur environ et qui est grosse à peu près comme le pouce. Pas d'ulcération sur le reste du cardia ; il s'agit vraisemblablement soit d'une volumineuse varice, soit d'une sorte d'angiome du cardia. Nous admettrions plutôt ce dernier diagnostic.

Cinq applications de radium sont successivement faites : applications de 3 à 5 cg. et de 3 à 4 heures de durée. La perméabilité œsophagienne est toutefois plus accrue après ces applications et le malade reprend une alimentation à peu près normale.

Durant les applications, il y a pas mal de salivation.

Nouvel examen œsophagoscopique, fait le 12 février, nous montre qu'il existe encore un peu d'angiome, au niveau du cardia, mais il semble avoir beaucoup diminué. Il y a une sorte de place blanc-grisâtre, comme cicatricielle, à la partie tout à fait inférieure de l'angiome. La perméabilité du cardia est à peu près normale, puisque le tube de 12 mm. pénètre très facilement dans l'estomac. Il semble que ce soit bien aux applications de radium que l'on doive attribuer cette notoire amélioration.

Nous refaisons de nouveau deux applications, les 20 et 23 février : applications de un centigramme et demi.

2° *Trachée et Bronches.* — Au point de vue de la trachée et des bronches, l'endoscopie nous a permis certaines particularités diagnostiques et thérapeutiques.

Dans un petit nombre de cas, il s'agissait d'une malformation de la paroi trachéale ; c'est ainsi que chez une malade adressée par le Dr Hirtz, atteinte d'accès de suffocation, de dyspnée depuis son enfance, la trachéoscopie permit de reconnaître comme cause de ces troubles *une double valvule intratrachéale* siégeant au niveau des premiers anneaux de la trachée. Rien ne fut plus facile que de sectionner la valvule avec la lame de l'œsophagotome et de compléter la dilatation avec les bougies olivaires.

Dans plusieurs autres cas il s'agissait d'*hernie de la paroi postérieure de la trachée* dans la lumière de ce conduit. Chez une malade l'obstruction était telle que la lumière de l'œsophage était à peu près complètement fermée au moment de l'effort.

Rien n'est plus difficile que le diagnostic du *cancer de l'appareil broncho-pulmonaire*, et ce n'est souvent qu'une trouvaille d'autopsie.

Jusque dans ces derniers temps c'était presque exclusivement sur l'examen méthodique des produits d'expectoration, joint aux signes cliniques, qu'on pouvait établir le diagnostic ; mais les résultats de ce mode d'exploration sont souvent négatifs. L'expectoration, en particulier, est souvent nulle et l'auscultation ne donne point souvent de renseignements précis.

C'est dans les cas de cette nature que l'examen bronchoscopique prend une grande valeur.

L'histoire de deux malades, adressés par le Dr Renon et que nous avons eu à examiner en 1910–11, dira mieux qu'une description, toute la précision qu'a apporté au diagnostic l'examen trachéo-bronchoscopique.

Un homme de 54 ans se plaint, depuis 7 mois, d'une dyspnée progressive, croissante ; il expectore depuis quelques semaines des crachats

sanguinolents ; il est pâle, amaigri. L'expectoration attire immédiatement l'attention du côté des poumons. De ce côté les modes d'explorations donnent des résultats tout à fait déconcertants. On constate simplement, dans le côté gauche de la poitrine, une diminution des vibrations thoraciques et une abolition complète du murmure vésiculaire, sauf à la portion toute supérieure du poumon, qui respirait encore d'une façon assez nette.

De fait, deux diagnostics se présentaient aussitôt à l'esprit ; c'était soit *une tumeur intra-pulmonaire*, soit bien plutôt une *compression de la bronche gauche* par une *tumeur du médiastin*. Mais, contre cette dernière, il n'y avait ni souffle interscapulaire révélateur d'une trachéo-bronchite volumineuse, ni aucun signe d'anévrysme de la crosse aortique. L'examen bactériologique et histologique des crachats ne donnait aucun résultat. La radiographie, faite par le Dr Jaugeas, révèle une opacité complète de l'hémi-thorax gauche, mais l'examen oblique montra l'intégrité absolue de l'espace médian. Il s'agissait donc *d'une tumeur intra-pulmonaire*. La *syphilis* fut éliminée, par ce fait que le traitement mercuriel, soigneusement fait, dans le service du Dr Renon, ne donna aucun résultat.

L'examen bronchoscopique montre que la bronche gauche est obstruée par une masse qui la remplit toute, en respectant néanmoins la partie supérieure. Cette masse est rouge, saigne au moindre contact du porte-coton et présente tous les caractères d'un néoplasme. Cependant, pour contrôler de façon exacte ce diagnostic objectif, nous en prenons plusieurs fragments, pour un examen biopsique. Le fragment examiné au microscope montre qu'il s'agit nettement d'un épithélioma bronchique.

Dans un autre cas, il s'agissait également, chez une malade qui nous fut adressée en juin 1911, pour de la dyspnée, avec toux coqueluchoïde, amaigrissement progressif, mauvais faciès, diminution du murmure vésiculaire du côté gauche. Il s'agissait d'un cancer de l'éperon bronchique, avec propagation à l'intérieur de la bronche gauche. Le tube, introduit dans cette bronche, montre que celle-ci est en partie obstruée par une masse bourgeonnante, macérée, qui fait corps avec la paroi interne.

Ici, également, l'examen biopsique montre qu'il s'agit d'un épithélioma, formé par des masses de cellules fusiformes imbriquées, qui est l'aspect caractéristique du cancer bronchique.

Dans ces deux cas, grâce au diagnostic exactement établi, le pronostic put être fixé de façon définitive et, en outre, il fut possible d'agir localement par des injections de sulfate de radium.

Le premier malade reçut une série d'injections intra-veineuses. On sait que MM. Dominici et Faure-Beaulieu ont démontré que ces sels insolubles, injectés dans les veines, s'arrêtent au niveau des capillaires pulmonaires et y séjournent pendant plus d'un an. La médication a produit, dans le premier cas, une légère diminution de la congestion.[1]

Dans le second, nous avons fait ces injections directement par la voie intratrachéale, et l'action nous a paru beaucoup plus manifeste, puisque la malade, soignée de cette façon, n'a plus eu aucun accès de suffocation, a paru beaucoup soulagée pendant plus de trois mois.

Par la suite de circonstances indépendantes de notre volonté, il ne nous a pas été donné de la suivre, et de recommencer un autre traitement, mais qui, à notre sens, aurait été tout à fait efficace.

[1] Voir *Presse médicale*, 28 mai 1910.

On sait, d'autre part, que dans un cas de cancer du lobe supérieur droit, chez un homme de 31 ans, Lénaerts était intervenu avec succès. Le patient, opéré depuis plus de 5 ans, n'a pas eu de récidive et a repris son travail. Peut-être y aurait-il là, dans cette intervention, évidemment grave, une chance de salut.

La *thérapeutique par les injections intra-bronchiques*, et que nous avons exposée dans plusieurs communications et travaux ci-joints, en particulier à la Société médicale des Hôpitaux,[1] repose essentiellement sur le principe suivant :

Il est possible d'injecter, sans inconvénient ni douleur, 15 à 20 centimètres cubes d'huile antiseptique dans l'une ou l'autre bronche, ou successivement dans les deux bronches, pour agir localement sur un état pulmonaire déterminé.

La technique en est facile, elle nécessite simplement une seringue spéciale de 20 cm³, munie d'une longue canule recourbée et un miroir laryngé. Après anesthésie du larynx à la cocaïne, et anesthésie de la partie supérieure de la trachée, par instillation de quelques gouttes d'alipine, il est possible de franchir la glotte, avec la portion courbée de la canule et d'injecter directement, soit dans la trachée, soit exclusivement dans l'une ou l'autre bronche, le liquide actif. L'injection, quoique abondante (20 à 25 cm³), est *très bien supportée*, réveillant à peine une quinte de toux, inconsciente, du reste, dès que le malade est entraîné. La totalité du liquide injecté reste donc dans l'arbre aérien. D'expériences très précises sur les chiens, entreprises avec le Dr Stodel à la Sorbonne, dont nous avons publié les résultats dans un article récent (*Presse médicale*, 18 septembre 1912), il résulte qu'il ne faut que quelques minutes à un liquide colorant, injecté dans la bronche, pour imbiber tout le parenchyme pulmonaire.

Les poumons des chiens sacrifiés un quart d'heure après l'injection dans la trachée d'huile colorée au bleu de méthylène, présentaient à leur surface de larges plaques bleues, séparées par des espaces sains. Tout le parenchyme pulmonaire, à la section, était imbibé par le produit colorant. Ces expériences démontrent donc qu'il est possible, avec cette méthode, d'injecter l'arbre pulmonaire et surtout d'imprégner tout le parenchyme pulmonaire. On réalise, véritablement, en pratiquant ces injections, un *véritable pansement intra-pulmonaire* et, dans les cas d'infection, on pourra véritablement porter l'antiseptique au sein même de la lésion. L'expérimentation, très méticuleuse, à laquelle nous nous sommes attaché, a montré, en plus, le contact prolongé entre l'agent bactéricide et le tissu pulmonaire.

La voie d'administration laryngo-trachéo-bronchique est donc tout à fait rationnelle pour agir sur une lésion pulmonaire. Comme médicament actif, nous avons employé successivement différents agents ; actuellement, nous nous en tenons exclusivement aux solutions huileuses

[1] Voir *Gazette des Hôpitaux*, mai 1910, et *Bulletin de la Société méd. des Hôpitaux*, 6 mai 1910, 24 novembre 1911 et 7 mars 1913.

goménolées, au 10° et au 20°, et, dans les cas graves, nous y joignons, soit moitié de gaïacol au 20°, soit quelques gouttes d'iodoforme pulvérulent. Pour anesthésier la trachée, nous employons actuellement l'alipine, beaucoup moins toxique que la cocaïne.

Depuis 1908, date de nos premières recherches et de la publication de notre premier cas de guérison de gangrène pulmonaire, par cette thérapeutique, nous avons appliqué cette thérapeutique à toute une série de malades, envoyés à nous pour des affections broncho-pulmonaires, à forme suppurative.

Les affections trachéales ont été rapidement modifiées par ces injections, toutes sans exception. Nous avons pu guérir ainsi des *trachéites anciennes rebelles, purulentes*, toute une série de cas *d'asthme trachéo-bronchique*, et une affection, qui jusqu'à présent était rebelle à tous les traitements, l'ozène trachéal.

Pour la guérison de l'*ozène trachéal*, nous avons employé des solutions de nitrate d'argent faibles, injectées directement dans la trachée et dans les bronches ; injections avec des solutions au millième.

Pour ce qui est des affections pulmonaires, c'est la *gangrène pulmonaire*, certainement, qui semble avoir le plus bénéficié de cette thérapeutique. Il semble que cette injection agit surtout dans les affections aiguës. Dans la tuberculose, les résultats sont loin d'être aussi bons, sans doute parce qu'il y a des lésions de sclérose de protection, autour des foyers.

C'est surtout le traitement de la gangrène du poumon que nous avons en vue dans ce travail ; nous avons eu à soigner par cette thérapeutique 7 cas de gangrène pulmonaire, à forme grave. Dans les 7 cas, la guérison est survenue complète. Dans tous, l'histoire clinique était à peu près analogue.

Le premier était un malade, que nous avons vu avec le Dr Walther, à la Maison de Santé de la rue Blomet, et qui avait fait un foyer de gangrène pulmonaire du poumon gauche, très étendu, avec fièvre élevée, prostration, température à 40°, vomique purulente, sphacélique, abondante. On nous avait demandé de le désinfecter, par cette méthode, pour pouvoir l'opérer ; et il a guéri complètement par cette technique, à notre grande surprise du reste.[1]

Le deuxième malade, âgé de 26 ans, a été soigné dans le service du professeur Chauffard. Il présentait une gangrène peu fébrile, mais très tenace, puisqu'elle datait de plusieurs semaines, sans que rien ait pu la modifier. Dix injections d'huile goménolée, de 15 à 20 cm³, ont fait disparaître tout signe de gangrène.

Dans le troisième cas, qui, avec le précédent, a fait l'objet de notre deuxième communication à la Société médicale des Hôpitaux,[1] il s'agissait d'un malade envoyé par le Dr Rabant, de Paris, et radiographié par le Dr Rist, qui présentait un foyer de gangrène pulmonaire très étendu, du côté droit, expectoration abondante, température variant entre 39° et 39,5°, depuis quinze jours.

[1] Voir *Bulletin de la Société médicale des Hôpitaux*, 7 juillet 1911.

Ce cas a été assez rebelle aux injections, à cause de la diffusion du foyer ; toutefois, la température est tombée définitivement, l'expectoration a également très rapidement disparu. La fétidité a persisté pendant assez longtemps, et n'a cédé qu'au bout de 18 injections.

Dans le quatrième cas, il s'agissait d'un malade, hospitalisé à Saint-Antoine, dans le service des Drs Decloux et Béclère, qui présentait un foyer de gangrène pulmonaire du côté gauche, avec fièvre de 38,5° à 39°, datant de quinze jours. La fétidité et tous les signes de suppuration ont disparu au bout de la onzième injection.

Notre cinquième cas[1] concernait une malade, âgée de 59 ans, soignée avec les Drs Stodel et Rénon. Elle présentait une gangrène pulmonaire ancienne, peu fébrile, développée sur un terrain tuberculeux. Il y avait du côté droit, vers le sommet, les signes nets de caverne pulmonaire. Les signes de suppuration et de fétidité ont disparu assez rapidement. La cicatrisation s'est faite complète pour le poumon droit, sans qu'il persiste aucun signe de caverne, et même les bacilles de Koch dont la recherche a été faite à plusieurs reprises par M. Huré, du laboratoire de la Sorbonne, ont complètement disparu, pour ne plus reparaître, depuis un an et demi que cette malade est véritablement guérie.

Dans le sixième cas, le moins grave de tous, il s'agissait d'un malade, que nous avons vu à la consultation de l'Hôtel-Dieu, qui présentait une expectoration muco-purulente, consécutive à une suppuration intra-bronchique ancienne, et apparue comme complication de celle-ci. Tous les signes ont disparu assez rapidement et progressivement, mais le malade a gardé une expectoration purulente, due à une bronchite antérieure.

Le septième cas, que nous avons soigné au mois de décembre 1912, avec le Dr Main, concernait un malade alcoolique qui présentait, au moment où nous l'avons vu, des signes de gangrène bilatérale. Il y avait une expectoration fétide très abondante, dont la quantité totale pouvait être évaluée à $\frac{3}{4}$ de litre par jour ; expectoration spumeuse, sanguinolente certains jours ; exhalant une horrible fétidité. La température était très élevée, et depuis 15 jours était aux environs de 39° ou 40°. La fétidité de l'expectoration a disparu la première, à la sixième injection, et la guérison complète a été obtenue en 12 injections. L'auscultation a démontré que tous les signes locaux ont disparu tout à fait rapidement. Malheureusement le malade, alcoolique invétéré, a succombé ultérieurement à d'autres accidents. Dans deux autres cas, soignés tout récemment, l'amélioration a été également facile à obtenir ; la température s'est abaissée rapidement et tout est redevenu bientôt normal. Toutefois les cas sont trop récents pour qu'on puisse prononcer le mot de guérison.

On possède donc là, dans ce mode d'administration médicamenteux, un moyen d'agir directement sur les bronches et le parenchyme pulmonaire, pouvant donner dans certaines affections particulières rebelles des résultats inespérés.

[1] Voir *Bulletin de la Société médicale des Hôpitaux*, 24 novembre 1911.

SECTION XV

RHINO-LARYNGOLOGY

INDEPENDENT PAPER

REMARKS UPON 54 CASES OF FOREIGN BODIES IN THE ŒSOPHAGUS, TRACHEA, AND BRONCHI

By Dr. ERNESTO BOTELLA, Madrid

My first observation of œsophagoscopy and tracheo-bronchoscopy dates from the year 1905, after my return from Vienna, where I was instructed in their technique by my distinguished friend, H. von Schrötter; since then, I have had occasion to observe 54 cases of foreign bodies in the œsophagus, trachea, and bronchi, besides very numerous cases of œsophageal stenosis and cancer, tracheal tumours, and stenoses of the trachea and bronchi, with which, however, I do not intend to deal in this paper.

The experience acquired by such plentiful statistics regarding foreign bodies, entitles me to state the following considerations:

It is of the highest interest to collect the fullest data regarding the nature, form, and dimensions of the foreign body; data which an examination by means of X-rays will very often not furnish, either because the urgency of the case will not allow of their application or because the foreign body is not visible on the fluoroscopic screen.

The importance of such data lies in the very direct relation that exists between the nature, form, and size of the foreign body and the symptoms to which they give rise, especially in the trachea and bronchi; thus, metals cause but little local reaction; bits of bone, fruit stones and seeds are very septic; whilst beans (French kidney) and peas are very dangerous, owing to the increment in size to which they are liable through moisture of the secretions; those provided with sharp points get stuck fast and produce bleeding, tubular ones cause hardly any symptoms of stenosis, whilst small bodies may change their site and change, consequently, their clinical aspect, &c.

The pain, tumefaction, fœtidity of breath—in a word, the œsophagitis and peri-œsophagitis, which occasionally accompany foreign bodies—I do not consider as contra-indications for œsophagoscopy, but rather as indications for performing the examination with great care. On the other hand, in children, I do not think it prudent to attempt performing upper tracheo-bronchoscopy with general anæsthesia, if the foreign body produce great respiratory difficulty.

When foreign bodies are present in the trachea or bronchi, we have observed the almost constant presence of a symptom, viz. a peculiar hoarse barking cough.[1] To this symptom we attribute a pathognomonic value, when, with the history of a foreign body having been inhaled, this peculiar cough is present from the moment of the accident, with a clear voice, though there may be no other thoracic symptoms.

The Technique

Instrumental. Although the bevelled tubes of Brünings are easier to introduce, we prefer, for œsophagoscopy, to use those of v. Schrötter, fitted with my conductor, which is rigid, bevelled, and does not project beyond the tube more than 1½ cm. The field in Brünings' tubes, unless an extension tube be used, is beset by difficulties on account of the prolapse of the œsophageal mucosa, which overhangs that part of the tube where the bevel begins. This difficulty and inconvenience is not observed in the trachea and bronchi, the light of which is not a virtual one, as in the case of œsophagus, but real.

With regard to an illuminator, we consider that of Kahler to be the most advantageous both for exploring and extracting, because it gives a wider field than that of Brünings. All manipulations, however, are far easier with the use of a Kirstein or Killian lamp.

Position. When resorting to general anæsthesia, in both œsophagoscopy and tracheo-bronchoscopy, we employ the position of Rose; and with local anæsthesia for tracheo-bronchoscopy, we make use of the so-called classic position; whilst for œsophagoscopy, we turn to our own position, i.e. Botella's position.

Anæsthesia. For œsophagoscopy we do not utilize any anæsthetic, either in infants or in adults, and only for the latter if they are very sensitive, we resort to a very moderate use of a 5 per cent. solution of cocaine. For tracheo-bronchoscopy, in adults we employ a 10 per cent. solution, and in children chloroform anæsthesia, *but without cocaine.*

The foreign bodies extracted from the œsophagus were bits of bone, artificial teeth, pins, coins, and fruit stones; those from the trachea and bronchi were of great variety: a slice of a cane, a pencil protector, an artificial pearl, a seed of a pine cone (*pignon*), a water-melon seed, a cherry-stone, beans, &c.

Summary of Observations and Results

Thirty-eight cases of foreign bodies in the œsophagus

These should be divided into two groups; in the first group, which embraces 13 cases, I employed the classic technic and tubes of 9 mm., and the results were: six were extracted by œsophagoscopy; in three, after various attempts to extract, I had to resort to œsophagotomy (in

[1] Toux aboyante.

one of these three cases a piece of denture with gold clasps was deeply fastened in the œsophagus); one was pushed into the stomach at the moment of introducing the tube, from which a rubber-sound projected far and served as conductor; another escaped whilst being caught by the forceps, and likewise slipped into the stomach; and in two other cases we had no confirmation of the presence of foreign bodies.

In the 25 cases of the second group, I employed my simplified technique, and tubes of 12 and 14 mm., introduced by my conductor, and the results were: one, œsophagotomy, because the foreign body (a piece of a bone) was firmly fixed and a peri-œsophageal abscess was present; one, on being loosened from its hold, slipped into the stomach; two, on making the examination, had already followed the same way; six, the presence of which could not be ascertained, though in some of these we were able to detect erosions of the mucosa; fifteen, in which the foreign bodies were extracted by œsophagoscopy.

Out of the 38 cases of foreign bodies in the œsophagus, only one death occurred. The patient in this case had a peri-œsophageal abscess; œsophagotomy was performed, and death from mediastinitis followed.

The number of foreign bodies extracted from the respiratory tract by direct means were 16; one in the larynx, two tracheal, and thirteen bronchial; of these last, two were at first tracheal, seven were located in the left bronchus, and six in the right. The results were as follows: A pin, located in the vestibule of the larynx, was extracted by direct laryngoscopy; two tracheal (beans), in infants of 10 and 12 months, threatened by suffocation, it was necessary to perform rapid tracheotomy, and then lower tracheoscopy; in one case out of the thirteen the presence of a foreign body could not be detected; one was expelled spontaneously (a piece of an acorn); one (the scale of a pine cone) in its reactionary stage, and without distinct localization, was explored by upper bronchoscopy without result—the operation was stopped, because respiratory syncope set in and the parents were not willing to have the exploration repeated; one (also the scale of a pine cone) had a large abscess in the right lung, which was partly emptied during upper bronchoscopy; it was impossible to discover the foreign body in this sewer—the parents objected to a repetition of the exploration; one, a pin in the base of the left lung—first an upper bronchoscopy was made, and then lower bronchoscopy—explorations having been made during several attendances, up to ramifications of second and third order, without having found anything, though use was made of a strong electro-magnet; three, which were respectively a seed of a pine cone (*pignon*), a water-melon seed, and a bean, in a state of comparative tolerance, were extracted with great facility by upper bronchoscopy; one, a very interesting case, because we had to deal with a crystal pearl which got fast in the trachea, remaining there for five months without causing any great mischief, until one day it got broken and the pieces fell into the right bronchus, giving rise to intense reaction; on beginning with chloroform anæsthesia, an attack

of asphyxia set in which demanded rapid tracheotomy; thereupon lower bronchoscopy was performed and the pieces of this foreign body were extracted; one, which was already tracheotomized and which had been explored by lower bronchoscopy without success, on account, perhaps, of the difficulty of getting hold with fenestrated forceps of the foreign body as it fitted perfectly into the right bronchus, was extracted with great facility by lower bronchoscopy with Brünings' forceps for hollow bodies ('Hohlkörperzange'); one, in which the slice of a cane, without being localized, and presenting no other symptom than that of hoarse cough, was extracted, at the third visit, by lower bronchoscopy; lastly, in two cases (a monkey-nut and a cherry-stone), upon performing an exploration by upper bronchoscopy, an attack of asphyxia supervened, which compelled us to perform tracheotomy, and immediately after regular respiration was restored the foreign bodies were extracted by lower bronchoscopy.

In these 16 cases, two deaths occurred, but not in consequence of intervention which was not repeated. Death was caused by the presence of the foreign body exciting violent pulmonary reaction, which in one case assumed an acute form, the girl dying of septic pneumonia, and in the other followed a chronic course, the boy dying after some time of pulmonary gangrene.

DISCUSSION

Professor KUBO (Japan). Bei dieser Gelegenheit möchte ich Ihnen die Exemplare der Fremdkörper in den Speise- und Luftwegen zeigen, die ich in Japan behandelt habe. Die Arten der Fremdkörper sind bei uns etwas anders als in Europa, da die Gewohnheiten und Nahrungsmaterialien verschieden sind. Wie die Abbildungen (gezeigt) und Exemplare (gezeigt) Ihnen zeigen, handelt es sich meistens um Münzen, Fischgräten, Zahnprothesen, Spielzeuge u.s.w. Bis jetzt wurden 127 Oesophagusfremdkörperfälle in meiner Klinik ösophagoskopisch behandelt, in denen die Fremdkörper in 84 Fällen positiv konstatiert und extrahiert wurden. Nur in einem Falle (grosse Zahnprothese) war Oesophagotomie notwendig.

Was die Fremdkörper in den Luftwegen anbetrifft, haben ich und meine Assistenten 20 Fälle behandelt. Von diesen sind 14 Bronchialfremdkörper, die meist durch Bronchoscopia sup. (nur einmal Bronchosc. inf.) entfernt wurden. Davon gingen 2 zu Grunde, welche von meinen Assistenten behandelt wurden. Die Fremdkörper waren Bohnen (4 mal), Obstkerne (3 mal), Pfeifen (2 mal), Pflanzensamen (2 mal), Nagel (1 mal), Trachealkanüle (1 mal) und Hummerschale (1 mal).

SECTION XV

RHINO-LARYNGOLOGY

INDEPENDENT PAPER

RADIUM IN THE TREATMENT OF MULTIPLE PAPILLOMATA OF THE LARYNX

By THOMAS J. HARRIS, A.M., M.D., New York, N.Y.

The unsatisfactory results of surgical treatment 93
The total number of cases treated with radium, small; Polyák's cases. A report of six cases treated by this method in America . . 93
The harmlessness of its application. Method of introduction . . 96
Proper radio-activity and time of application important in securing a cure and preventing recurrence 96

At the Third International Laryngo-Rhinological Congress, Polyák of Budapest read a paper on the subject of the 'Treatment of Multiple Papillomas of the Larynx by means of Radium', in which he reported three cases treated by this method, and referred to three other cases treated by Mazzocchi. In the discussion which followed, Killian referred to a case cured by the same method, which suffered a relapse and was later cured by the Röentgen rays.

The treatment of laryngeal papilloma, whether by the internal or the external route, has up to the present time been unsatisfactory in the extreme. Because of the large number of recurrences and of the more or less permanent impairment of the voice, as a result of the repeated operations often necessary to effect a cure, any procedure which will avoid operation and reduce the liability of recurrence cannot be too earnestly sought after, and its merits too often brought before the profession. It is because I confidently believe that in radium we have such a remedy, I desire to bring the subject again before the Laryngological Section of the present Congress.

A great deal has been written upon the use of this wonderful agent in other conditions of the upper respiratory tract, but, as far as we know, Ferreri, at the Second Laryngo-Rhinological Congress in Vienna, was the first to speak about its use in the larynx. The total number of cases in the literature where radium has been used for papilloma of the larynx, is a small one. The reason for this can be easily understood when the high cost of the metal is borne in mind, as well as the location of the disease and the consequent difficulty of application. We desire, therefore, to report a number of cases which have been treated during the last few

years in New York. Before doing so, however, we would briefly refer to the cases of Polyák.

The first case was a woman of 26, who had been operated upon on several occasions for recurring papilloma. 15 mg. of pure radium bromide was applied through the mouth in 16 sittings of 17 hours and 15 minutes, the radium being left in position each time for from 10 to 45 minutes. The papilloma entirely disappeared and the vocal cords became white and smooth. The voice returned full and strong. This condition lasted for a year when there was a recurrence. At the time of Polyák's communication he was about to reapply the radium.

Case II was a child of nine who had had a laryngo-fissure performed for multiple papillomas. There were frequent recurrences, the tumours returning in large masses. A reduced strength of radium was applied in 15 sittings, through the external opening, for 21 hours altogether. The larynx became entirely free. Four months later a small growth appeared in the anterior commissure. $8\frac{1}{2}$ hours of applications in 5 sittings cleared it up. From then up to the time of the meeting of the Congress there had been no recurrence.

Case III was a man of 35 who had been treated for extensive papillomas of both vocal cords for a year and a half. Radium was applied in 15 sittings for 20 hours and 54 minutes. The papillomas disappeared entirely, the only remaining evidence of the former condition was a thickening of the right vocal cord. No recurrence took place.

Polyák calls attention to the absence of all reaction in all of his cases. While in two of the three cases there was a recurrence, he insists that a reapplication of the radium has the same happy result and is far simpler than a second operation.

The three cases of Mazzocchi treated by radium were all cured, and at the time of his personal communication to Polyák had had no recurrence. The seventh case was that of Killian, previously referred to.

In New York we have had six cases of papilloma treated by radium belonging either to Dr. Robert Abbe or Dr. Wolff Freudenthal. The first case is one of Dr. Abbe's, a Mrs. P., age 30. This case will be reported at length later by Dr. Abbe, who has kindly supplied me with the following notes :

He first saw the case in February 1898, when she had for five years been suffering from aphonia and attacks of choking cough. During this time there had been removal of papillomata on three occasions. A laryngotomy was performed after a preliminary tracheotomy, the papillomata entirely covering both cords were removed, and the cords carefully cauterized. In the next nine years there were continual recurrences requiring endolaryngeal operation. In February 1907, 20 mg. of radium was applied. The growth, with the exception of a minute remnant in the anterior commissure, disappeared. On account of the irritability of the throat, the radium could be applied for only two or three minutes at a time, a total of 20 minutes. The following January she reported that she could talk loudly again. Two years later a small bit of growth could be seen on the right cord. At present she talks loudly and plainly. The attending laryngologist has once snipped off a little bit of growth, but he regards the case as practically cured. This, as far as we know, is the first case of papilloma of the larynx treated by radium.

Case II was a patient of Dr. William Ledlie Culbert's. This patient, a woman advanced in years, had had her larynx cleared out every six months for over 47 years, for papillomata. In February 1908, she was treated by radium. The growths were non-malignant but obstructive. A large part rapidly disappeared by the radium application. No further operation was required, and Dr. Culbert, in a recent communication, states that up to the time of her death, three years later, there was no recurrence of the growth.

Case III is a remarkable case, also a patient of Dr. Culbert's, treated by Dr. Robert Abbe. This case has been already reported (*New York Medical Record*, April 13, 1912). The patient was a girl of 17 who became hoarse in 1910. She was operated upon for fibroma of the left vocal cord by Dr. Culbert in September 1910. There was a rapid recurrence of the growth, which this time proved to be papilloma. A second operation was followed by recurrence. June 1911, two years ago, the voice was entirely gone and the respiration was obstructed. On the 14th of that month a tracheotomy under ether was performed. 100 mg. of radium bromide in a small, smooth capsule was drawn into the larynx and allowed to remain 30 minutes. Three months later no trace of the growth was to be seen. Six months later the condition was unaltered. At the present time, two years after the operation, the cords appear perfectly healthy, no disease, no scarring, perfect voice.

Case IV was a patient of Dr. Wolff Freudenthal's. It has not yet been published, but he has been kind enough to supply me with the notes. The patient was a man of 34 who for several months had been hoarse. The growth was discrete and situated on the anterior commissure and the left vocal cord. Radium was applied in 15 sittings of 9 hours. The mass entirely disappeared and the voice became, Dr. Freudenthal reports, as good as ever.

Case V was a case also of Dr. Freudenthal's, a man of 40, whose voice had been husky for ten years. Two months previous to being seen by Dr. Freudenthal, the voice became particularly bad. Examination showed a mass on the anterior portion of the left vocal cord, with the entire larynx in a state of inflammation. Radium was applied altogether for 14 hours, when the neoplasm disappeared.

Case VI, Mrs. S., age 43, presented herself at the Manhattan Eye, Ear, Nose, and Throat Hospital in the service of Dr. Lewis A. Coffin, and by him was assigned to our care. She complained of complete aphonia which had existed for three years. Examination showed a cauliflower growth covering both vocal cords, the arytenoid cartilages, the anterior commissure, and the posterior wall. A portion of the growth was removed and submitted to the laboratory for examination. The pathologist reported it a simple papilloma. At the suggestion of Dr. Coffin, and by the courtesy of Dr. Abbe, 100 mg. of radium bromide, 1,800,000 radio-activity, was applied for 35 minutes in March, 1913. At the end of eight weeks the papillomas had, with the exception of a number of small tabs, entirely disappeared. At that time radium was applied for 15 minutes. When seen upon May 29, the larynx appeared entirely clear with the slight exception of a small mass on the right arytenoid, and the voice was strong and pure.

In addition to this series of cases, Dr. Abbe has at present a girl under observation who has been treated by radium with distinct improvement to the voice. Examination of the larynx is almost impossible, so that it cannot be said how completely the growth has disappeared. The voice,

however, is decidedly better. We also recently treated a child of six, previously operated upon, with the same strength of radium. In this case the child's throat was so tolerant that the radium could be applied directly through the mouth, but on account of the size of the carrier, its introduction caused obstruction to breathing, so it could be left in only for a minute or two at a time, a total of 13 minutes. It is too early to report upon the outcome of the case.

This report represents 13 cases treated by radium. All authorities are agreed upon the entire freedom from reaction or other unpleasant results attending its application. Its mode of action is mysterious. Without any apparent slough, the growth gradually disappears. There is no immediate change, possibly a little alteration in the colour of the tumour. The transformation can be likened to the work of Nature in the development of the flowers. With a single exception, all the cases treated by us in New York permitted the introduction of the radium per vias naturales, after proper cocainization. A celluloid retainer firmly attached to a copper handle was the method employed. The great advantage of such a procedure over any cutting method from the outside is self-evident. The only criticism which has been brought against its use is that of Killian, who, in the Berlin Congress, dwelt upon the recurrence wont to follow. This is undoubtedly true in the earlier cases reported. It will be noted, however, that in the cases treated in New York there has been virtually no recurrence in any of them. In two of them the observation has extended over a period of from two to three years. This is to be undoubtedly ascribed to the greater radio-activity of the radium employed. Instead of the weaker strength of 15 to 20 mg., 100 mg. have been used in the cases treated by Abbe, Culbert, and myself.

Experience would seem to show that the secret of success in the use of radium both in this situation and elsewhere, rests upon the knowledge of the strength of the radium to be employed and the length of the application. Too few cases have yet been treated to definitely decide these facts, but our results in New York satisfy us that the use of such a strength reduces the necessity of frequent and prolonged applications, which are both troublesome and difficult in many instances, and in all probability are attended with more or less irritative effects, possibly sufficient to cause a recurrence.

DISCUSSION

Dr. W. Freudenthal (New York). During the last few years I have had opportunities of observing a comparatively large number of malignant neoplasms. It was at the suggestion of Dr. Harris, at a meeting of the New York Academy of Medicine, that the laryngologists of that city and elsewhere referred such cases to me. A certain percentage of these cases have been cured, as reported on several previous occasions. The two cases of benign neoplasms referred to in Dr. Harris's paper have been cured. Dr. Harris has the details, which I hope he will fully relate in his paper.

SECTION XV

RHINOLOGY AND LARYNGOLOGY

INDEPENDENT PAPER

EXPERIMENTELLE UNTERSUCHUNGEN ZUR MECHANIK DER INTRACRANIELLEN UND CEREBRALEN KOMPLIKATIONEN DER STIRNHÖHLENENTZÜNDUNGEN

Von Dr. HUGO ZWILLINGER, Budapest

Im Anschlusse an meine Untersuchungen über ' Die Lymphbahnen des oberen Nasenabschnittes und deren Beziehungen zu den perimeningealen Lymphbahnen ' hatten sich die Fragen ergeben, ob es Verbindungen der Lymphbahnen der Stirnhöhlenschleimhaut mit den genannten Räumen auch beim Menschen gibt, und welcher Art dieselben wären, welche Rolle denselben in der Mechanik der von der erkrankten Stirnhöhle verursachten oder postoperativen intracraniellen und cerebralen Komplikationen zukommt. Der experimentellen Lösung dieser Fragen nähertretend, hatten sich die Schwierigkeiten der Beschaffung entsprechenden Materiales in den Weg gestellt; meine ausgeführten Injektionsversuche hatten es auch mir klar dargetan, dass dieselben an jugendlichen Individuen mit reichlichem und wegsamem Lymphgefässnetze bessere Aussicht auf Erfolg darbieten, ein abschliessendes Urteil jedoch nur durch am Erwachsenen ausgeführten Untersuchungen zu gewinnen sein würde. Die Lehre über die Entwickelung und das Wachstum der Nebenhöhlen der Nase hat uns bezüglich der Stirnhöhle neue Erkenntniss gebracht, die früher divergierenden Angaben sind der Erkenntniss gewichen, dass die Entwicklung der Stirnhöhle am Ende des ersten und Anfangs des zweiten Lebensjahres beginnt, die Untersuchungen hauptsächlich Haikes und Onodis u. A., welch letzterer in tabellarischer Uebersicht genaue Masse der Stirnhöhle angibt, belehren uns darüber, wie das für genannte Untersuchungen verwendbare Material beschaffen sein müsste. Das Bestreben, frisches, unserem Zwecke entsprechendes Material zu erhalten, erklärt die lange Dauer und die Spärlichkeit der Versuche.

Die Methode, welche wir bei unseren Untersuchungen ausschliesslich am Menschen ausgeführt befolgten, ist die Methode der indirekten Injektion, der Injektion von den perimeningealen Räumen, hauptsächlich vom Subduralraume aus, und bestand darin, dass am frischen Präparate, welches in bekannterweise in erhöhte Temperatur gebracht wurde, die

entsprechend hergestellte Karmingelatinlösung in die Blutgefässe eingebracht wurde, um vorerst eine Injektion derselben zu erhalten, oder es wurde in anderen Fällen ohne erst die Blutgefässe zu füllen, gleich die Gerota'sche Preussischblau-Terpentine-Aetherlösung in eine der perimeningealen Höhlen injiziert, auf die Weise, dass früher ein grösseres Stück des Knochens in der parietalen Region entfernt wurde. In den ersten Fällen hatten wir bei der Entfernung des Knochens mit Meissel und Hammer, wie es sich nachher bei der Injektion herausstellte, die Dura verletzt, so dass in den späteren Fällen, ein kleineres Stück des Knochens entfernt, eine kleinere Oeffnung gemacht wurde, um der Verletzung der Dura zu entgehen. Es wurde hierauf die mit Schlauch und Glastrichter verbundene Pravaznadel, später fein ausgezogene Glaskanüle bei Aufheben der Dura mit fein geriefter Pincette in die harte Hirnhaut vorsichtig eingestochen, die Kanülenspitze drang so in den Subduralraum, bei langsamen Heben des Trichters sank die Flüssigkeit durch eigene Schwere in den Subdural- oder in den Subarachnoidealraum. Man sah die Dura sich langsam emporheben und sich blähen, indem nun der Trichter bis über ein und einen halben Meter hoch vorsichtig gehoben wurde, drang die Flüssigkeit unter entsprechendem Drucke bei mehrfacher Unterbrechung in den Subduralraum ein. Bei der Injektion in den Subarachnoidealraum wurde die Dura in der Gegend der Fossa Sylvii gespalten, die Arachnoidea mit feiner Pincette vorsichtig in die Höhe gehoben, die conische Glaskanüle dann eingestochen. Wenn keine Verletzung stattgefunden hatte, der Druck kein zu hoher war, erfolgte keine Extravasation der Injektionsflüssigkeit. Es wurde hierauf das Präparat in der Weise verkleinert, dass Nasenhöhle und Nebenhöhlen in einem Stücke belassen wurden, das ganze dann in 5 prozentige alkoholische Formalinlösung gebracht; diese Fixation hat den Vorteil, dass sowohl die Karmingelatine als auch die Gerotamasse rasch präzipitiert werden. Nach ein bis zwei Tagen wurde dann das Präparat in der Mitte oder auch seitlich geteilt, behufs Untersuchung der Schleimhaut der Stirnhöhle dieselbe vom Knochen losgelöst, in grösseren oder kleineren Lappen entfernt, welche dann in Kanadabalsam eingelegt, untersucht werden konnten. In den Fällen, in welchen der Knochen untersucht werden sollte, wurde in der Weise verfahren, dass derselbe erst entkalkt, das Präparat in Alkohol gelegt und Schnitte verfertigt wurden, welche nach entsprechendem Verfahren eingeschlossen und untersucht wurden. Die Anfertigung mikroskopischer Präparate war deshalb notwendig, weil das Gelingen der Injektion sich mit freiem Auge oder geringer Vergrösserung nur selten beurteilen lässt, und nur die mikroskopische Untersuchung mit Sicherheit das Gelingen der Injektion erkennen lässt.

Eigene Versuchsreihe

Cap. I. Durchspülung der Gefässe des Kopfes mit sterilem Wasser, nachher Injektion von der 1. A. Carotis aus, mit Abklammerung aller sichtbaren Gefässmündungen. Ausfluss von Karmingelatine aus den Rückenmarksgefässen. Trepanation der Seitenwandbeine, es wird ein

über fünfkronenstückgrosser Teil des Knochens entfernt, die Dura mit Hilfe einer Pincette aufgehoben und Gerotas Masse mittelst fein ausgezogener Glaskanüle in der Gegend der Fossa Sylvii in den Subduralraum injiziert. Die Injektion geschah mit Hilfe von Glastrichter und Schlauch, aus der Höhe von 1½ Meter. Man sah in der Umgebung der Kanüle den Raum sich füllen, die Lösung schien bläulich durch als sich der Trichter langsam entleerte, spannte sich die Dura immer mehr und trat ein wenig hervor. Keine Extravasation. Hierauf wurde die Kanüle zurückgezogen, das Präparat für 48 Stunden zum Zwecke des besseren Eindringens der Masse konserviert. Nach dieser Zeit, welche, wie erwähnt, dazu dient, um die Injektionsmasse in die feinsten Gefässverzweigungen dringen zu lassen, wurde das Präparat in der Weise verkleinert, dass Stirnhöhle, Nasenhöhle und die übrigen Nebenhöhlen in einem zusammenhängenden Stücke gelassen wurden, welches dann in die Fixationsflüssigkeit, alkoholische Formalinlösung gebracht wurde. Bei dem Verkleinern wurde die Stirnhöhle, welche sich hoch nach oben erstreckte, eröffnet, man sah mit freiem Auge einige bläuliche streifenförmig gefärbte Stellen.

Cap. II. Durchspülung, Injektion von der Carotis aus, unter den erwähnten Kautelen, Karmingelatine. Entfernung des oberen Teiles der rechten Stirnbeinschuppe, die Dura wird vorsichtig mittelst gezähnter Pincette aufgehoben und mit Kanüle erst rechts eingestochen, die Injektionsflüssigkeit hebt die Dura empor, durch eine kleine Lücke Extravasation, hierauf wird links eingestochen, die Injektionsflüssigkeit spannt und hebt die Dura auch hier, doch nicht so vollkommen wie rechts, auch an dieser Stelle geringer Ausfluss von Gerotas Masse. Nach 24 Stunden Heraussägen des aus Stirn, Nasen, und übrigen Nebenhöhlen bestehenden Knochenstückes und Einlegen in alkoholische Formalinlösung. Nach entsprechendem Zuwarten, Durchsägen des Knochenstückes in sagittaler Richtung; es wurde eine mittelgrosse Stirnhöhle gefunden, vordere Wand von bedeutender Dicke, Spongiosa des Knochens sehr entwickelt, hintere Wand dünn. Masse: Höhe der Stirnhöhle 20 mm., sagittaler Durchmesser 6 mm., Dicke der hinteren Wand 2 mm. Makroskopischer Befund des Knochens der Hinterwand: derselbe von beiden Seiten blau, streifenförmige blasse Verfärbung. Ebenso blaue verschwommene Flecken an der Schleimhaut der Hinterwand.

Cap. III. Präparat in erhöhte Temperatur gebracht, Durchspülung, Injektion durch die Carotis, nach Unterbindung sämtlicher Gefässmündungen, dünne Karmingelatinlösung. Die Flüssigkeit kommt an einer Stelle zurück, welche Stelle tamponiert wird. Präparat wird in Eis gelegt, um rasches Erstarren herbeizuführen, dann subarachnoideale Injektion mit Gerotas Masse in der Gegend des aufsteigenden Astes der Fossa Sylvii. Untersuchung, sowohl hintere Wand als auch Schleimhaut, negativ.

Cap. IV. Aelteres Präparat, beschriebene Behandlung, Konservierung der hinteren und vorderen Wand der Stirnhöhle und der Stirnhöhlenschleimhaut in Alkohol.

Cap. V. Ebenfalls subdurale Injektion, mit Gerotas Preussischblau. Untersuchung ergibt makroskopisch negatives Resultat. Hinterwand und Schleimhaut werden konserviert.

Cap. VI. Subdural injiziert, Untersuchung ergibt Mangel der Stirnhöhle.

Cap. VII. 31 J. alt, subdurale Injektion, Untersuchung zeigt weite gefächerte Stirnhöhle mit dünner Hinterwand, welche entfernt wird. Hinterwand und Stirnhöhlenschleimhaut, welche abgelöst wird, werden fixiert.

Das untersuchte Material beläuft sich auf sieben Cap. Erwachsener, im Alter von wenigstens 31 J. Es würden bei jugendlichen Individuen mit günstigeren Bedingungen wahrscheinlich bessere Ergebnisse zu Tage gefördert werden, doch konnte entsprechenderes Material nicht beschafft werden. Die Untersuchung geschah in jedem Falle in möglichst frischem Zustande, ausschliesslich auf dem Wege indirekter Injektion, meist vom subduralen Raume aus, in der beschriebenen Weise. Die grossen Schwierigkeiten der Untersuchung Erwachsener, die Schwierigkeiten der befolgten Technik, machen es erklärlich, dass das für die Deutung gewisser Komplikationen so wichtige Erlangen entsprechender Versuchsresultate so schwer ist. Es ist mir in einem Falle gelungen, nach der Injektion, in der Schleimhaut der Hinterwand der Stirnhöhle eine nicht vollkommene Blaufärbung zu finden. Im Schnitte stellt sich die Färbung als aus einzelnen blauen Strichen und Punkten bestehend dar. Wir finden eine, mit der Oberfläche der Schleimhaut parallel verlaufende nicht zusammenhängende Injektion vor. Die fragliche Partie, mit starker Vergrösserung genau betrachtet (Fig. V), tritt in der Verbindungslinie der einzelnen blauen Massen und schollenförmigen Anhäufungen ein sich vertiefender, ziemlich scharf konturierter Kanal hervor, welcher sich an drei Stellen verzweigend, an mehreren Stellen unregelmässiges Kaliber zeigt, in der Weise, dass den ampullenförmigen Ausweitungen Einschnürungen folgen. Zwei der Zweige, der obere und einer der Endzweige, verlieren sich in tiefere Schichten. Dieser Befund muss dahin gedeutet werden, dass es sich um eine wenn auch unvollkommen injizierte Lymphgefässverbindung handelt. Dass diese Lymphgefässverbindung mit den perimeningealen Räumen, speziell mit dem Subduralraume zusammenhängt, beweist der Umstand, dass ihre Füllung durch Injektion in den Subduralraum zustande kam. Auf welchem Wege dies geschah, ob auf dem Wege eigener, durch den Knochen hindurchgehender selbstständiger Wege, wie dies bereits nachgewiesen wurde, konnte in unseren Fällen nicht entschieden werden. Die Untersuchung einer grossen Anzahl von Schnitten angefertigt aus der entkalkten Hinterwand der Stirnhöhle führte uns zu keinem positiven Resultate. Hingegen kann darauf hingewiesen werden, dass Lymphgefässe der Nasenschleimhaut nicht injiziert waren.

Wir können somit auf die Frage, ob es in der Stirnhöhlenschleimhaut beim Menschen Lymphwege gibt, gestützt auf das Ergebnis unserer mehr

als individuelle Geltung besitzender und früherer Untersuchungen bestätigend antworten, auch wir konnten beim Menschen kein wirkliches Netz, sondern nur mit blauer Injektionsmasse gefüllte, unregelmässige an einzelnen Stellen verzweigte und ampullenförmig erweiterte Kanäle beobachten, welche, wie nun nachgewiesen, mit den perimeningealen Räumen des Gehirns zusammenhängen. Es sind somit nicht nur für das Tier, sondern auch für den Menschen, die von den perimeningealen Lymphräumen des Gehirns führenden Lymphbahnen der Stirnhöhlenschleimhaut nachgewiesen. Wege, welche für den Transport des infektiösen Materiales ebenfalls verantwortlich gemacht werden müssen. Ob nur eine kleine Anzahl von Komplikationen als direkt auf diesem Wege entstanden angenommen werden muss, dafür wird der klinische und anatomische Beweis in den Fällen von infolge von Stirnhöhlenentzündung entstandener intracranieller und cerebraler Komplikationen, durch entsprechend genau ausgeführte Untersuchungen erbracht werden müssen.

Wir kommen auf Grund der angeführten und unserer eigenen Untersuchungen, welche wir im I. Anatomischen Institute der kgl. ung. Universität in Budapest machen konnten, wofür wir dessen Direktor Herrn Hofrath Professor Doktor Michael von Lenhossék, und dem Adjunkten an diesem Institute, Herrn Dozenten Dr. v. Nagy für dessen werktätige Hilfe, besten Dank sagen, zu folgenden Schlüssen :

I. Der Zusammenhang der perimeningealen Räume des Subdural- und des Subarachnoidealraumes mit dem Lymphgefässnetze der Stirnhöhlenschleimhaut, auf dem Wege der Lymphbahnen der Nasenschleimhaut, beim Tiere (Kaninchen) ist bekannt.

II. Der direkte Zusammenhang der perimeningealen Lymphräume mit dem Lymphnetze der Stirnhöhlenschleimhaut ist festgestellt.

III. Der Zusammenhang der perimeningealen Lymphräume mit dem Lymphnetze der Stirnhöhlenschleimhaut auf selbstständigen, den Knochen passierenden Wegen, ist sichergestellt.

IV. Der anatomische Nachweis des Zusammenhanges der Lymphwege der Stirnhöhlenschleimhaut mit den Lymphräumen des centralen Nervensystems beim Menschen ist erbracht.

V. Die Wege, auf welchen intracranielle und cerebrale Komplikationen von der Stirnhöhle aus stattfinden können, sind ausser dem bekannten, die in direktem Zusammenhange mit den perimeningealen Lymphräumen stehenden Lymphwege der Stirnhöhlenschleimhaut.

SECTION XV

RHINOLOGY AND LARYNGOLOGY

INDEPENDENT PAPER

UNTOWARD RESULTS FOLLOWING THE EXTERNAL OPERATION ON THE FRONTAL SINUS. A CRITICAL REVIEW OF TWENTY CASES

By ROSS HALL SKILLERN, Philadelphia, U.S.A.

Less than two years have elapsed since Boenninghaus collected and published the unfortunate results of the Killian operation. He laid, however, especial stress upon the fatal terminations, and did not enter into detail regarding those sequelæ which were not dangerous, but could readily be of temporary annoyance and even actual permanent discomfort to the patient. It is these very end-results which I have the honour to bring before you for your consideration this afternoon, and which are taken from all the cases that have come under my care. In each one of these the Killian procedure had been followed. Before entering into the individual cases, let us consider the untoward conditions which may follow the operative procedure :

1. Œdema of eyelids, particularly the superior.
2. Paralysis of the upper lid.
3. Continuation of the discharge.
4. Fistula or abscess formation.
5. Hemicranial anæsthesia.
6. Neuralgia : (*a*) local, (*b*) hemicranial.
7. Deformity : (*a*) sinking in of forehead, (*b*) contraction of scar, (*c*) falling out of eyebrows, (*d*) excessive growth of eyebrows, (*e*) excessive growth of scar tissue (keloid).
8. Formation of pneumatocele.
9. Temporary and permanent diplopia.
10. Blindness on operated side.

1. *Œdema of the eyelids.* This occurred in every case, beginning from two to four days after the operation. The duration and extent of the œdema depended upon the amount of bone resection required for the inferior wall. Thus the more extensive the resection, the longer the duration of the œdema. Pressure and traction upon the eyeball exercised no little influence upon the prompt appearance of the turgidity, as was exemplified in our earlier cases when the optical structures were

subjected to more or less traumatism due to our imperfection of finer technique. Since using a specially designed eye-protector, the subsequent œdema has not appeared so promptly, neither has it been so well marked nor persisted so long.

As a general rule the œdema appears at the end of about forty-eight hours, and reaches its greatest maximum on the fourth or fifth day. This gradually subsides, requiring often three or four weeks before the last traces disappear. Care should be taken not to confuse a partial ptosis with a persistent œdema. Where considerable traumatism has occurred from the orbital retractor, the swelling appears on both the upper and lower lids, while the upper lid is alone affected from the supra-orbital incision, which has of course severed the main return channels of the venous blood.

Pathological importance cannot be attributed to this œdema unless it assumes proportions such as would lead one to believe that breaking down and suppuration were imminent ; such an eventuality, however, has not as yet come under our observation. In one case (with tubercular history) the œdema has persisted, sometimes involving the entire forehead, but more often being confined to the upper eyelid of the operated side. Curiously enough, this œdematous swelling is recurrent in type, one day practically disappearing only to suddenly reappear without any apparent cause. The possibilities of an angio-neurotic type might be considered in this connexion.

2. *Paralysis of the upper lid.* Immediately after the operation this condition was always present, but as it co-existed with the œdema it was difficult to judge whether it was due to trophic or mechanical disturbances. In only two cases did it show any disposition to become permanent. The ptosis here is not well marked and does not include the entire lid, but only a portion of its internal edge.

3. *Continuation of the discharge.* The length of time the discharge continues from the affected nostril will depend largely upon the extent of the surgical intervention. Thus, if the frontal sinus was small, and only the anterior ethmoid cells co-affected, the purulent outflow will be practically nil, and entirely disappear at the end of two or three weeks. If, on the other hand, it has been necessary to completely eradicate the ethmoid labyrinth and resect the anterior sphenoidal wall as well, we can confidently expect a discharge for weeks and even months, which gradually diminishes both in amount and purulent consistency, until a thin serous or sero-purulent secretion remains. This appears to continue indefinitely, despite frequent applications of the various silver preparations, as has been observed in about 50 per cent. of our cases.

It must be emphasized that the post-operative discharge can hardly be compared with the original condition, that it is at first the secretion from a wounded surface, and, even though it become semi-purulent, stagnation is impossible, thereby eliminating the danger and previous discomfort to the patient.

4. *Fistula or abscess formation.* These may be classed as primary and secondary. Primary, those appearing before healing of the parts has occurred; secondary, those resulting from reinfection and appearing long after the external incision had healed. These happened in five of our cases. Two formed fistulas during the process of healing, and three became reinfected, which resulted in abscess formation along the line of the original incision.

In the first case of primary fistula formation the sinus was very large, extending well up toward the vertex. During retraction the skin was torn, and a fistula formed below and above the ridge of bone. This necessitated packing with iodoform gauze, and the parts healed by granulation, the entire process requiring about one month.

The second occurred at the outer corner of the wound in a large sinus, which extended to the malar bone, and was due to stagnation in this part. The insertion of iodoform gauze drains soon brought about healing.

The three cases of abscess formation occurred five weeks, eight months, and eleven months after operation. The first one was due to a piece of buried silkworm gut. No definite cause could be assigned to the others. It would appear that the longer the primary sutures are allowed to remain after the healing process has well begun, just so much more will be the danger of subsequent infection with abscess formation.

Necrosis and breaking down of the ridge of bone has not occurred in any of our cases.

5. *Hemicranial anæsthesia.* This form of neurosis occurred in every case, which was only to be expected, as the supra-orbital nerve had invariably been severed. The curious part was the difference in discomfort; in other words, the attitude of the different patients toward this condition. The complaints were limited entirely to the females, being described as a numb sensation, as though the part were dead. Sensation returned in every case, although the time of the establishment of the collateral nerve function differed markedly. In the majority of instances six months were sufficient, while in six cases certain areas of anæsthesia are still present, and two of these belong to the earlier operations. There appears also to be some connexion between the duration of the anæsthesia and subsequent neuralgia. Thus in, I think, all of the cases in which the hemicranial anæsthesia was unduly prolonged, neuralgic manifestations always appeared subsequently.

6. *Neuralgia.* This is one of the most annoying sequelæ that can follow operative intervention on the frontal sinus. Not only is the patient distressed, but the surgeon also, from the persistent importunities of the former to rid her of this continual pain. This is especially the case should neurasthenia be combined, as not infrequently appears in women, the sinus disease first causing the neurasthenia. Neuralgia or neuralgic manifestations were present in seven of our patients, but only in two were the symptoms sufficiently severe to demand surgical

intervention. In one, despite resection of the nerve, the neuralgia recurred several weeks later. It would appear as though several conditions may give rise to this neurosis.

(1) The nerve endings becoming caught in and bound down by the scar.

(2) Continuation of the original neuralgia which accompanied the sinus disease.

(3) Neurasthenia.

(4) Bulbar enlargement at end of nerve.

It has been our observation, the more marked the deformity, the greater the neuralgic manifestations.

7. *Deformity.* The cosmetic result of the operation depends largely upon the size of the sinus, particularly the antero-posterior dimensions. Another very important factor is the method of healing which has taken place. If by first intention, the resulting scar is so slight that it becomes almost unnoticeable. If, for any reason, by granulation, the scar tissue is very prominent, and by subsequent shrinking not only produces greater or lesser deformity, but also adds to the discomfort of the patient by embracing the terminal ends of the severed supra-orbital nerve. The scar is always more prominent from the inner end of the eyebrow downward along the side of the nose, while the depression is always found above the eyebrow toward the median line.

Our cases may be classified as follows: thirteen cases practically perfect, no scar or other defect recognizable unless special attention were called.

Four cases, slight depression over operated eye, and some contraction of scar tissue along descending incision, this being due to subsequent reinfection. Result may be called very good.

Two cases, the cosmetic result was not quite so good. In one the depression above the eye was marked, being due to an exceptionally high and deep sinus. This could have easily been corrected with paraffin, but the patient (a miner) was perfectly satisfied, and would not consider any further surgical treatment. In the other case there was no deformity, but an œdema persisted, which gave the forehead a swollen shiny appearance. She also had reinfection along line of incision. This case has already been referred to.

One case showed considerable deformity, both from the depression and from scar-tissue contraction. During the operation the skin was torn from too great retraction. Healing took place by third intention, a fistula beneath the bridge of the bone persisting for several weeks. Reinfection occurred several months afterward.

Present condition: some sinking in of forehead. Scar tissue prominent, drawing skin for several centimetres. The entire condition is much better after several injections of paraffin, so that deformity is not particularly noticeable.

From the retrospect of our cases it is unwise to give a prognosis

of the deformity immediately after the operation, as the parts will not assume their permanent position until several months have elapsed.

8. *Formation of pneumatocele.* We noted a bulging over the anterior wall on blowing the nose in two cases on the first dressing. After compression with tight bandage this disappeared. In none of the cases was this visible after the bandages were permanently removed.

9. *Temporary and permanent diplopia.* In fourteen of the cases, double vision was present immediately after the first bandage was removed. This lasted a variable length of time, but in the majority of instances disappeared in a few days. Thus in eleven the diplopia had completely disappeared at the end of the first month. In one case it lasted for nearly four months, and in two cases it appeared permanent. These were number five and number fifteen, both young girls, in which the sinuses were not especially large. In Case 5 the diplopia first appeared some two weeks after the operation, and became more marked rather than less as time elapsed. In the other case, double vision appeared on removal of the first dressing, and has persisted. The ocular disturbances in the first case can be explained when one takes into consideration its delayed appearance. During the process of healing the trochlea became involved in the new forming scar tissue, and became anchored, as it were, with resulting loss of function. The cause of the diplopia of the second case is not clear, but it would seem as though hysteria played a not inconsiderable rôle, as at first it was not only binocular but monocular as well. Repeated examinations failed to reveal the cause of this curious condition. After several weeks the monocular diplopia disappeared, but the binocular persists even to the present time (operation April 28, 1911).

In both of these cases the diplopia was counteracted by the wearing of appropriate lenses, and it would seem that the visual disturbance is also improved, although on removing the glasses the double vision (two years and one year after operation) is still marked.

10. *Blindness on operated side.* This unfortunate sequela occurred in Case 16., curiously enough, one of our later operations in which our technique was established. The disease involved the frontal, entire ethmoid and sphenoid on the right side, and complete exenteration of these parts was demanded. The operation progressed as usual, offering no especial difficulties, and it was not until the first dressing was removed and the patient tested for diplopia, that it was discovered that the eye was sightless. The eye ground was gone over immediately by an experienced ophthalmologist, who reported absolutely negative findings except pupil did not react to light. Despite every endeavour, the eye remained blind. In reviewing this case several possibilities may be advanced. First, in breaking away the lamina papyracea the bone may have been fractured into the optic foramen. Second, the optic nerve may have become surrounded by an extravasation of blood, which became organized. Third, a dehiscence may have existed in the sphenoid

sinus and the optic nerve sheath may have been injured while opening this sinus. The last explanation seems the most plausible. It is interesting to note that this occurrence has been reported twice previously.

Case 1. Agnes McAvoy, 28 years old.
Operation, Killian complete, June 25, 1909, Polyclinic Hospital.
Severe supra-orbital headache, impossible to concentrate thoughts or study. Purulent fœtid discharge from right nostril, three years.

1. Six or seven weeks.
2. Several weeks ptosis.
3. Five or six weeks.
4. Stitch abscess one week after operation.
5. Eighteen months gradually disappeared.
6. Slight supra-orbital; several months.
7. (*a*) Slight depression; (*c*) yes, but returned.
8. Four to five weeks after operation.
9. None.
10. None.

Case 2. Mary Wynne, 50 years old.
Operation, Killian, November 18, 1910, Rush Hospital.
Increasing frontal pain, great tenderness over frontal sinus; purulent fœtid discharge from nose; over two years.

1. Disappeared in few days.
2. None.
3. Several months; exacerbation with every fresh cold.
4. None.
5. Several months, somewhat annoying.
6. Slight neuralgia on exposure to cold or wet.
7. None; result perfect.
8. None.
9. Evanescent.
10. None.

Case 3. Grace Powell, 29 years old.
Operation, Killian, December 15, 1910, Rush Hospital.
Chronic frontal sinusitis; pain and discharge; small sinus; about one year; complete cure.

1. Several days.
2. None.
3. Several weeks.
4. None.
5. Slight, not complained of.
6. None.
7. None.
8. None.
9. None.
10. None.

Case 4. James Berry, 40 years old.
Operation, Killian, March 7, 1911, Rush Hospital.
Chronic frontal sinusitis; discharge fœtid; headaches violent several years; cured.

1. About three weeks.
2. Slight; evanescent.
3. Gradually ceased after several months.

4. None.
5. Not complained of.
6. None.
7. Slight, due to sinking in.
8. None.
9. None.
10. None.

CASE 5. Sadie McShane, 21 years old.
Operation, Killian complete, April 28, 1911, Rush Hospital.
Continual left supra-orbital headache; profuse purulent discharge from nose, also from post-nasal; gradually worse over a year; complete relief.

1. Lasted three months; now entirely gone.
2. No.
3. Two weeks; becomes affected with coryza.
4. No.
5. Almost one year.
6. Super-sensitiveness; now about the same.
7. (*a*) Slight flatness; (*b*) slightly visible; (*c*) none; (*d*) no; (*e*) no.
8. No.
9. Two weeks after operation; still in lower field; gradually getting better.
10. No.

CASE 6. Mrs. J. H. Carman, 46 years old.
Operation, Killian, May 18, 1911, Rush Hospital.
Severe headache and tenseness in right side of head and face; dizziness marked; copious discharge from right nostril; one year; instant relief.

1. Two or three weeks.
2. None.
3. Four or five months.
4. None.
5. Nine or ten months.
6. Over site of scar.
7. None; cosmetic result perfect.
8. None.
9. None (never appeared).
10. None.

CASE 7. Ida Lewis.
Operation, Killian, August 22, 1911, Rush Hospital.
Continual colds, gradually becoming worse; supra-orbital pain, unilateral; nasal discharge; discharged cured.

1. Almost disappeared at time of discharge.
2. None.
3. No data available.
4. None.
5. No data available.
6. No data available.
7. No data available; none at time of discharge.
8. None.
9. Had disappeared.
10. None.

Case 8. Elizabeth McWhirter, 26 years old.

Operation, Killian, September 8, 1911, Rush Hospital.

Intermittent supra-orbital headache; inability to work; pus from nose; impossible to use eyes for sewing, &c.; nearly two years; great relief.

1. Six or eight months; still slightly noticeable.
2. No.
3. Intermittent to present.
4. Abscess, December 18, 1912; swelling and tenderness along line of supra-orbital incision; intra-nasal treatment; sudden discharge of large quantity of fœtid pus (12/23); recovery.
5. None.
6. Anæsthesia for several weeks.
7. (*a*) none; (*b*) none; (*c*) none; (*d*) none; (*e*) slightly visible.
8. No.
9. None.
10. None.

Case 9. Frances Kane, 31 years old.

Operation, Killian complete, November 10, 1911, Medico-Chirurgical Hospital.

Continual and excessive headaches and purulent fœtid discharge from nose. Stenosis of lachrymal duct; lachrymation; twelve years. Absolute relief until August, 1912; contracted grippal cold; nose began to discharge, not so profusely nor fœtid. Some return of headaches, not so severe, but similar; trifle neuralgia over scar; depression in forehead; reinfection of remains of ethmoid cells and sphenoid; operation ethmoid, January 21, 1913, and result good; paraffin injection January 28, 1913.

1. One month.
2. Three or four months before drooping disappeared.
3. Still slight; inclined to crust; some bother (due to ethmoid).
4. None.
5. Six to eight months.
6. Slight local; several months; never at any time very severe.
7. (*a*) Yes, quite noticeable; remedied by paraffin, January 1913, which also cured anæsthesia.
8. No.
9. No.
10. No.

Case 11. H. Agranowitz, 20 years old.

Operation, Killian complete, February 6, 1912, Medico-Chirurgical Hospital.

Sudden exophthalmos, with fever, 104°. Great pain and prostration; no discharge.

1. Several months.
2. Difficult to determine, on account of œdema.
3. Several months.
4. Eight months.
5. Not complained of.
6. None.
7. None, except in soft parts.
8. None.
9. None.
10. None.

Case 12. P. Condon, 40 years old.

Operation, Killian complete, March 23, 1912, Medico-Chirurgical Hospital.

Severe pains over frontal for several years; gradually becoming worse; no tenderness over frontal; purulent discharge from nose since attack of grippe over one year previous; operation March 23, 1912; enormous sinus filled with pus and granulations; frontal wall partly necrotic; present condition, patient entirely well; considerable sinking in.

1. Disappeared in few days.
2. None.
3. No data available.
4. None.
5. No data available.
6. No data available.
7. Considerable flattening.
8. None.
9. None.
10. None.

Case 13. Jennie Alexander, 31 years old.

Operation, Killian, May 15, 1912, Rush Hospital.

Severe frontal headache, practically continuous, extended to temporal and occipital regions; profuse purulent discharge from nose; marked indigestion and sore throat; twelve years' tubercular history; absolute relief from headaches; some slight discharge from nose remaining; November 23, 1912, reinfection swelling over scar, with pain, abscess. Present time, intermittent neuralgia like sudden sting of bee, gradually abating; worse in bad weather; still some discharge from nose; sore throat and indigestion disappeared; 4/9 complains of drawing in scar tissue.

1. Intermittent; when present extends upward to forehead.
2. Very slight.
3. Still present, though not so profuse.
4. Has occurred three times along line of perpendicular incision.
5. Present.
6. Present.
7. Slight contraction of scar tissue.
8. None.
9. None.
10. None.

Case 14. Mary Rogers, 50 years old.

Killian operation, anterior and posterior ethmoid and sphenoid, June 12, 1912, Medico-Chirurgical Hospital.

Constant pain in supra-orbital region and parietal; feeling as though something was loose in head; some purulent discharge from nose; several years; during operation skin torn; fistula above and below bridge healed in two weeks, leaving depressed scar; five weeks after operation began to feel drawing sensation about one inch above scar; developed into true neuralgia; intermittent attacks; begins about 8 a.m., one hour, then about 4 p.m.; almost unbearable; January 4, 1913, paraffin injected; cosmetic result good; neuralgia better; January 13, alcohol injections; no result.

1. Several months.
2. No droop.

3. Still present.
4. None after healing.
5. Several months followed by
6. Stinging sensations, sometimes darting.
7. Considerable sinking.
8. None.
9. Lasted seven months.
10. None.

Case 15. Bessie Sturgis, 22 years old.
Killian complete, July 22, 1912, Medico-Chirurgical Hospital.
Severe headache over left supra-orbital region; discharge from nose and into throat; dizziness on stooping; could not use eyes for work; kakosmia; sore throat; several months; great relief.

1. About nine weeks.
2. Partial several months.
3. Continues, thinner, no crusting, not so profuse; uses five handkerchiefs daily.
4. No.
5. Six months or so; still anæsthesia immediately above scar tissue.
6. No.
7. None; result excellent.
8. No.
9. Diplopia in all fields except to right.
10. No.

Case 16. Jane A., 31 years old.
Killian, December 11, 1912, Medico-Chirurgical Hospital.
Severe supra-orbital pain and profuse discharge; for past ten years gradually growing worse; general system affected; patient anæmic; sleeps badly, no appetite; loss of weight and strength, coughs, &c. Immediately after operation patient complains of blindness in right eye; absolute relief of headaches, and discharge thin and scanty; some new pain in eyeball and temple; sight never returned, and at present patient still blind on right side.

1. About six weeks.
2. Still slight droop.
3. Exacerbates on exposure to cold or wet.
4. None.
5. Marked.
6. Darting pains through eyeball and temple.
7. None.
8. None.
9. None.
10. Complete.

Case 17. Charles Lohss, 62 years old.
Killian operation, December 18, 1912, Medico-Chirurgical Hospital.
Large mucocele of left frontal sinus; several years; complete cure.

1. Several months.
2. Very slight ptosis.
3. Two or three weeks.
4. None.
5. Not complained of.
6. None.
7. (*a*) Slight.

8. None.
9. Present before operation; not now.
10. None.

CASE 18. Alice Griffith, 32 years old.

Killian complete, January 29, 1913, Medico-Chirurgical Hospital.

Pain in frontal sinus and left eye; gradual exophthalmos; œdema of eyelid; no discharge from nose; ethmoid cells polypoid and degenerated; sphenoid also; mucosa thickened; sight almost gone.

1. Still present.
2. Great ptosis.
3. No discharge.
4. None.
5. Not complained of.
6. None.
7. None.
8. None.
9. Eyesight almost gone; no better.
10. Was almost blind before operation.

CASE 19. F. E. Lanious, 41 years old, April 22, 1913.

Patient in accident; injured in automobile; Medico-Chirurgica Hospital.

Left frontal sinus crushed in with subsequent reinfection; necrosis of anterior wall with fistula formation; over one year.

1. Slight; soon disappeared.
2. None.
3. None.
4. Healed.
5. Not complained of.
6. None.
7. Marked depression.
8. None.
9. None.
10. None.

CASE 20. Elberta Lanahan, 18 years old.

Operation, April 19, 1913, Medico-Chirurgical Hospital; healing by first intention.

Continual and excessive headaches and purulent discharge from left side for one year; absolute relief from pain; discharge gradually ceasing (May 17); anæsthesia frontal and parietal; some tenderness in scar; no visible depression; slight œdema of left eyelid; diplopia set in one week after operation; everywhere except to left; now very much better.

1. About ten weeks.
2. None.
3. Still present at end of twelve weeks, though greatly lessened.
4. None.
5. Present.
6. (*a*) Some tenderness in scar.
7. Vertical scar prominent; no depression.
8. None.
9. On certain positions of head diplopia.
10. None.

DISCUSSION

Dr. R. F. Ridpath (Philadelphia). As I have had the honour of assisting Dr. Skillern in many of his operations, I feel it my duty to state that in his clinic no fewer than three, and very often all the members of his staff, go very carefully over the patients, and make their respective diagnoses and recommendations for operative or medicinal treatment. In all of the cases included in his report this preliminary precaution has been taken, so that the natural question, Were these cases necessarily operative ones ? or, Was a radical Killian necessary ? can be eliminated.

It is to be understood that the technique employed was the same as advocated by Professor Killian, all the normal tissue being saved, all the diseased portions removed, and all possible traumatism eliminated, under which circumstances similar results would have followed in any event. In spite of the somewhat disagreeable sequelæ, many of those operated upon stated that they would much rather be in their present state than as they were before the operation.

SECTION XV

RHINOLOGY AND LARYNGOLOGY

INDEPENDENT PAPER

WEITERE ERFAHRUNGEN MIT MEINER METHODE ZUR DIREKTEN BESICHTIGUNG DES NASEN-RACHENS UND DESSEN DIREKTES PHOTOGRAPHIEREN

Von A. GYERGYAI, Kolozsvár, Ungarn

Meine Herren! Es sind schon drei und ein halb Jahre verflossen, seit ich meine Methode zur direkten Untersuchung des Nasenrachenraumes, der Ohrtrompete, des hintersten Teiles der Nase und mein Verfahren zur direkten Operation der genannten Teile anwende. In den verflossenen letzten drei Jahren verwendete ich fast meine ganze freie Zeit zur Vervollkommnung meiner Methode, zum Studium der anatomischen und pathologischen Verhältnisse der untersuchbaren Teile und ihrer Umgebung und versuchte die Vorteile meiner Methode auf dem Gebiete der Therapie der Krankheiten des Nasenrachenraumes und des Gehörorganes zu verwerten.

Meine Erfahrungen, die ich bei der Anwendung meiner Methode sammelte, möchte ich kurz erwähnen.

Die Abänderungen fasse ich in Folgendem zusammen:

1. Den Kranken untersuche ich nicht in liegender Stellung mit herabhängendem Kopfe, sondern meist in sitzender Lage, was einfacher, für den Patienten weniger unangenehm und auch für den Untersucher bequemer ist; ferner verdeckt kein Speichel, bei Operationen kein Blut, störend das Gesichtsfeld.

2. Statt Untersuchungsröhren, die nur an ihren beiden Enden offen sind, gebrauche ich jetzt immer solche, deren Enden seitlich an zwei Stellen so ausgeschnitten sind, dass oberhalb des weichen Gaumens hinten, unterhalb desselben aber die Röhre vorne ganz offen ist.[1] Durch diese Abänderung ist das Gesichtsfeld viel grösser geworden, so dass ein Untersuchungsrohr von 12 mm vollkommen ausreicht, und zur Beleuchtung entspricht statt des Brünings'schen Elektroskopes auch der einfache

[1] Diesbezügliche Instrumente sind bei F. L. Fischer, Freiburg i. Br., Kaiserstrasse 113, zu haben.

Stirnreflektor, da durch den Ausschnitt der Röhre mehr Licht auf die zu untersuchende Fläche gelangen kann.

3. Bei der Einführung der Untersuchungsröhre wird der weiche Gaumen jetzt mit einem ziemlich kurzen Haken vorwärtsgezogen, der weniger irritiert wie der Finger — daher ist auch geringere Kokainisierung genügend und hie und da gelingt die Untersuchung auch ohne Kokain.

Ich befand es für gut, wenn ich bei geschlossenem Mund einen mit Kokain getränkten Tamponträger ¼–1 Minute lang im Nasenrachenraum halten liess, das Kokainisieren nicht mehrmals zu gebrauchen, wie 3–4 mal und hielt sowohl in der Zwischenzeit, als auch vor der Einführung der Röhre, Pausen von nur einigen Minuten (2 bis höchstens 4).

Den Mundwinkel pflege ich vor allem gegen die Mitte der unteren Lippe einzuölen, da ja sonst die Einführung stossweise vor sich geht und sich die Lippe leicht zwischen die Röhre und Zähne einklemmt, was unangenehm ist und auch die Untersuchung erschwert. Es ist nämlich wichtig, dass die Einführung der Röhre glatt und leicht geschehe.

Während des ganzen Verfahrens müssen wir achtgeben, dass der Kranke keinen Brechreiz bekomme, was wir hauptsächlich dadurch vermeiden können, dass wir die hintere Wand des Rachens, vor allem deren oralen Teil, nicht berühren, sondern den Kokainträger, den Haken oder die Röhre nur auf der hinteren Fläche des weichen Gaumens hinaufführen.

Wichtig ist es, dass wir den Haken und die Röhre auf der linken Seite der Uvula einführen, wenn wir die rechte Tubenöffnung untersuchen wollen und umgekehrt. Das innere Ende des Hakens halten wir in der Richtung der zu untersuchenden Fläche, zum Beispiel in der Richtung der anderseitigen Tubenöffnung, die Röhre führen wir unmittelbar neben dem Haken, lateral von ihm, ein und geben acht, dass wir den Haken mit der Röhre nicht andrücken, denn sonst verursacht seine Spitze leicht ein stechendes unangenehmes Gefühl und erregt Brechreiz. Die hineingeführte Röhre halten wir mit einem sicheren Griff an Ort und Stelle, ohne sie stärker anzudrücken, den Haken aber nehmen wir, ohne die hintere Wand zu berühren, heraus.

Alle diese Veränderungen machten meine Methode leicht ausführbar und einfach, auch dem Kranken ist sie kaum unangenehm, so dass ich sie oft sowohl auf der Klinik, als auch bei meinen Privatpatienten ohne jedwede Hilfe ausführe. Von den früheren zirka 400 Fällen abgesehen, nahm ich im letzten halben Jahr bei 64 Kranken direkte Untersuchung vor, zusammen 180 mal ; jedesmal ist sie schon zum erstenmal gut gelungen.

Eine unangenehme Nachwirkung habe ich überhaupt nicht wahrgenommen, höchstens hie und da nach der allerersten Untersuchung ein unangenehmes Gefühl beim Schlucken, welches nur einige Stunden lang dauerte ; bei den genannten Kranken war nur in acht Fällen an der einen Hälfte der Uvula eine kleine Erosion. Bei der Wiederholung der direkten Untersuchung habe ich ein unangenehmes Gefühl, oder eine Erosion niemals wahrgenommen.

Hiemit habe ich ganz kurz meine Erfahrungen bezüglich der Aus-

führung der Technik meiner Methode geschildert, jetzt will ich in einigen Worten ihre Bedeutung, ihren Wert erwähnen, wie ich dies heute nach meinen Erfahrungen beurteilen kann.

Den Nasenrachenraum kann man mit dem Spiegel oder Salpingoskop, falls man Schwierigkeiten hat, bei Anwendung eines Gaumenhakens, ziemlich gut untersuchen.

Mit meiner Methode sehen wir den Nasenrachenraum aus einer anderen Richtung direkt, dennoch schreibe ich ihr als einer Untersuchungsmethode des Nasenrachenraumes nur bei der Untersuchung der Rosenmüller'schen Grube, der Rezesse der hinteren and und der Bursa pharyngea grössere Bedeutung zu. Anders gestaltet sich die Sache bezüglich der Besichtigung des Inneren der Ohrtrompete, die nur durch meine Methode ermöglicht wurde, der wir bezüglich der Ohrenheilkunde grosse Wichtigkeit zuschreiben müssen.

Meinerseits untersuche ich den Nasenrachenraum zuerst immer mit dem Spiegel, manchmal mit dem Pharyngoskop und nur wenn ich eine solche Veränderung fand, die entweder auf die Erkrankung der Rezesse hinweist oder meiner Ansicht nach einen direkten Eingriff erfordert, untersuche ich ihn mittelst meines Verfahrens.

Wenn von einem grösseren Eingriff die Rede ist, führe ich ihn nicht gleich bei der ersten Untersuchung aus, sondern ein andermal, denn die Kranken können zum zweitenmal meine Untersuchungsröhre besser und länger halten.

Der Hauptvorteil der direkten Methode bezüglich des Nasenrachenraumes tritt bei operativen Eingriffen zum Vorschein. Mein Verfahren gestattet nämlich, exakte operative Eingriffe unter der Kontrolle des Auges auszuführen.

Die gewöhnliche, erprobte Operationsmethode mit den Ringmessern entspricht dem Zweck, der Entfernung des mittleren grossen Teiles von adenoiden Wucherungen ganz. Aber zur Entfernung von adenoiden Geweben (eventuell auch nach einer Adenotomie zurückgebliebenen), die sich in den Rezessen der hinteren Wand, in der Rosenmüller'schen Grube, in der Fossa supra tubaria, auf dem Torus tubarius, um die Tubenöffnung herum befinden, ist die direkte Methode die exakteste und einfachste. Solche adenoiden Gewebe kommen viel öfters vor, als man denken würde, und nach ihrer Entfernung heilt oft ein hartnäckiger Rachen- oder Ohrtrompetenkatarrh sehr schnell. Die Operation pflege ich durch die Röhre hindurch hauptsächlich mit einer Kneipzange oder mit dem scharfen Löffel, seltener mit einer Schnüre zu vollbringen.

Das Entstehen von Pseudobändern, das Verkleben der Wände der Rosenmüller'schen Grube auf einer kurzen oder längeren Strecke ist sehr häufig, auch eine stärkere Verwachsung ist nicht selten. All das verhindert die freie Bewegung des Rachens, des Torus tubularius und hält oft katarrhalische Zustände des Rachens und der Ohrtrompete aufrecht.

Die Trennung der Verwachsungen in der Rosenmüller'schen Grube kann durch die Untersuchungsröhre hindurch mit einem stumpfen

Elevatorium oder vorsichtig mit einem scharfen Instrument, oder mit einem zu diesem Zwecke neuerdings konstruierten Erweiterer der Rosenmüller'schen Grube durchgeführt werden.

Mit der Komposition von Stents habe ich Abdrücke von der Rosenmüller'schen Grube auf Grund frisch bei Sektionen entnommener Präparate verfertigt. Nach diesen habe ich Erweiterer machen lassen, die das Negativ des tiefsten Teiles der Rosenmüller'schen Grube geben und schon durch meine Untersuchungsröhre mit dem Durchmesser von 12 mm hindurch einführbar sind.

Hier will ich meinen Tubenerweiterer erwähnen, der in die Ohrtrompete ganz einführbar ist und bei dessen Verwendung ich bei sehr vielen Ohrenleiden auffallend guten Erfolg erzielen konnte. Ueber meinen Tubenerweiterer und über die Fälle, die ich mit ihm behandelt habe, werde ich in der 'Otologischen Sektion' ausführlicher referieren.

Ausser der Tubenerweiterung gebrauche ich meine Methode am meisten zur Entfernung von adenoiden Wucherungen der lateralen Rachenwand und zur Trennung von Verwachsungen in der Rosenmüller'schen Grube.

Die anderen direkten Eingriffe, die ich im Nasenrachenraum vorgenommen habe, sind bedeutend weniger an Zahl, diese will ich nur eben erwähnen.

In einigen Fällen habe ich von der hinteren Wand des Nasenrachenraumes Cysten entfernt mit der grösseren Kneipzange; hier kann ich das direkte Bild einer Cyste zeigen, welche von der Rosenmüller'schen Grube der rechten Seite ausging (dieser Kranke kam zu Professor Chiari zur Operation). Oefters habe ich von Geschwülsten der oberen und der Seitenwand ein kleines Stück zu histologischen Probeuntersuchungen excidiert. In zwei Fällen habe ich die Membran der retrochoanalen Atresie entfernt, was nach der direkten Methode leicht ausführbar war. In einem Fall war die Atresie eine vollkommene, den beim Abdruck gewonnenen positiven Gipsabguss kann ich hier zeigen; im zweiten Fall waren beiderseits zwei Oeffnungen (vielleicht die Folge früherer Operationen), den Abdruck können die Herren hier sehen.

Den zurückgebliebenen Stumpf eines Fibroms der Schädelbasis entfernte ich vom Dach des Rachens.

In einem Fall ergriff ich einen grossen infolge von Syphilis nekrotisierten Knochen, der durch die Nasenöffnung wegen seiner Grösse nicht zu entfernen war, durch meine Röhre hindurch und nahm ihn mit der Röhre zusammen heraus.

Ich will nur noch meinen Photographhapparat den Herren vorführen, der zum direkten Photographieren des Nasenrachenraumes und des Kehlkopfes dient.[1]

Wir können im Nasenrachenraum nacheinander 8 Aufnahmen machen. Auf dem Film sind die negativen Bilder ungefähr 14 mal kleiner als die

[1] Photographhapparat ist bei F. Leiter, Mariannengasse No. 11, Wien, zu bekommen; die anderen Instrumente bei F. L. Fischer, Kaiserstrasse 113, Freiburg i. Br.

aufgenommenen Bilder. Bilder, die auf die natürliche Grösse vergrössert sind, kann ich den Herren hiemit vorführen.

Alles zusammengefasst kann ich sagen, dass mein Verfahren bei genügender Uebung leicht ausführbar ist und dass es von den Kranken gut ertragen wird.

Bei der Untersuchung der Rezesse, der Rosenmüller'schen Grube und vor allem der Tube gibt es einen guten Aufschluss. Seine Hauptbedeutung ist aber auf dem Gebiete der operativen Eingriffe zu suchen, nicht so sehr der seltenen, sondern gerade der alltäglichen, wie beispielsweise der adenoiden Gewebe der Seitenwand des Nasenrachenraumes, der Trennung von Verwachsungen, der Ausführung einer Erweiterung der Rosenmüller'schen Grube und der Ohrtrompete.

Hiemit können wir den Nasenrachenraum oft in einen fast normalen Zustand bringen, was sowohl bei den Katarrhen des Rachens und der Umgebung, als auch bezüglich der Ohrenkrankheiten sehr wichtig ist.

SECTION XV

RHINOLOGY AND LARYNGOLOGY

INDEPENDENT PAPER

ZUR ANATOMIE DER UNTEREN NASENMUSCHEL

Von Dr. R. HOFFMANN, Dresden

Die sensiblen Endigungen der Nerven in der respiratorischen Schleimhaut der Nase sind von Retzius bei der Katze und der Maus untersucht worden, von Kallius beim Kalb.

Vortragender gibt eine Darstellung der Verteilung der sensiblen Nerven (Trigeminus) in der unteren Nasenmuschel.

Der Trigeminus tritt an der Insertionsfläche der unteren Nasenmuschel mit starken einzelnen Nerven, bezw. schwächeren aber mehrfachen, in die Schleimhaut ein.

Die Nerven umspinnen die Gefässe (Arterien und Venen), das cavernöse Geflecht und die Drüsen. Sie lassen sich weiter verfolgen in stärkeren bezw. schwächeren mehrfach sich verzweigenden Aesten durch die Basalmembran hindurch, in das Epithel, zwischen dessen Zellen aufsteigend, dies umspinnend. Teilweis sieht man Aehnliches, wie es Kallius beim Kalb gefunden hat, nämlich, dass die feinen Fäserchen, ehe sie die Oberfläche erreicht haben, umbiegen und dann nach der basalen Seite der Epithelzelle hingewendet, aufhören.

SECTION XV

RHINOLOGY AND LARYNGOLOGY

INDEPENDENT PAPER

EINE SCHLEIMCYSTE DES NASENSEPTUMS MIT POSTOPERATIVER HYDRORRHOEA NASALIS

Von Dr. VICTOR FRÜHWALD, Assistent der K. K. Universitäts-Klinik für Kehlkopf- und Nasenkrankheiten in Wien (Vorstand: Hofrat Professor Dr. O. Chiari)

Meine Herren! Die Seltenheit des Falles, den ich an der Klinik meines Chefs, des Herrn Hofrat Chiari zu beobachten Gelegenheit hatte, lässt mich hoffen, bei Ihnen für meine kurze Mitteilung einiges Interesse zu finden.

Das Vorkommen von Cysten in der Nase und in ihren Nebenhöhlen ist eine schon längst bekannte Tatsache und von zahlreichen Autoren, wie Chiari, Killian, Heymann, u. a., in neuester Zeit von Sojo, Hall, McCullagh, Glas, und Kessler wurden Fälle beschrieben, bei denen es sich um cystische Gebilde handelt, die nach ihrem Inhalt als Hydro-, Muko- und Pyokelen oder als pneumatische Kavitäten zu bezeichnen sind. In unserem Falle war nicht nur der Sitz der Cyste ein aussergewöhnlicher, sondern die operative Behandlung zog auch ganz unerwartete Komplikationen nach sich, für die sich in der Literatur kein Analogon finden lässt.

Die Krankengeschichte dieses Falles ist folgende:

Am 10. Juni dieses Jahres kam ein 28 jähriger Mann, Dr. M. G., mit der Angabe an unsere Klinik, dass er seit Oktober vorigen Jahres an zunehmender Behinderung der Nasenatmung, besonders auf der linken Seite, leide. Vor 3 Jahren wurde bei dem Patienten wegen einer Deviatio septi eine submuköse Resektion vorgenommen, und zwar ebenfalls an unserer Klinik. Nach dieser Operation fühlte sich der Pat. wohl bis zum vergangenen Herbst, wo sich die bereits erwähnten Nasenbeschwerden einstellten. Gleichzeitig traten auch häufige Kopfschmerzen auf, die hauptsächlich in der linken Stirngegend lokalisiert wurden. Es wurden dem Pat. von ärztlicher Seite Nasenspülungen und Mentholöl angeraten, da diese Medikation aber gar keinen Erfolg hatte, so wandte er sich an unsere Klinik, wo wir folgenden Befund erheben konnten:

Status praesens: Mittelgrosser, kräftiger Pat. mit gut entwickelter Muskulatur und mässigem Fettpolster. Die sichtbaren Schleimhäute sind gut gefärbt.

Herz- und Lungenbefund: Normal.

Harnbefund: Vermehrte Epithelien und Lymphkörperchen im Sediment, sonst keine pathologischen Veränderungen.

Status localis: Bei der Rhinoscopia anterior zeigt sich, dass auf der linken Seite das Vestibulum nasi von einer hypertrophischen Schleimhaut, die von der unteren Muschel herzurühren scheint, fast vollständig ausgefüllt ist. Die Konsistenz dieses Tumors, der ca. haselnussgross ist, ist weich, die Farbe blassrot. Auf der rechten Seite lässt sich bis auf eine bedeutende hintere Schleimhautverdickung des Septums nichts abnormales finden. Die Rhinoscopia posterior ergibt einen normalen Befund.

Die Diagnose wird daher auf eine mächtige Hypertrophie der linken unteren Muschel gestellt und dem Pat. eine Operation vorgeschlagen, zu der er sich sofort entschliesst.

Operation. Bei der Anästhesierung — Bepinselungen mit 20 Prozent Kocain und Adrenalin — zeigt es sich nun, dass es erstens unmöglich ist, mit den Wattepinseln zwischen Nasenscheidewand und Tumor durchzukommen, und zweitens, dass diese Schwellung trotz ausgiebiger Anwendung von Adrenalin, nicht geringer wird. Man erkennt jetzt, dass die Schwellung nicht der unteren Nasenmuschel angehört, sondern vom Septum ausgeht und somit wird die eingangs gestellte Diagnose fallen gelassen.

Bei dem ersten Scheerenschnitt fliesst reichlich eine bräunliche, fadenziehende Flüssigkeit aus, die der chemischen Untersuchung zugeführt wird, und sich als Schleim erweist (Institut, Hofrat Ludwig). Die Reste der nun eingefallenen Cyste werden mit der Brünings'schen Zange fast vollständig entfernt. Die Nase wird nicht tamponiert.

Die histologische Untersuchung dieses exstirpierten Gewebes ergibt folgenden Befund (Professor Stoerk): Chronisch entzündliche, stark ödematös aufgelockerte und infiltrierte Schleimhaut, scheinbar ohne Besonderheiten. Inmitten dieses ödematös aufgelockerten Bindegewebes findet sich ein ziemlich dickes Nervenstämmchen, welches eine ganz eigenartige durch Flüssigkeitsdurchtränkung bedingte Auflockerung seiner Elemente, insbesondere auch in seinem peripheren Anteilen erkennen lässt, wie auch eine Trennung des Nervengewebes vom umscheidenden Zwischengewebe durch Flüssigkeitsansammlung in spaltförmiger Erstreckung.

Die Oberfläche (ausser durch Manipulation) unverändert. Ein zweites Stückchen, welches etwa der Umgrenzung eines Hohlraumes entspricht, besteht aus ziemlich derbem, fibrösem Gewebe und zeigt an seiner Oberfläche ein dichtes Lager von Kapillaren und Präkapillaren, stellenweise Gruppen von grossen Zellen, die den Eindruck von Phagocyten machen, deren Protoplasma von Erythrocyten vollgefüllt ist.

1½ Stunden nach der Operation bemerkt der Pat., dass ihm aus dem linken Nasenloch eine fast klare Flüssigkeit ausfliesst, die in Eprouvetten aufgefangen wird. Am folgende Tage hält diese Hydrorrhoea unvermindert an. Die Menge beträgt ungefähr 10–12 Eprouvetten in 24 Stunden, das ist ca. 300 ccm. Man kann beobachten, dass, wenn der Pat. den Kopf nach vorne beugt, die seröse Flüssigkeit an der Nasenspitze neben dem Septum herabtropft, wenn er hingegen auf dem Rücken liegt, dass sie ihm in den Rachen rinnt und dadurch auch seinen Schlaf stört. Da es sich möglicherweise um Liquor handelt, der aus einer Meningokele stammt, bekommt der Pat. abends 2 gr. Urotropin intern. Die chemische Untersuchung dieser Flüssigkeit, die ohne Unterbrechung aus dem linken Nasenloch des Pat. fliesst, ergibt, dass es sich nicht um Liquor cerebrospinalis handelt (Hofrat Ludwig). Das für die Cerebrospinalflüssigkeit

charakteristische Reduktionsvermögen war nicht nachzuweisen. Temperatur und Puls normal.

Bei der Röntgenuntersuchung kann man keine Oeffnung in der Lamina cribrosa erkennen. Keine Anzeichen von Hirndruck. Ausserdem zeigt das Röntgenogramm, dass die Stirnhöhle auf der rechten Seite fehlt und gleichfalls, dass die Kieferhöhle dieser Seite nur mangelhaft entwickelt ist.

Der Pat. erhält nun durch weitere 10 Tage 5 gr. Urotropin und zur Herabsetzung des Hirndruckes zweimal täglich 5 Tropfen Tinct. opii. Die Sekretion, die anfangs stationär blieb, nahm nach einer Woche merklich ab und fehlte sogar vollständig, wenn der Pat. am Rücken, mit nach rückwärts überhängendem Kopfe, lag. Am 16. Tage nach der Operation hörte die Sekretion gänzlich auf und konnte der Pat. geheilt entlassen werden. Auf der linken Seite des Septums sieht man ungefähr in der Höhe des vorderen Endes der mittleren Muschel geringe Reste der Cystenwand.

Wenn wir den Sachverhalt kurz rekapitulieren, meine Herren, so bestand eine Cyste des Septums, deren Inhalt sich als Schleim erwies, und im Anschluss an die Operation trat eine starke Sekretion aus der Nase auf, die für den ersten Moment deswegen beängstigend war, weil es sich eventuell um Liquor cerebrospinalis handeln konnte. Aus diesem Grunde wurde auch dem Pat. Urotropin in regelmässigen Dosen verabreicht. Es war die Möglichkeit vorhanden, dass eine Meningokele zwischen den beiden Septumblättern bestand, die nach Entfernung der Schleimcyste zur Ruptur gebracht wurde, da der Gegendruck aufgehoben war.

Die vorderen nasalen Hirnbrüche — und in unserem Falle hätte es sich ja nur um einen solchen handeln können — sind zwar eine äusserst seltene aber umsomehr wichtige Erkrankung und kommen nur angeboren vor.

In eingehender Weise hat Chiari in dem *Handbuch der ärztlichen Sachverständigen-Tätigkeit* das Kapitel der Meningokelen behandelt und teilt diese in extranasale, Hernia naso-frontalis und Hernia naso-ethmoidalis, und in intranasale, zu welchen er die Cephalokele spheno-pharyngealis und die Encephalokele basalis intranasalis rechnet. Besonders letztere hat für den Rhinologen eine höhere Bedeutung, und zwar deswegen, weil sie sehr leicht mit einem Polypen verwechselt werden kann. Da diese intranasalen Hernien keine respiratorische und pulsatorische Bewegung zeigen und man ausserdem durch Druck auf sie keine Gehirnerscheinungen auslösen kann, so ist ihre Erkennung sehr schwer. Die Vermutung aber konnten wir nicht haben, dass es sich um einen Hirnbruch, also um eine kongenitale Erkrankung, handeln könnte, da ja dieser pathologische Zustand bei der seinerzeitigen operativen Behandlung der Deviation hätte bemerkt werden müssen.

Für die Entstehung dieser Cyste mussten folgende zwei Möglichkeiten in Erwägung gezogen werden und zwar: Entweder wurde bei der vor drei Jahren vorgenommenen Septumresektion das eine Schleimhautblatt invertiert und in der Folge ist es dann zu einer Retentionscyste zwischen die zwei Septumblätter gekommen, oder aber muss man für die Ursache dieser Cyste den Verschluss eines Ausführungsganges einer Schleimdrüse

und eine sich daran schliessende Umwandlung der Drüse (Zarniko, Zuckerkandl) annehmen.

Nach dem chemischen Befunde, der das Vorhandensein von Liquor ausschliesst und die Flüssigkeit als Lymphe anspricht, liesse sich diese tagelang anhaltende Lymphorrhoe in der Weise erklären, dass durch die Entfernung der Septumcyste die oberflächlichen Saft- und Plasmakanälchen verletzt wurden, und dass es, da diese ja einerseits durch kleine Lücken in der Basalmembran wieder mit einzelnen Lücken zwischen den Epithelzellen, andererseits aber mit zahlreichen Lymphgefässen in Verbindung stehen, zur Eröffnung eines grösseren Lymphweges gekommen ist.

Der pathologisch-anatomische Befund spricht mehr für einen Abfluss von Liquor cerebrospinalis entlang eines Nerven. Schwalbe, Michel, Key, Retzius und Fischer konnten vom Subarachnoidealraum und vom Subduralraum aus die dichten Netze von Lymphgefässen der Nasenschleimhaut injizieren, so dass also, wie Zuckerkandl konstatierte, ein offener Zusammenhang der subarachnoidealen Räume mit der äusseren Luft besteht. Das sonderbare Bild des Nervenstämmchens im mikroskopischen Präparate würde die Annahme einer Sekretion von Liquor gerechtfertigt erscheinen lassen.

Anomalien der Sekretion der Nase gehören nicht zu den Seltenheiten und wurden schon öfters ganz enorme Mengen beobachtet, die aus der Nase abgesondert wurden. Chiari berichtet in seinem Lehrbuch über eine 35 jährige Frau, bei der ein seröser Ausfluss aus der Nase bestand, und zwar in Mengen bis zu einem Liter im Tage. Trotz der chemischen Untersuchung dieser Flüssigkeit konnte damals nicht entschieden werden, ob es sich um Lymphe oder um Liquor cerebrospinalis gehandelt hat. Einen ähnlichen Fall beschreibt Körner, bei welchem oft drei Liter täglich abgesondert wurden. Die Ursache dieser Hydrorrhoea nasalis konnte auch in diesen beiden Fällen nicht eindeutig erklärt werden.

Wie bereits eingangs erwähnt, berichten verschiedene Autoren über das Vorkommen von Cysten und pneumatischen Kavitäten in der Nase. Der Inhalt bestand entweder aus einer serösen Flüssigkeit, Schleim, Eiter oder Luft. McCullagh beschreibt eine Cyste an der Lamina perpendicularis des Siebbeines, Hellat demonstrierte eine Pat. mit einer Cyste im linken Nasenflügel vor dem vorderen Ende der unteren Muschel. Der Inhalt der Cyste war eine klare, gelbliche Flüssigkeit.

Heister und Nessi, dann Pallucci und Kessler erwähnen Fälle von Cysten in Nasenpolypen, während Chiari, Heymann, Killian, Sojo, Hall und viele andere besonders über das Auftreten von Cysten in der Schleimhaut der Nebenhöhlen der Nase berichten.

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SECTION XV

RHINOLOGY AND LARYNGOLOGY

DISCUSSION No. 2

(Jointly with Section XVI, Otology, Discussion No. 3)

THE METHODS AND RESULTS OF TREATMENT OF DISEASES OF THE THROAT, NOSE, AND EAR BY SALVARSAN AND OTHER ARSENICAL COMPOUNDS

(For Reports by Dr. André Castex and Professor Dr. P. Gerber, see Part I, pp. 35–110)

DISCUSSION

Dr. H. L. Whale (London). During the last eight months I have treated with some form of salvarsan, and followed a series of twenty cases of nasal and laryngeal syphilis. All the patients, except one, were in the tertiary stage of the disease.

The nasal cases treated may be divided into those of disease of bone or cartilage, and those of sinus disease.

Disease of bone and cartilage. In an early stage, with cardinal signs of active inflammation, but before any necrosis has occurred, the pathological process may actually abort, and the parts return to normal. Later, if local death has supervened, by giving the drug sequestration is materially assisted, and the dead tissue is much sooner ready for removal by scraping, &c. This applies, for instance, to necrosis of the ethmoid, and perforations of the septum or of the hard palate. It is important not to delay in beginning the treatment. Thus, the series includes three cases of perforation of the hard palate; in two of these the lesion was of at least two years' standing, and the drug merely checked the process; in the third, perforation had existed only one week, and within a fortnight the mucous membrane had entirely healed over.

Sinus disease. It seems that the cure of this may be hastened, but if the sinus is being irrigated at the same time, too much credit must not be claimed for the salvarsan.

Laryngeal disease. The results here are encouraging. In two out of three patients with gummatous laryngitis, a sort of pachydermatous nodule in the anterior commissure prevented adduction and the phonation

of 'Ah'. In one case after one, and in the other after three, injections, these nodules disappeared, and phonation was possible.

Deafness. Two cases were quite cured. I might have regarded them as cases of cured syphilitic deafness had I not taken the tuning-fork tests previously, and known that the deafness was due to concomitant post-nasal and Eustachian catarrh.

The Wassermann test bears no constant relation to the clinical result. In successful cases, generally the cure is complete before the Wassermann becomes negative; in such cases I have temporarily stopped the administration of the drug. When the Wassermann is negative, a 'provocative' dose of the drug will sometimes change it in forty-eight hours. Between injections of salvarsan, mercury seems to be of greater assistance than potassium iodide.

The preparation employed. As regards the results, there seems to be nothing to choose. The relative advantages of different preparations may therefore be decided on two grounds: first, the amount of local and general reaction, and second, the number of doses necessary for a cure. I speak of 'cure' irrespective of recurrence, which I have encountered only once. If we consider three classes of preparation, viz. intramuscular, '606' or emulsion; intravenous, '606'; and intravenous, '914' or neo-salvarsan; the intramuscular preparations appear to cause the greatest local and general reaction, and neo-salvarsan the least. On the other hand, intramuscular preparations are very easy to give and cure with a minimum number of injections, while neo-salvarsan requires a maximum. Neo-salvarsan may with confidence be given to out-patients, but after the seventh or eighth dose (at intervals of one week) most of the available veins may be partially or completely fibrosed, and an open dissection necessary; this is a disadvantage. On these two grounds, the reaction and the number of necessary doses, intravenous '606' may be placed intermediate between intramuscular '606', and intravenous neo-salvarsan.

The apparatus ordinarily used has certain disadvantages, to obviate which I have had an apparatus made by Messrs. Mayer and Meltzer, which will be shown on the instrument table.

Professor ALEXANDER (Wien) wünscht in der Salvarsanfrage den historischen und den gegenwärtigen Standpunkt zu unterscheiden. Für den Beginn hält er daran fest, dass die s. Zt. in dem Beginn der Salvarsan-Aera beobachteten ungünstigen Zufälle in der Quecksilber-Aera kein Analogon finden. Benarios Ausführungen haben hier zu einem Missverständnis geführt, dem auch Gerber unterlegen ist. Benario findet nur den Prozentsatz der VIII-Schädigungen bei Hg und Salvarsan gleich (im Rahmen der Affektionen der übrigen Hirnnerven), nicht aber die absolute Zahl; auf diese letztere aber allein kommt es hier an. Benario selbst gibt aber zu, dass die Salv.-VIII-Fälle häufiger sind als die Hg-VIII-Fälle. Der 'Gefahrenquotient' hat sich seit Verbesserung de

Technik, vor allem aber seit Einführung der intravenösen Injektion und der kombinierten Hg-Salvarsanbehandlung wesentlich verringert. Es ist gegenwärtig nötig, exakte Kontraindikationen aufzustellen. So ist Salvarsan kontraindiziert bei intaktem Oktavus im Stadium der Frühlues, während des Exanthems oder kurz vor Ausbruch des Exanthems, und bei frischen luetischen Labyrinthaffektionen. Hier ist Hg oder Hg-Salv. zu empfehlen.

Einen interessanten Standpunkt erhalten wir durch das Studium der tabischen Ohraffektionen. Gerade diese geben der Meinung Ehrlichs recht, dass bei Salvarsan nur die Gefahr bestehe, dass eine Labyrinthaffektion, die sonst später aufgetreten wäre, durch Salvarsan früher ausbricht. Die günstigen Einzelerfolge des Salvarsans sind unbestreitbar. Es ist zu hoffen, dass der Prozentsatz der günstig beeinflussten Fälle von Labyrinthsyphilis sich mit dem Fortschreiten der Technik der Salvarsanmedikation erhöhen wird. Auch ist zu hoffen, dass es möglich sein wird, häufiger als es derzeit der Fall ist, *Dauerresultate* zu erhalten.

Dr. Maurice (Paris). On nous a parlé des accidents provoqués par le 606 et pas du tout du traitement à leur appliquer. N'y aurait-il donc rien à faire ? Nous pouvons rapporter l'observation de nombreux malades atteints de labyrinthite syphilitique ou toxique due au 606 et qui ont obtenu la guérison ou une amélioration considérable par notre méthode de rééducation auditive ou d'exercises acoustiques. Nous glissons sur notre technique que nous avons décrite très longuement ailleurs (voir n^{os} 15, 16 et 17 de la *Revue hebdomadaire* du D^{r} Moure, 1913). Nous concluons : de même qu'une paralysie musculaire aiguë ou subaiguë, toxique ou traumatique, peut guérir spontanément ou être grandement améliorée par la gymnastique, le massage ou l'électricité, de même la surdité due au 606 ou à la syphilis peut se guérir ou s'améliorer par les exercises acoustiques pratiqués selon notre technique.

Le professeur docteur de Stella (Gand) a abandonné l'emploi du salvarsan, parce que son injection demande de nombreuses manipulations qui l'exposent à des altérations qui le rendent toxique. Il se sert uniquement de néo-salvarsan injecté dans les veines en solution concentrée, dix centimètres cubes d'eau fraîchement distillée. Il n'a jamais observé d'accidents ni immédiats ni tardifs, soit du côté de l'acoustique, soit dans les autres organes. Il a vu disparaître rapidement tous les accidents syphilitiques et le Wassermann devenir négatif. Quant à l'avenir de ces malades, il estime qu'il faut réserver cette question.

Dr. A. Lieven (Aachen) erinnert, als historisch interessant, daran, dass die Kombination von Hg, Jod und Arsen in England von Alters her als sogenannte 'Donovan's solution' gekannt und als Syphilismittel gebraucht wurde. Er hält es für absolut feststehend, dass seit der Salvarsanaera mehr Acusticusaffektionen gesehen werden als früher.

Den Grund sieht er in ungenügender Salvarsanbehandlung; seine Fälle waren fast sämtlich solche, in denen eine einmalige Salvarsandosis ohne jede weitere Therapie im Frühstadium gegeben worden war. Solche Dosen wirken provokatorisch, verwandeln den negativen oft in einen positiven Wassermann und erzeugen die gefürchteten Neurorezidive. Deswegen müssen energische kombinierte Kuren mit Hg, wie Gerber sie vorschlägt, gegeben werden. Die so behandelnden Aerzte erleben, wie die Erfahrung lehrt, keine Rezidive.

If the patient refuse repeated salvarsan injections it is better to withhold them altogether and trust to mercury alone. If this conception becomes general the occurrence of neural accidents will become as rare as they were when mercury was the only known remedy.

Dr. H. MARSCHIK (Wien). Ein Fall von luetischer Zerstörung der Nase und des Gaumens mit frischen Ulzerationen, der vergeblich mit Salvarsan und Quecksilber behandelt worden war, zeigt, dass man neben der Allgemeinbehandlung die lokale nicht vernachlässigen darf. Es wurden vier Salvarsaninjektionen und zwei Quecksilberinjektionen ohne Erfolg gemacht; die Ulzerationen vergrösserten sich immer mehr, so dass die konsultierten Dermatologen malignes Neoplasma auf luetischer Basis annahmen. Mikroskopische Untersuchung verschiedener Probeexzisionen war immer negativ und zeigte nur Granulationsgewebe. Endlich applizierte Redner Arg. nitr. in Substanz, worauf die Ulzerationen, die bereits die Oberlippe perforiert hatten, innerhalb zwei Wochen geheilt waren, so dass eine totale Rhinoplastik gemacht werden konnte.

Professor E. V. SEGURA (Buenos Ayres). I have had opportunities of observing several cases of labyrinthine complications after the use of salvarsan. Among these was a married couple. The husband had contracted a chancre and infected his wife. Both were treated with salvarsan by a distinguished syphilologist. Two months after the injections both developed grave symptoms of labyrinthine disease. Vertigo, noises, deafness, and vomiting set in. The husband consequently was almost in a state of insanity, and his wife had so much vomiting and dizziness that she had to be kept in bed for a fortnight. After the usual treatment the husband became better, his hearing being completely restored, but his wife did not improve, and almost entirely lost her hearing.

Professor BRÜHL (Berlin) stimmt mit Alexander überein, dass seit der Salvarsanbehandlung Labyrinthaffektionen viel häufiger zu beobachten sind als früher. Dies ist um so verhängnisvoller, als nach seinen Erfahrungen eine Besserung des Gehörs in solchen Fällen durch erneute Injektionen nicht sicher zu erwarten ist. Ob dann die Erkrankung selbst etwa durch die Injektion mobil gemacht, oder *toxische* Wirkung der Einspritzung Ursache der dauernden Hörstörung ist, bleibt für den Patienten ganz gleich, und da er in *schweren* alten Fällen hereditärer

oder erworbener Lues der Hörstörung oder gar Taubheit keinen Erfolg mit Salvarsan gesehen hat, in frischeren aber auch mit Quecksilber und Jod gute Erfolge bekam, so hält er, ungeachtet der unbezweifelbar ausgezeichneten Wirkung des Salvarsans für andere Affektionen, bei Behandlung der *Labyrinthsyphilis* mit Salvarsan Vorsicht am Platz.

Sir Robert Woods (Dublin) mentioned two of his cases which demonstrated the utility of salvarsan. The first was that of a boy, who had been under his observation for fourteen years, suffering from severe congenital syphilis of the nose and throat, with destruction of nearly all the bones of the nose and a large part of the soft tissues. He had during these years been treated by mercury, iodide of potassium, arsenic, quinine, &c., but only with temporarily good results. He returned with a new patch of ulceration or necrosis on an average every three months. The speaker injected salvarsan in November 1910, and since that time the patient had been free from fresh symptoms. His general health had so much improved that it was possible to perform a plastic operation for restoration of the nose with every success.

The second case was that of a sailor, aged 22, who early this year was brought ashore from his gunboat suffering from giddiness, deafness, and vomiting. He was in the first instance taken to an emergency hospital and after six days transferred to the speaker's care in Sir Patrick Dun's Hospital. He was found to be absolutely deaf in both ears. Inquiry elicited the history of a precisely similar attack five years previously, which resulted in complete deafness of the right ear. The teeth, though not of the typical Hutchinson variety, were irregular and suggested the possibility of congenital syphilis. An injection of salvarsan was given with the result that within forty-eight hours the hearing in the left ear had begun to improve, and was almost completely restored within a month. Wassermann was found to be weakly positive. The case was interesting as it was really a differential experiment, and one could hardly doubt that had it not been for the salvarsan injection the left ear would have followed the same course as the right, and the man would have been stone deaf.

Professor Finder (Berlin) berichtet über den Fall eines 43 jährigen Mannes, der eine sehr bösartig verlaufende Lues hatte und von anderer Seite bereits zweimal mit Kombination von Salvarsan und Quecksilber behandelt worden war, mit dem Effekt, dass die gerade vorhandenen Erscheinungen prompt zurückgingen. Er kam zu ihm mit einem Gummi des harten Gaumens, das eben im Begriff war zu zerfallen. Patient war heiser ; er behauptete, dies schon seit langer Zeit zu sein ; er war Lehrer und musste seine Stimme professionell viel anstrengen. Die Larynxschleimhaut war im ganzen stark gerötet ; es bestand jedoch nicht das geringste Zeichen von Stenose. Patient erhielt eine intravenöse Injektion von 0,6 Gramm Salvarsan. Am folgenden Tage

starke Dyspnöe. Man sah eine starke subglottische Schwellung, die aussah, als ob es sich um eine Perichondritis cricoidea handelte. Der Redner kann sich des Eindrucks nicht erwehren, dass ein ursächlicher Zusammenhang bestand zwischen der Injektion und der subglottischen Schwellung. Es musste die Tracheotomie gemacht werden. Er fragt an, ob ähnliche Fälle beobachtet sind ; sie werden eventuell zur Vorsicht bei Anwendung von Salvarsan bei Larynxaffektionen ermahnen.

Dr. DUNDAS GRANT (London). Among the most interesting points and conclusions related by Professor Gerber is his recommendation that in cases of syphilitic laryngeal stenosis, tracheotomy should not be performed without a previous injection of salvarsan. An illustrative case has recently come under my care.

I have seen several cases of necrosis, and to my surprise I was informed by the patients that they had had treatment by injections of salvarsan. This was possibly a mere coincidence, but I venture to ask whether others have had similar experience. Perhaps mercurial treatment had been neglected and too much reliance placed on the salvarsan.

Dr. DAN MCKENZIE (London) gave an account of the experiences at the Central London Throat and Ear Hospital in cases under treatment by the staff. In most of the cases—indeed in all the recent cases—treated by salvarsan the results had shown immediate and dramatic improvement, especially in extensive nasal and pharyngeal ulceration.

Cases which had proved refractory to the older method of treatment responded rapidly to the action of the drug. In one case under his own care there was great destruction of the nose—septum, turbinals, antral wall, and ethmoidal region, with great flattening of the organ. For months treatment by iodine and mercury had been tried with little or no result. Salvarsan was then given in two separate doses, and in less than a month after the speaker was able to remove a large sequestrum of the outer wall of the nose.

In spite of Dr. Finder's experience, the treatment of laryngeal hyperplasia and gummatous ulceration was undertaken without fear of the onset of œdema of the larynx, which was so much dreaded when giving iodide of potassium in such cases.

For these reasons, in his opinion, the dangers of salvarsan, such as they were, were well worth risking in order to bring to a standstill the deforming and disfiguring destruction that attended syphilis of the nose. Further, it was obvious that as long as those gratifying results were obtained, even although they might be transitory, salvarsan or its related remedies would occupy an important place in the treatment of nasal and pharyngeal syphilitic ulceration.

With regard to the ear, the speaker had had no experience. He had not yet tried the drug in syphilitic deafness, and did not feel inclined to do so after what had been said that day. Further, on theoretical grounds

there were objections, and in this connexion he drew attention to the Herxheimer reaction of the eighth nerve. In the skin, a Herxheimer was transitory in action and left no ill-effects, but the occurrence of a Herxheimer swelling in a delicate nerve-trunk already slightly damaged by disease was obviously liable to be followed by permanent and serious nerve-atrophy.

Dr. Kœnig (Paris). I had a case a year ago which would tend to show that the curability of syphilitic labyrinthitis is more a question of degree and intensity of the infection than one of recency or chronicity. The patient contracted syphilis in the Philippine Islands and immediately received a single injection of salvarsan. One year after, in Paris, he became *suddenly*, completely deaf, with vertigo. I put him into the hands of Dr. Leredde for salvarsan treatment without the slightest result, and the patient returned to America completely deaf.

Reply

Dr. Castex (Paris). J'ai voulu surtout montrer, dans mon rapport, que le salvarsan est parfois dangereux pour l'audition. Mais déjà le néo-salvarsan semble moins nuisible. C'est comme otologiste que j'ai considéré la question.

Professor Gerber (Königsberg i. Pr.). Auf die Ausführungen des Kollegen De Stella möchte ich erwidern, dass die von ihm angeführte Injektionsmethode, wenn ich sie verstanden habe, sich mit den von Duhot, Stern u. a. angegebenen wohl decken dürfte. Auch wir haben diese Injektionsmethode, allerdings bisher nur in wenigen Fällen, angewendet, und waren mit ihr zufrieden. Wenn sie sich auch weiterhin bewährt, so stellt sie eine Vereinfachung des Verfahrens dar.

Herrn Kollegen Lieven muss ich durchaus darin recht geben, dass verzettelte Dosen von Salvarsan sehr gefährlich werden können ; das beweist eben auch der Fall von Kœnig.

Den Herren Kollegen Brühl und McKenzie möchte ich erwidern, dass die veralteten spezifischen Affektionen des innern Ohres bisher doch eigentlich für die Therapie eine verlorene Provinz bedeuteten, und man daher *hiergegen* auch vom Salvarsan nicht zuviel erwarten dürfte. Erfolge, und zwar erhebliche, sind aber auch auf diesem Gebiet erzielt, wie aus der Literatur und eben noch aus den sehr dankenswerten Bemerkungen des Kollegen Alexander zu ersehen ist.

Zu dem Falle Finders bemerke ich, dass solche Schwellungen im Larynx nach Salvarsan hin und wieder, aber selten, beobachtet worden und als Herxheimer'sche Reaktion aufgefasst worden sind. Uebrigens muss man bei schon mit Arsenpräparaten vorbehandelten Fällen vorsichtig sein. Was das Salvarsan im Larynx leistet, zeigt ja auch der Fall von Dundas Grant.

Fasse ich die Resultate unserer Diskussion zusammen, so müssen wir zunächst obere Luftwege und Gehörorgan trennen. In Hinsicht der ersteren herrscht jetzt doch wohl keine Differenz mehr. Wir besitzen im Salvarsan für die spezifischen Affektionen wie auch für die lokalen Spirochaetosen des Halses und der Nase geradezu ein *Zaubermittel*. Aber auch hinsichtlich des Gehörorgans bedeutet das Ehrlich'sche Mittel eine entschiedene Bereicherung.

SECTION XV

RHINOLOGY AND LARYNGOLOGY

INDEPENDENT PAPER

DIE BEHANDLUNG DER NARBENSTENOSEN DES OESOPHAGUS MIT RADIUM

Von Dr. FRIEDRICH NEUMANN, Assistent der k.k. Universitäts-klinik für Kehlkopf- und Nasenkrankheiten in Wien (Vorstand: Hofrat Professor Dr. O. CHIARI)

Meine Herren! Um Ihre kostbare Zeit nicht allzu sehr in Anspruch zu nehmen, werde ich mich möglichst kurz fassen. In erster Linie will ich Ihnen das Stenoseninstrumentarium unserer Klinik vorführen und ihre Anwendungsweise kurz besprechen.

Als erstes Prinzip an unserer Klinik gilt die Inspektion der Stenose mit dem Oesophagoskop und die Einführung einer Sonde in das vorhandene Lumen. Nur in einem Falle waren wir nicht imstande, ein Lumen zu finden, wir mussten den Patienten mit seiner Gastrostomie wieder entlassen. Als erste Sonde gebrauchen wir gewöhnlich das Instrument von Lerche [1] (Fig. 1), an dessen Ende eine zarte Olive (Fig. 1 *c*) angebracht ist, das Lerche'sche Instrument hat den Vorteil, dass es infolge seiner Beweglichkeit auch leicht in eine exzentrische Stenose eingeführt werden kann. Sie sehen hier das Instrument von Lerche (Fig. 1). Seine Handhabung lässt sich leicht erklären. Drücke ich hier an den Hebel (Fig. 1 *a*), so wird der Draht (Fig. 1 *b*) gespannt und die Spitze bezw. die Olive des Instrumentes (Fig. 1 *c*) bewegt sich aus der Längsachse des Instrumentes heraus und erleichtert so die Einführung in ein exzentrisch gelegenes Lumen. Erwähnt sei noch, dass wir bei endo-oesophagealen Eingriffen, bei welchen sich eine Verletzung der Schleimhaut erwarten lässt, den Patienten die Tage vorher Formamint essen lassen und unmittelbar vor dem Eingriffe (nach der Anästhesierung mit Kokain) mit Hydrogenum hyperoxydatum das Operationsfeld sterilisieren.

Ist die Oesophagusstenose soweit, dass man mit dem Lerche'schen Messer dieselbe entrieren kann, so tritt die Oesophagotomie interna in ihre Rechte. Sie sehen hier, meine Herren, das Lerche'sche Messer (Fig. 2). An Stelle der früheren Endolive ist eine Bougie filiforme (Fig. 2 *a*)

[1] Lerche, *Surg., Gyn., and Obstet.*, 1910.

angebracht. Hinter derselben folgt ein auswechselbares Messer (Fig. 2 *b*) von 1–3 mm Breite. Nach Anästhesierung und Anämisierung und Desinfektion der Stenose wird das Messer unter Leitung des Auges in die Stenose eingeführt und unter Leitung des Auges die Narbe diszindiert an einer oder mehreren Stellen je nachdem das Lumen exzentrisch oder zentral gelegen ist. Dadurch dass man den Hebel des Griffes anzieht, wird das Messer gegen die Narbe gepresst und so dieselbe eingeschnitten. Die Zartheit des Lerche'schen Instrumentes gestattet es, den dabei ausgeübten Druck genau zu nüanzieren. Nach der Inzision legt man, um die Blutung zu stillen, Tupfer mit Hydrogenum hyperoxydatum ein, tuschiert dann die Wundfläche mit 5 prozentigen Lapis und legt nun, noch immer unter Leitung des Auges, eine konische Bougie ein, um ein sofortiges Verkleben der Wundränder zu verhindern. Ueber die Bougie hinweg wird das Oesophagoskop entfernt und die Bougie ungefähr 10 Minuten in der Stenose gelassen. Das dadurch erreichte Lumen hat gewöhnlich einen Durchmesser von 5–8 mm. Man muss nun einige Tage abwarten, während welcher Zeit der Patient nur gekochte Nahrung und viel Formamint zu sich nimmt. Gastrostomierte Patienten bekommen überhaupt keine Nahrung per os in dieser Zeit, sondern nur Formamint. Ist ein eventuell post operativ aufgetretenes Fieber geschwunden, so kann man mit der progressiven Dilatation beginnen, welche mit sehr dünnen konischen Bougies begonnen wird. Um nun ein rascheres Vorgehen zu ermöglichen, hat Marschik[1] einen Dilatator (Fig. 3) an einer biegsamen Sonde konstruiert, den man unter Leitung des Auges in die Stenose einführen kann. In der Stenose wird nun der Dilatator geöffnet und durch eine Schraube (Fig. 3, *a*) in der geöffneten Stellung erhalten. Ein Zeiger am Griff des Dilatators zeigt an, wieweit er geöffnet ist, man kann jetzt den Griff abnehmen und den Oesophagoskoptubus über

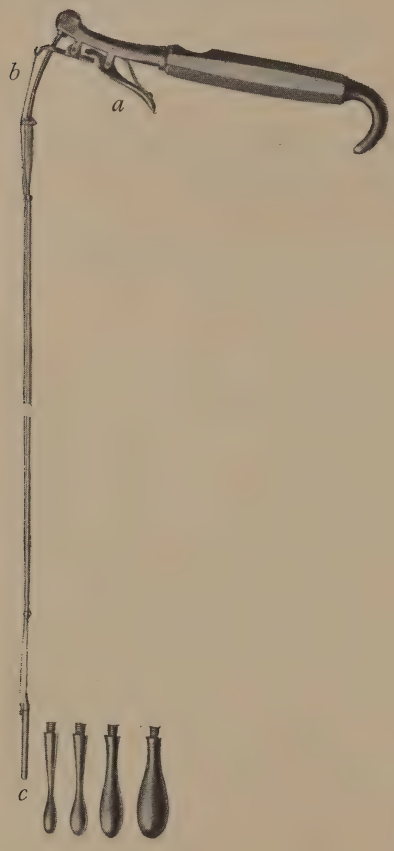


Fig. 1.

[1] Marschik, 'Behandlung der Narbenstenosen des Oesophagus,' *Monatsschrift für Ohrenheilkunde und Laryngo-Rhinologie*, 1913, II. Heft.

die Sonde des Dilatators entfernen. Weitere Behelfe zur Dilatation der Narbenstenosen sind das Fibrolysin und die Elektrolyse. Da wir nach Fibrolysin manchmal lokale Phlegmonen hatten, wird dasselbe an unserer Klinik jetzt nicht mehr verwendet, da ausserdem die Wirkung des Fibrolysins bei Oesophagusstenosen im Stiche lässt. Die Elektrolyse wird so ausgeführt, dass eine Metallolive an einer leitenden

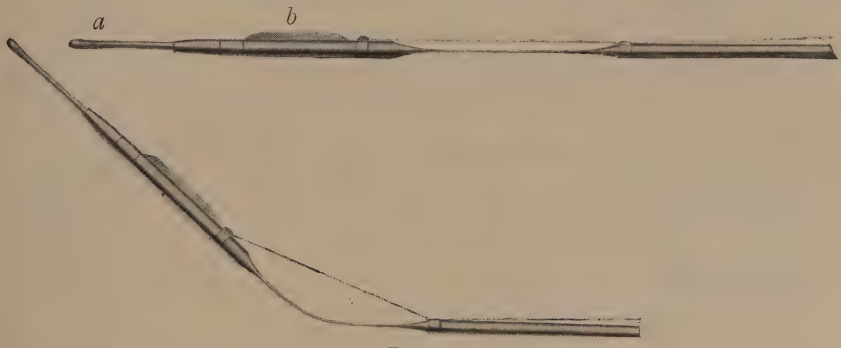


Fig. 2.

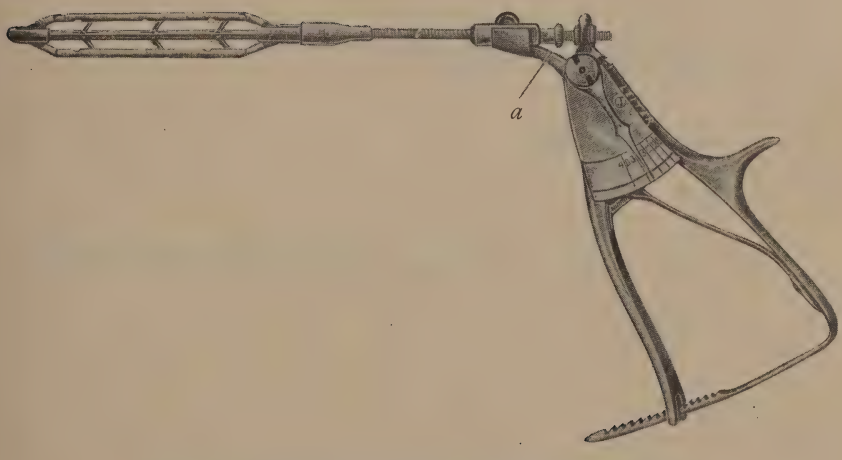


Fig. 3. Biegsamer Dilatator nach Marschik-Leiter.

Schlundsonde als negative Elektrode eingeführt wird, während der positive Pol als Plattenelektrode dem Patienten auf die Brust gelegt wird. Schon nach einigen Sitzungen, von welchen jede ungefähr 10 Minuten dauert und wobei ein Strom von 2 Milliampère in Anwendung gelangt, ist ein sichtlicher Erfolg vorhanden. Um ein Verschieben der Olive in der Stenose unmöglich zu machen, hat Kofler diese Endstücke in verschiedener Grösse und in Biskuitform anfertigen lassen (Fig. 4). An der Leitungssonde, an welcher diese Endstücke befestigt sind, ist ausserdem ein Zentimetermass eingezeichnet, so dass man sich während der Sitzung von dem richtigen Stand der Elektrode immer überzeugen

kann. Der Erfolg dieser elektrolytischen Behandlung ist leider nur temporär. Durch die Elektrolyse wird Gewebe zerstört, so dass diese Behandlung abermals eine Narbenbildung im Gefolge hat.

Mit allen diesen hier angeführten Methoden erzielt man momentan sehr schöne Erfolge, aber man erzielt keine Dauererfolge. Durch die Publikationen von Wickham und Degrais[1] angeregt, versuchte ich durch Radiumapplikation die stenosierenden Narben des Oesophagus zum Schwinden zu bringen, wie die obengenannten Autoren Narben der äusseren Haut zum Schwinden brachten. Zu diesem Zwecke liess ich mir eine 3 cm lange und im äusseren Querdurchmesser 6 mm dicke

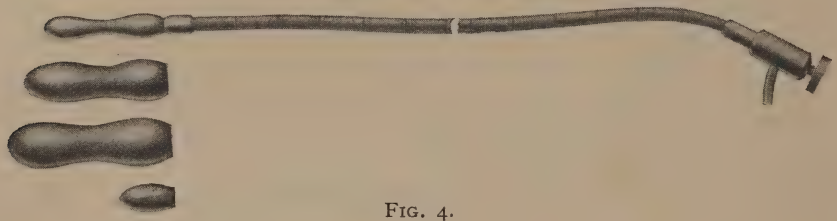


Fig. 4.



Fig. 5.

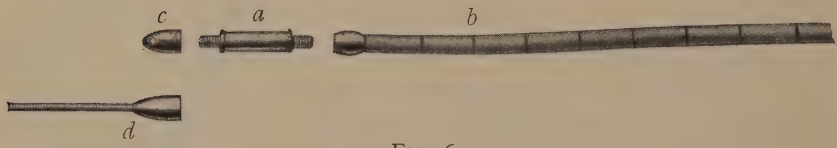


Fig. 6.

Nickelkapsel (Fig. 5, *a*) anfertigen, die einen 8 mm dicken abschraubbaren Kopf trägt. An diesem Kopf ist ein dünnes 50 cm langes Drahtseil befestigt. Diese Nickelkapsel dient zur Aufnahme des Dominici-Röhrchens (Fig. 5, *b*) ; dasselbe ist ein Radiumträger, dessen Inhalt 18 mgr Radiummetall entspricht. Seit Juli 1913 steht obiger Klinik ein Dominici-Röhrchen mit 30 mgr Radium zur Verfügung und ferner ein von meinem Chef Herrn Hofrat Chiari angegebener Träger für den Oesophagus (Fig. 6, *a*). Derselbe besteht aus einer 4 mm dicken Kapsel, aus 0,15 mm dicken Platin-Blech ; der Inhalt dieser Kapsel entspricht 10 mgr Radiummetall. Diese Radiumkapsel endet beiderseits in eine Schraube, welche zur Befestigung einer dünnen Bougie (Fig. 6, *b*) und eines olivenförmigen Endstückes (Fig. 6, *c*) oder einer Bougie filiforme

[1] *Die Radiumtherapie von Wickham und Degrais.*

(Fig. 6, *d*) dient. Diese dünne Kapsel bietet den Vorteil, dass man sie schon bei sehr kleinem Lumen (4 mm) applizieren kann. Es stehen gegenwärtig einige Fälle in Behandlung mit Narbenstenosen des Oesophagus. Bei zwei Fällen ist die Behandlung bis auf weiteres abgeschlossen und über diese will ich kurz berichten.

M. A., ein 17 jähriges Mädchen, trank vor fünf Jahren Laugenessenz. Fünf Monate später traten die ersten Stenoseerscheinungen auf. Seit $4\frac{1}{2}$ Jahren wird das Mädchen an verschiedenen Stationen ambulatorisch mittelst Bougies behandelt. Als das Mädchen am 20. April 1913 in unsere ambulatorische Behandlung trat, konnte ich mittelst Oesophagoskopie 20 cm unter der oberen Zahnreihe eine punktförmige Stenoseöffnung entdecken, welche ich mit dem Lerche'schen Instrument soweit erweitern konnte, dass es mir nun unter Leitung des Auges gelang, die Nickelkapsel (Fig. 5, *a*) mit dem Dominici-Röhrchen (18 mgr) in die Stenose einzuführen. Eine halbe Stunde konnte die Kapsel in der Stenose belassen werden, nach dieser Zeit bekam Pat. Schmerzen und einen derartigen Würgreiz, dass ich die Kapsel entfernen musste. Eine Stunde nachher fühlte Pat. keinerlei Schmerzen, blieb fieberfrei und konnte flüssige und breiige Nahrung leicht schlucken. Am 28. April ergab die oesophagoskopische Untersuchung ein Lumen von ungefähr 2 mm Durchmesser. Es gelang leicht ohne Schmerzen und mit ganz geringer Blutung, die Stenose mit konischen Bougies zu dilatieren und die obenerwähnte Nickelkapsel in das Lumen hineinzubringen und daselbst eine halbe Stunde zu belassen. Da Pat. schmerz- und fieberfrei blieb, wurde am nächsten Tag, d. i. am 29. April, die Radiumkapsel abermals auf eine halbe Stunde angelegt. Am vierten Tage nach der letzten Applikation, d. i. am 3. Mai, bekam Pat. Schmerzen in der Stenosengegend, konnte nur mit Mühe flüssige Nahrung zu sich nehmen. Fieber bestand keines. Am 10. Mai waren bereits die Schmerzen geschwunden, Pat. konnte aber noch immer keine dickbreiige Nahrung zu sich nehmen. Die am 14. Mai vorgenommene Oesophagoskopie ergab 3 cm ober der Stenose ein zirkuläres Oedem der Oesophagusschleimhaut. Bei dem Versuch, die Tube No. 8 hindurchzudrängen, tritt leichte Blutung ein, weshalb der Versuch aufgegeben wird. Nach dieser Oesophagoskopie blieb Pat. schmerz- und fieberfrei. Am 19. Mai gibt die Pat. an, sie fühle die Speiseröhre sei weiter, sie konnte tatsächlich fein gekautes Brot und Gemüse schlucken, Pat. nährt sich jetzt mit breiiger Nahrung, faschiertem Fleisch etc. Am 24. Mai abermalige Oesophagoskopie. Die Stenose ist etwas mehr als 6 mm weit, der sonst weisse, narbige, scharfe Rand erscheint rot und abgerundet, unmittelbar ober und unter der Stenose ist die Oesophagusschleimhaut noch immer leicht oedematös. Die Radiumkapsel wird abermals appliziert. Der dünne Teil der Kapsel fällt förmlich in das Lumen hinein, so dass die Kapsel an ihrem Kopfe hängt, sie bleibt abermals eine halbe Stunde in der Stenose. Pat. bleibt schmerz- und fieberfrei. Zwischen 29. Mai und 3. Juni hat Pat. beim Schlucken manchmal leichte Schmerzen, Fieber tritt nicht ein. Pat. nimmt jetzt normale Kost zu sich, muss aber dieselbe sehr fein zerkauen. Am 9. Juni ergibt die Oesophagoskopie, dass die Stenose bereits so weit geworden ist, dass man mit der Oesophagoskopietube No. 8 (8 mm) durch die Stenose hindurchgehen kann, um die tieferen Partien des Oesophagus zu inspizieren. Oberhalb der Cardia sieht man einige längsverlaufende Narbenzüge, welche das Lumen aber nicht einengen. Bei dem Versuch, abermals die Radiumkapsel in der Stenose zu plazieren, gleitet dieselbe durch die Stenose hindurch. Der Kopf der Kapsel muss

mit einem Kautschukring versehen werden, um ein Hindurchgleiten zu verhindern. Dauer der Applikation eine halbe Stunde. Die Oesophagoskopie am 25. Juni ergibt, dass die Stenose fast 10 mm weit ist und keine Entzündungserscheinungen zeigt. Die stenosierende Falte ist rötlich und sehr flach. Am 14. Juli wird derselbe Befund erhoben.

Bei dieser Patientin habe ich also im ganzen $2\frac{1}{2}$ Stunden Radium appliziert und zwar 18 mgr gefiltert durch 0,1 mm dickes Silberblech und ein 0,2 mm dickes Nickelblech. Die Erweiterung der Stenose nach den drei ersten Applikationen ist sicherlich zum Teil auch auf die mechanische Dilatation zurückzuführen, der Erfolg nach den zwei letzten Applikationen aber ist reine Radiumwirkung, denn bei den zwei letzten Applikationen konnte die Radiumkapsel ohne Gewalt eingeführt werden. Die Schluckbeschwerden und die temporäre Verengerung zu Anfang Mai ist jedenfalls als Reaktion auf die Applikation vom 20., 28. und 29. April zu deuten. Dasselbe gilt für die Schmerzen, die zwischen 29. Mai und 3. Juni auftraten.

Der zweite Patient war ein 20 jähriger Schlosser, E. S., der im Januar 1913 Laugenessenz trank. 14 Tage später bestand vollkommener Verschluss der Speiseröhre, der nach weiteren zwei Tagen soweit zurückgegangen war, dass Pat. flüssige Nahrung zu sich nehmen konnte. Bei seiner Aufnahme am 3. Juni konnte Pat. dünnbreiige Nahrung schlucken. 17 cm unter der oberen Zahnreihe konnte ein 2 mm grosses Lumen umgeben von narbigen Falten gesichtet werden. Dilatation der Stenose mit Lerche und Einführung der Bougie No. 7. Applikation des Dominici-Röhrchens in der Nickelkapsel $1\frac{1}{2}$ Stunden. Am 20. Juni Radiumapplikation eine Stunde, 28. Juni eine Stunde, 10 Minuten. Am 29. Juni starke Schmerzen, starker Speichelfluss. Nach den ersten drei Applikationen blieb Pat. schmerzfrei. 1. Juli sehr heftige Schmerzen. Pat. kann nicht schlucken. Temperatur 38°. Am 14. Juli Versuch die Radiumkapsel einzuführen. Diese gleitet jedoch stets heraus. Beim abermaligen Versuch hat Pat. so heftige Schmerzen, dass die Oesophagoskopie unterbrochen wird. Das Lumen der Stenose ist 4 mm im Durchmesser. Am 17. Juli Pat. klagt über Schmerzen im Bereiche des rechten Recessus piriformis. Pat. kann seit zwei Tagen nicht schlucken. Die Oesophagoskopie ergibt, dass die Stenose ungefähr 7 mm weit ist, die einengenden Narbenzüge und narbigen Falten sind leicht gerötet, es gelingt unter mässigem Druck die Tube No. 8 durch die Stenose hindurchzuführen. Man gelangt dabei auf eine zweite Stenose, 25 cm hinter der Zahnreihe, welche ungefähr 2–3 mm im Durchmesser hat, durch diese zweite Stenose wird eine Bougie eingeführt, welche in einer Höhe von 38 cm auf einen Widerstand stosst, jedenfalls eine tiefe Stenose, welche mit der Sonde nicht passiert werden kann. Da der Pat. infolge dieses tiefsitzenden Verschlusses des Oesophagus keine Nahrung zu sich nehmen kann, musste an dem Pat. die Gastrostomie vorgenommen werden.

Wenn wir mit der Radiumbehandlung bei diesem Patienten auch keinen endgültigen Erfolg erzielten, da er eine tiefsitzende Stenose im Oesophagus hatte, welche erst nach der Erweiterung der ersten Stenose festgestellt werden konnte, und welche uns zwang, bei dem Patienten eine Gastrostomie anzulegen, so haben wir doch bei dem Patienten die

hochliegende Stenose (17 cm hinter der oberen Zahnreihe) von 2 mm auf 7 mm Durchmesser erweitert. Wir haben bei diesem Patienten im ganzen 3 Stunden 40 Minuten Radium appliziert, ohne davon schwere Nachteile gesehen zu haben. Die Radiumapplikation erstreckte sich mit Pausen vom 10. bis 28. Juni.

Der Vorteil der Radiumbehandlung ist der, dass die Wirkung, obwohl sie einige Tage später eintritt, scheinbar eine dauernde ist. Die bis jetzt gewonnenen Erfolge regen zu weiteren Versuchen an. Die Frage, wann man eine Stenosenbehandlung des Oesophagus beginnen soll, wurde früher immer dahin beantwortet, dass man sie nicht vor dem sechsten Monat beginnen soll. Ein Patient unserer Klinik zeigt aber, dass auch diese Zeit noch viel zu kurz bemessen ist. Die Krankengeschichte folgt in kurzem:

K. G., 6jähriges Kind, aufgenommen am 26. März 1913. Pat. trank aus Versehen vor sechs Monaten Laugenessenz. Seit dieser Zeit kann Pat. keine festen Speisen schlucken, flüssige Nahrung kann Pat. zu sich nehmen. Am 31. März 1913 Oesophagoskopie in Narkose. 12 cm hinter der oberen Zahnreihe eine Stenose mit einem Lumen von 2 mm im Durchmesser. Von dem Lumen gehen gegen die dorsale und linke Oesophaguswand strahlige Narben aus. Es gelingt, ein konisches Bougie durch das Lumen hindurchzuschieben, welches in den Magen gelangt. Die Stenose lässt sich mit konischen Bougies soweit erweitern, dass man die Oesophagoskopietube No. 8 in die Stenose einführen kann und bis in den Magen des Kindes gelangt. Der Pat. war sowohl vor der Oesophagoskopie als nach derselben fieberfrei. Die Oesophagoskopie wurde um 10^h vorgenommen, um 12^h hatte sich der Knabe von der Narkose erholt gehabt, spielte sich im Bette mit seinen Spielsachen und äusserte keinerlei Schmerzen. Zwischen 7 und 8^h abends hustete Pat. einigemale sehr heftig, begann zu schreien, wurde cyanotisch und starb binnen wenigen Minuten. Der Obduktionsbefund ergab: Narbige Stenose des Oesophagus in seinem oberen Drittel, oberhalb der Stenose beginnt ein 3–4 cm langer schlauchförmiger Kanal mit auskleidendem Granulationsgewebe, der in einem mächtigen perioesophagealen Lungenabszess führt. Dieser Abszess hat das Zwerchfell usuriert und zeigt ausserdem einen Durchbruch in die linke Pleurahöhle. Linker Pyopneumothorax. Rechter Pyothorax. Kompressionsatelektase der linken Lunge. Partielle Atelektase der rechten Lunge. Kompression der Hauptbronchien durch den Abszess. Sekretstauung in der Lunge. Bronchitis.

Obwohl dieser Patient bei seiner Aufnahme fieberfrei war und den Eindruck eines gesunden Kindes machte, zeigte er in seinen thorakalen Organen so pathologische Veränderungen, die sicherlich Monate lang bereits bestanden. Fig. 7 zeigt das pathologische Präparat dieses Patienten.

Ich wollte mit diesen Worten nicht eine geschlossene Arbeit Ihnen vorführen, sondern Ihnen nur die Anwendungsweise des Radiums auf narbige Oesophagusstenosen vorführen. Zu gleicher Zeit habe ich mir erlaubt, Ihnen von dem kleinen Patienten zu berichten, dessen pathologisches Präparat ich Ihnen soeben demonstriert habe und welches

uns zeigt, dass, obwohl bereits sechs Monate nach der Laugenverätzung vergangen waren, der Patient keinerlei Krankheitssymptome aufwies,



Fig. 7.

dennoch schwere Veränderungen vorhanden waren. Die Oesophagoskopie hat sicherlich nicht den Tod des Patienten bewirkt, sie kann ihn zwar beschleunigt haben, denn der Patient hat einigemale nach der Oesophagoskopie kräftig gehustet und infolge dieser Hustenstösse dürfte

die Perforation des bereits vom Eiter usurierten Zwerchfells und die Perforation des Abszesses in die Pleura und so der Pyopneumothorax zustande gekommen sein. Die Obduktion, die vom Herrn Dozenten Wiesner 14 Stunden nach dem Tode des Patienten vorgenommen wurde, zeigte keinerlei frische Verletzungen.

DISCUSSION

Professor KUBO (Japan). Bei der narbigen Stenose, besonders nach einer chemischen Verätzung, habe ich eine systematische Bougierung nach Magenfistelanlegung durch Oesophagoskopie probiert. Dazu habe ich eine Reihe von Bougies aus Celluloid und Weichgummi konstruieren lassen, die an mehreren Stellen sanduhrförmige Verdickung von allmählich steigernder Grösse haben. Vier Fälle wurden in dieser Weise behandelt ; davon sind 2 ganz geheilt und 2 befinden sich noch in Behandlung. Blinde Sondierung ist sehr gefährlich. Ich habe in einem anderen Falle bei Obduktion eine Taschenbildung an der stenosierten Stelle gesehen. Der Taschengrund wurde bei einer blinden Sondierung mit Olivenbougie von einem anderen Arzt perforiert, was eine eitrige Mediastinitis und Pyothorax verursachte und dem betreffenden Kranken Todesunglück brachte.

SECTION XV

RHINOLOGY AND LARYNGOLOGY

INDEPENDENT PAPER

POST-CRICOID CARCINOMA

By E. B. WAGGETT, Surgeon, Charing Cross Hospital, London

Mr. President,

As a member of the Committee I wish to express the regret we all feel that this discussion is not being opened by one of our foreign confrères. During the course of protracted correspondence upon the subject with certain continental authorities it became necessary for the honorary secretaries to complete their programme at a day's notice, and it was thought fit to associate with Mr. Trotter a member of the Committee who was in part responsible for the inclusion of the subject in our programme.

My object in suggesting the subject of post-cricoid carcinoma had a definite and practical purpose. In this country, and I presume in all countries, we are constantly seeing patients with carcinoma in the lower pharynx who come to us at a stage when hope of cure by operation is out of the question. In a large number of instances the patient fails to present him or herself to the surgeon until dysphagia of severe degree has developed, but in the majority this is not the case, and inquiry shows that palliative treatment has been employed by one or more medical advisers for many months. It is my hope that the discussion this afternoon will result in dispelling from the minds of the profession at large the idea too commonly held, that all these cases are fit only for a 'noli me tangere' attitude.

In the first place it may be asked: Is this disease sufficiently common to be worthy of serious discussion? I have spent a good deal of time in searching the literature and have come to the conclusion that statistical data of real value are not available on a large scale. It is quite evident that in many tables no distinction is made between growths originating at or below the œsophageal ostium and those which are at the outset genuinely post-cricoidal. For example, F. W. Wright's collection from fifty years' records of the Cancer Research Laboratories at the Middlesex Hospital (vide *Archives of the Middlesex Hospital*, 1906), comprising 275 examples of primary cancer of the lower pharynx and œsophagus, include only six examples of pharyngeal cancer. On the other hand,

a careful study of 164 cases of malignant disease in the larynx, lower pharynx, and œsophagus very generously made for me by my friend Mr. Whale at St. Bartholomew's Hospital, shows no less than 19 cases which fall within our category. All these occurred at St. Bartholomew's within five years, and are those only which are recorded in the Surgical Registrar's in-patient reports. Data so diverse as these are irreconcilable, and I am confident that a very large number of primary pharyngeal cases are not recorded as such, and I have therefore elected to make no statistical table, believing that such a table would be merely misleading. The practical question is this : Do these cases form an appreciable element in laryngological practice ? The answer must certainly be in the affirmative. Without exception, every laryngologist whom I have interrogated upon the subject has replied that these cases amount to a notable and a very depressing element in his practice. I have myself four of them on my hands at the moment of writing.

Secondly it must be asked : Are these cases worthy, on practical grounds, of serious discussion ? It is my theme to give an answer in the affirmative, and for the reasons I have already mentioned I will ask you to allow me to avoid elaborate statistical data and to appeal to the personal experience of confrères present this afternoon.

At the present time the majority of cases come to us when dysphagia and cachexia have fully developed, and even on general grounds the subjects are not such as we should choose for any large operation ; but these unfavourable conditions are not inherent to and essential of the disease. In all cases we get a fairly long history, often fully twelve months, of slight discomfort in swallowing which by no means interferes with the enjoyment of a full diet. Furthermore, quite a large proportion, if not the majority, of patients are youngish women. In one table of six women I find the average ages of four of them to be under 34, and two of my own operation cases have been 30 and 47 respectively. Trotter, in his *Hunterian Lectures* (1913), goes so far as to say that the majority of genuine hypopharyngeal cases occur in young and middle-aged women, and this corresponds with the opinion entertained by a number of British laryngologists. At all events, it may be claimed that, local considerations apart, the disease is apt to occur in favourable subjects, and certainly in a class which it is worth while making an effort to save : it is by no means a disease of the old and worn out. Moreover, even when the growth is extensive, the anatomical disposition of the parts favours the retention of the power of efficient swallowing and therefore of nutrition. I have more than once seen the pharynx almost encircled with growth and the patient swallowing solid food, the absence of functional obstruction obviously being due to the presence of the rigid anterior wall of this portion of the pharynx. In my experience, pain, even in fairly advanced cases, is not a marked feature, far less so than in disease of the laryngeal vestibule ; and, as a rule, it is not until the œsophageal ostium is invaded that intense dysphagia and consequent inanition set in.

The question of the degree of malignancy exhibited by these growths is all-important. I believe that all the true post-cricoid cases are squamous epitheliomata; certainly this is true of all I have seen, and the great majority of those reported. The growth is at first superficial, and until the second stage, that of penetration to submucous structures, has commenced, it can, like similar growths in the larynx and tongue, be removed with good prospect of immunity from local recurrence. There is on *a priori* grounds every reason to suppose that while these growths remain confined to the mucous membrane the results of operation should be quite as good as those obtained in epithelioma of the tongue, for experience with that organ has proved that where the glands have been freely removed recurrence does not occur in the lymphatic channels. Needless to say, as soon as the muscles have been invaded extension is accelerated and the disease becomes much more malignant, but even when this has occurred I have been struck repeatedly with the slow rate of progress of the disease. This comparatively low grade of malignancy is not an invariable feature, and it is not so often seen in the case of young women; but there seems to be a general consensus of opinion that these neoplasms are of fairly slow growth and certainly are much less malignant than those occurring in the tonsils, which we do not hesitate to attack by operation.

Malignancy is a relative quality, and wide experience in many parts of the body has proved that so long as an epithelioma remains superficial it may be removed with good hope of success; it becomes, therefore, a positive duty to remove it when the anatomical disposition and the general condition of the patient render operation at all feasible.

We have, then, to consider whether removal entails an operation which is unreasonably formidable; the answer must be an emphatic negative. I have no personal experience of resection of the œsophagus proper, but where the disease does not descend below the œsophageal ostium, removal presents no great difficulty; that is to say, the access to the part is good and easily obtained. Where the growth is still superficial no difficulty whatever is encountered in detaching the pharynx from the larynx, and the excision is accomplished in an operative field unobscured by blood and mucus. Where the tissues of the larynx are invaded the operation is no more difficult to perform than an ordinary laryngectomy, and I think every one will agree that the question of immediate success in that operation is almost entirely one of efficient hæmostasis. Probably for laryngologists the most troublesome and lengthy element in these cases is the removal of the gland area, and I would urge any one making his first essay to perform two separate operations.

I propose, however, to leave all questions of technique to be dealt with by Mr. Trotter, and the point I wish to make is that by reason of the ready access to the parts there is no difficulty to be encountered that is not encountered in other generally recognized throat operations, with the sole exception of the plastic manœuvres, which are merely a matter of technique and are only pertinent to the general question before

us in so far as they take time. These operations entail no special dangers with respect to respiration other than those which we are already quite used to, and post-operative shock is certainly not a marked feature of them.

It would be idle to deny that serious risks do exist. They are post-operative and consist in suction pneumonia on the one hand and in sepsis of the neck wound on the other, with consequent mediastinitis or secondary hæmorrhage. With regard to septic pneumonia I personally have never seen a death from it either in my own cases or in those with which I have been associated, amounting to more than a dozen, even where a total extirpation had been performed without employing the Solis-Cohen fixation of the trachea. Although I have only once made use of it, I cannot but think that Trotter's plan of retaining a tracheotomy tube for a week after the pharynx has been removed and the larynx preserved, makes greatly for safety in this respect, by excluding aspiration through the glottis. With regard to sepsis due to leakage from the pharyngeal wound I have seen several cases, but curiously enough none of them have ended fatally. In these cases sloughing, extensive in area but superficial in depth, took place, and in two instances secondary hæmorrhage occurred about the eighth day, in one from the inferior thyroid artery and in the other from a small vein ; but in no case have I seen mediastinitis occur. In a word it has seemed to me that if the septic cavity is kept widely open and the packing frequently changed, Nature, as exemplified in cases of sepsis of the deep structures of the neck, has behaved in a very kindly manner and has appeared to do all in her power to encourage us to a bold course of action. I have, on the other hand, lost three patients from operations of this kind in whom the surgery was entirely successful. One during an excellent recovery received a large dose of milk in his trachea ; another, healed by first intention, and already eating mutton-chops, suddenly succumbed to diarrhœa diagnosed as influenzal in origin ; while a third, an alcoholic subject, became maniacal and finally comatose, though his neck had healed by first intention. No doubt the operation was in a sense partly responsible for death in all these cases, but it seems to me that such occurrences should not be allowed to weigh too heavy in the balance when estimating the degree of danger, while they exemplify the misleading character of bald statistics, for, as I repeat, the cervical region appears to possess quite a singular faculty of resisting fatal septic phenomena.

Any one with experience of laryngectomy will admit that suction pneumonia and wound sepsis belong precisely to that category of surgical accidents which it is the duty of the surgeon to face and by technique and careful after-treatment to eliminate, and this in fact already has been done in a very large degree. To avoid an operation on account of the existence of dangers such as these is pusillanimous and retrogressive. One may cite in this connexion the figures of Gluck and Soreensen, which show an operation mortality of 19 in 72 pharyngeal cases, but these surgeons

had no death in their last 14 cases, thanks to ' asepsis and primary union ', in a word, to technical success.

We must next ask : Is the mutilation too great to be tolerable ? Again I would answer emphatically in the negative. I have never seen any of the cases in which one of the ingenious plastic methods such as those of Gluck, Trotter, and others have left a perfectly successful result ; the reports of such cases speak for themselves. It is more important for our present purpose to speak of the comparative failures, for a surgeon during his early attempts must be prepared to meet with lack of union and the formation of pharyngeal fistula. Upon this point I wish to draw attention to the remarkable adaptability of the cervical region to artificial conditions, and to the faculty which these parts possess for spontaneous cure. It would seem that no pharyngeal fistula is too large for unaided closure. I have seen a fistula of the pharynx 4 inches in length, the result of sloughing, heal to a pin-point in the course of a few months—while in another case a fistula 3 inches long, which could not be closed at the operation, healed up completely in the course of three weeks. The latter case illustrates another very important point, namely, that where a stricture ought by rights to be expected it does not necessarily develop. A mere strip of mucous membrane $\frac{1}{2}$ an inch in breadth in the right lateral wall alone could be preserved after removal of an extensive post-cricoid growth. I had fashioned the flaps with the intention of removing the larynx, but on inspection found that that organ could safely be left. As a consequence no skin flap was available for a plastic, and I had to be content to pass a nasal tube and leave the fistula widely open. Not only did the wound heal as I have said quite readily, but now after six months the patient swallows naturally and with daily increasing ease. In another pharyngeal case where the larynx was removed, just enough mucous membrane remained to stitch round a No. 3 catheter. The patient developed a large funnel-shaped pharynx without the least stricture.

I suggest that if Nature is prepared to encourage us with these gymnastics we ought at all events to accept her encouragement. With improved technique these operations are not difficult to perform, are attended with no essential dangers which are too formidable to be faced, and they result in mutilation which is remarkably and unexpectedly slight. Even where the larynx has to be removed we have no right to shirk operation. It is the opinion of some surgeons that a man deprived of his larynx is *ipso facto* a miserable being. My experience entirely contradicts this view. One may estimate the measure of a man's misery by the complaints which he utters. I have seen a clergyman with such a loss, complain that his bishop for three years forbad him to preach ; and I have seen a cook who was surprised and annoyed when a prospective employer detected her loss ; I have also known a man happily engaged in the control of a considerable business ; and yesterday, on inquiring of a hearty robust gentleman who lost his larynx four years

ago, whether he regretted the operation, I received a derisive laugh in reply. There is no question in my own mind but that what is said about this mutilation is very greatly exaggerated.

The final judgement upon all questions of cancer surgery must depend upon success *quâ* recurrence. Up to the present the records are bad. Taking the results of Gluck and Soreensen alone we find in 53 cases surviving convalescence, that in 27 recurrence took place within the year, while 10 more died of recurrence within three years, 10 remained free from recurrence, at what period after the operation is not stated. To call these results highly encouraging would be fantastic. I would like at this point to make it clear that my primary object is not to speak as apologist for the operation, it would be out of place for a surgeon who happens to have performed a couple of operations of the kind under discussion, to do so ; I speak as a laryngologist, that is to say, as a doctor equipped with precise and efficient means of early diagnosis, and my appeal to-day is to laryngologists to make a serious attempt to bring more suitable material into the field of operative surgery. Gluck and Soreensen make a notable remark, namely, that in the 27 cases of rapid recurrence the latter was seldom local but nearly always in the glands. The failure to cure depended, at all events in large measure, not upon difficulties presented by the primary growth, but upon details of cervical gland surgery ; in a word, the onus of failure is upon the shoulders of general and not of special surgery.

I put it that the present position of pharyngeal surgery is analogous to that of the tongue surgery of twenty years ago. The statistics for the tongue have greatly improved in this country since Butlin insisted upon the systematic removal of the gland area, gave data for early diagnosis and, in particular, urged the avoidance of palliative measures in doubtful cases. We have not before us to-day in the laryngoscopic aspects of the hypopharynx the data which Butlin brought forward concerning the tongue, but I claim that we do possess at this moment the means of furnishing precisely analogous data, and that it is our duty as laryngologists to relax no effort in using them. I fear that, in fact, we are not performing this duty, and herein lies the importance of this discussion.

Our special duty and function is to make the diagnosis early. In the case of cancer of the vocal cords we learnt to perform this function with ease and with extremely encouraging results : there the parts concerned are in full view and the early symptoms are often pronounced. In the case of the hypopharynx the matter is not so simple. At the present time we have but few opportunities of examining patients at the outset of the disease ; we have, however, no difficulty in learning by inquiry that they have experienced slight symptoms for many months. As a rule they have complained of a sense of localized rawness on swallowing dry food, and several of my cases have assured me that they have complained to their medical advisers that they felt the presence of an ulcer or a rough spot in the throat, but that the doctor refused to examine the organ.

Others have complained of localized pricking sensations on swallowing pungent food and drink. I believe that pain on empty swallowing and the radiating pain during rest, are symptoms denoting penetration of the submucous tissue. I have observed very definite pain on pressing the larynx back against the spine, but it was not present in the one case of purely superficial growth which I have had a chance of examining.

The sensation of rawness on swallowing dry food is not a symptom upon which a surgeon will care to place much reliance, knowing as we do that pharyngeal phenomena of the kind are often due to chronic irritation of the tonsils by calculus or septic plugs, as well as to marked granular pharyngitis, and in the hypersensitive and climacteric to slight aberrations from the normal which remain unnoticed by healthy patients. Where these well-known causes of slight dysphagia exist they can generally be recognized as such with the careful use of the probe, which elicits the sensations complained of. If, however, the symptoms persist after the removal of these common causes of discomfort, we should not, I contend, be satisfied by observing a normal picture on laryngoscopy.

In the one or two early cases which I have seen there has been none of that excessive mucus which is so common a feature in cases of cancer of the pyriform fossa. We are all familiar with the characteristic œdema of the posterior aspect of the arytenoids and with the subsequent development of a ridge or cockscomb of whitish growth, but it is our object to anticipate these comparatively late developments.

With regard to physical signs the early cases present nothing which can be detected by external palpation, and I cannot but think that, at least one case which I have seen would have escaped detection by internal palpation and certainly its extent could not have been estimated by that method. Even during operation I have been unable to detect a growth by palpation while the constrictor muscles remained intact. We have, however, in hypopharyngoscopy as described by von Eicken, and still more so in the direct method, absolutely certain means of diagnosis, and by the latter method of estimating the exact extent of the growth. I put it that a practice should invariably be made of hypopharyngoscopy, and in a tolerant patient, also of the direct method in all cases of persistent unexplained discomfort in swallowing.

It is not much to ask these patients to put up with the slight and momentary discomfort of hypopharyngoscopy. This method does not, in my hands, it is true, invariably afford a good view of the œsophageal ostium, but it always gives a view of the main area of the post-cricoid pharynx, i. e. that very area from which a growth early detected can be removed with good prospect of success. If only one such early case is detected in a thousand examinations the trouble involved will have been fully repaid. Moreover, a large number of patients, alive as the public now is to the cancer problem, will gladly accept this discomfort as the price of a favourable report from the surgeon.

Hypopharyngoscopy has frequently left me in doubt as to the presence

of disease low down and always as to the exact extent of disease wherever located. I therefore consider that when the surgeon has any genuine grounds of suspicion he should employ the direct method. In many cases this can readily be done under cocaine anæsthesia and reliable data obtained, but in certain of the deep cases a satisfactory examination cannot be made without narcosis, and in my experience this has always been necessary in order to make out exactly the limits of the disease. It is unnecessary to mention a fact which I believe every one present will endorse, that the direct observation of the region of the ostium under cocaine anæsthesia is far more difficult than that of any other region of the gullet.

The ideal method for examining the hypopharynx and the pyriform fossa is by means of the suspension apparatus of Killian, aided by the use of a strong probe of oval section by means of which the larynx can be retracted from the spine and tilted from side to side. I venture to take this opportunity of suggesting to some surgeons who have been disappointed with Killian's apparatus, that their failure to obtain good results may be due to their neglect of the fine adjustment provided by the horizontal excursion of the pillar. I have repeatedly found that a very imperfect view has been converted into an ideal one by a turn or two of the handle of the horizontal screw. With our hypersensitive town-bred patients we in London find that narcosis is generally required for this examination, but it cannot be said that narcosis which we employ here so freely for all kinds of minor surgical manœuvres is a high price for a cancrophobiac to pay for the definite assurance that cancer is absent. No apology need be made for its employment where cancer is actually found, for by means of a direct examination under narcosis not only is the diagnosis established by the removal of a fragment for the microscope, but definite data are provided upon which to estimate the chances of surgical success.

The question of operability or otherwise in any case must depend upon both local and general considerations and upon either count the individual surgeon will undoubtedly retain his own view, whatever may be the consensus of opinion expressed by this meeting, but I suggest that there is a decided tendency in this country at least, to overrate the dangers and difficulties to be encountered. I confess myself a sinner in this matter, for only a few months ago I made a direct examination on behalf of my colleague, Mr. Peter Daniel, of a case of malignant stricture of the ostium in a man of about 45, the subject of well-marked chronic alcoholism and debilitated to an extreme degree. I gave the opinion that the subject could not possibly survive radical operation. My colleague entertained a more sanguine view and removed the whole hypopharynx and an inch and a half of the œsophagus with complete success, and up to the present there is no recurrence. I have myself removed the larynx for a penetrating post-cricoid carcinoma in a young woman in a condition of extreme anæmia and apparently unfit to undergo

any serious operation whatever, and I undertook the operation only at her earnest request to be allowed to venture the ordeal. She made an excellent recovery. I have also performed the same operation, including removal of a portion of the tongue, in an elderly man the subject of a double aortic disease, and recently recovered from a severe attack of delirium tremens. His convalescence was troublesome, but he survived the operation four years. I suggest, therefore, that even in desperate cases it is the duty of the surgeon to take desperate risks granted that the patient has the spirit to make the hazard.

To conclude, will you allow me to put forward the following points as worthy of discussion :

In the first place, I suggest that these cases of post-cricoid carcinoma are far from uncommon, and that the disease deserves special consideration as a clinical entity.

Secondly, I suggest that these cases come within the scope of recognized everyday surgery and should not be attacked in a spasmodic manner as acts of ' virtuosity ', to quote Mr. Trotter's happy phrase, and in the spirit of the forlorn hope.

The disease frequently occurs in youngish women, favourable subjects for operation, and not only in the old and debilitated.

The growth is almost invariably squamous epithelioma, and like similar surface growths elsewhere is of mild malignancy in its early stages. The typical operation and even the atypical, is by no means a formidable one, seeing that access is easy and the operative field freely exposed to view. The difficulties are just such as can be met by technique, and are quite unlike those inherent to malignant disease of the œsophagus proper. There is no essential reason for a high operative mortality, and, indeed, the tissues involved seem to be peculiarly resistent to the ill effects of local sepsis. Septic pneumonia is most certainly an avoidable accident. The mutilation tends to be far less than one would naturally expect, thanks again to the natural adaptability of the parts concerned.

If it is worth while considering the condition at all, our aim must be early diagnosis, and for this we laryngologists have at hand perfectly and invariably effective means. It is my contention that it is our duty to employ them in all suspicious cases.

I earnestly hope that this meeting will, in the main, endorse these statements and thereby establish the attempt at early diagnosis by ocular inspection as a measure of daily routine. It is my belief that if this is done and if the rising generation of surgeons is encouraged to adopt a courageous attitude we shall by the date of the next congress have accumulated a mass of favourable evidence ; evidence which will induce the profession at large, on the one hand to have these cases of early dysphagia adequately examined, and on the other to obtain a surgical opinion before consigning their cases of evident post-cricoid cancer to the home for the dying. So we may hope that the most pathetic chapter in laryngology may come to be revised or wholly rewritten.

SECTION XV

RHINOLOGY AND LARYNGOLOGY

INDEPENDENT PAPER

POST-CRICOID CARCINOMA

Principles of Operative Treatment

By WILFRED TROTTER

ABSTRACT

Every region of the body possesses a local pathology which is not always obvious, but a knowledge of the peculiarities of which is essential for the practical surgery of the part.

Tumours of this region should be called 'hypopharyngeal' rather than post-cricoid, because they include growths of the lateral and posterior walls of the pharynx as well as those on the posterior aspect of the cricoid. The term hypopharyngeal should not include tumours originating in the sinus pyriformis or at the opening of the larynx.

Hypopharyngeal growths are common in young and middle-aged women, and are occasionally met with in men.

Malignant disease of the pharynx presents two difficulties, the first being the nature and situation of the disease, the second defective diagnosis. It is usually easy to recognize carcinoma of the hypopharynx in the early stages, because it causes definite symptoms, and can be felt with the finger introduced through the mouth. Diagnosis with the finger is much more certain than visual examinations. In the majority of cases, symptoms have been present for six to twelve months, and the disease is inoperable, or requires mutilating treatment. This is largely due to a widespread belief that cancer of the pharynx cannot be removed, and consequent postponement of operation until the symptoms are urgent.

The chief difficulties in the treatment are the relatively inaccessible situation and the virulence of the organisms normally inhabiting the pharynx; also the importance of the pharynx in respiration and deglutition. To obtain a cure, the following principles are essential:

1. *Early diagnosis.* During the first three months, epithelioma in this region is probably confined to the mucous membrane, is freely movable, and ulcerates slowly. At this stage it can be cured by local

excision. Later, the disease rapidly becomes diffuse, and invades the submucous tissues, so that extensive operations are required. The latter are more dangerous and less likely to give freedom from recurrence. The distinction between the superficial stage and the stage of submucous invasion is, therefore, of the greatest importance. The former condition is always curable, but in the latter there is never any certainty of eradicating the disease. Unfortunately, the majority of cases operated upon have belonged to the latter class.

2. *Exact diagnosis of the primary seat of the disease.* One of the greatest obstacles to early operation has been the widespread belief that the larynx must always be removed in these cases. True hypopharyngeal tumours, when seen early, can generally be removed locally without laryngectomy.

In advanced disease, three questions arise:

(*a*) Are the structures of the neck involved?

(*b*) Has the disease extended to the œsophagus? A positive answer to either should be decisive against operation.

(*c*) Has it originated in or extended to the sinus pyriformis? This can be determined by manual examination, and, if found, total laryngectomy will be necessary.

3. *Free exposure of the tumour by the* transthyroid *route before any attempt at removal is begun.* This requires an incision along the anterior border of the sterno-mastoid muscle, without disturbance of the carotid sheath; turning aside the infrahyoid muscles; excision of the apex of the lateral lobe of the thyroid body; division of the inferior constrictor muscle at its origin from the larynx; removal of the ala of the thyroid cartilage and of the great cornu of the hyoid; longitudinal incision of the pharynx.

4. *Bilateral gland dissection after the primary growth has been removed.* This is necessary in every case.

The dangers of these operations are undoubted, the chief risk being infection with micro-organisms from the pharynx, which may result in acute scepticæmia (death may occur in two to three days), spreading infection and sloughing cellulitis (generally fatal in ten to twelve days), sometimes extending to the mediastinum without causing definite symptoms, and secondary hæmorrhage, which always occurs from the eighth to the tenth day, just when the patient seems to be out of danger. These dangers can be minimized by (*a*) preliminary precautions, such as careful cleansing of the mouth. Such good results have been obtained in edentulous patients that it is questionable whether an operation should be undertaken unless all the teeth have been removed.

(*b*) The conduct of the operation. Special measures must be taken to protect the wound from pharyngeal mucus by packing it with gauze, and to prevent later infection by immediate suture (rarely possible), or by stitching a flap of skin into the pharynx at once. A rectangular flap, with its base over the sterno-mastoid, should therefore be cut with the

first incisions. The stitching should not be by ordinary edge-to-edge points, but by so-called mattress sutures. With proper precautions, primary healing of the flap may be obtained, and there is no infection or sloughing of tissues, which is of incalculable advantage. After some weeks, the pedicle of the flap may be divided and the small fistula into the pharynx closed. Frequent dressing of the wound during early convalescence is also very important.

Mutilation from the operation is not likely to occur when the above suggestions have been observed. Attention should be directed to (1) the possibility of dealing with hypopharyngeal tumours adequately without laryngectomy; (2) the value of the transthyroid method of approach; (3) the practicability and value of immediate plastic reconstitution of the pharynx, which renders a stricture unlikely to occur, and diminishes the danger of spreading infection.

SECTION XV

RHINOLOGY AND LARYNGOLOGY

POST-CRICOID CARCINOMA

DISCUSSION

Mr. Philip R. W. de Santi (London) could remember some 7 cases of distinct post-cricoid carcinoma under his own immediate care. Of these 5 were females, and 2 males : the females were 29, 30, 32, 42, and 47 years of age ; the males were between 50 and 60. Of all the cases, only three were operable. One, a female of 29, with quite an early growth, was shown by Mr. de Santi in 1905 at the London Laryngological Society. The diagnosis was confirmed by Butlin and Semon, but Chiari and Gluck looked upon the case as specific. The patient refused operation and died eight and a half months later from epithelioma. Another, a female of 31, had gastrostomy done, and at the same time removal of the lower part of the pharynx, the whole larynx, the trachea to the level of the top of the sternum, the œsophagus to the same level, the whole of the thyroid gland, and a mass of glands in the neck. She made a good immediate recovery from the operation but died within two and a half months, having led a miserable existence from the time of operation. She would have been better served by gastrostomy, and tracheotomy alone. The other patient was a man of 55, and the operation had to be abandoned on account of its extent.

The whole crux of the matter was the difficulty in getting these cases early. Their nature was not recognized by the practitioner and, as a rule, when seen by the specialist they were beyond operation altogether, or only a large mutilating operation could be done. The prognosis was always grave, as recurrence in the glands was the rule. At the same time the young age of the patient and the greater recuperating power must be borne in mind. Again, an improved operative and post-operative technique had been achieved. Mr. de Santi recommended a preliminary gastrostomy or œsophagostomy, and in the after-treatment the use of continuous saline rectal feeding.

Dr. A. Logan Turner (Edinburgh) said he had nothing to add to what he had recently written upon the subject of malignant disease of the lower pharynx and œsophagus. He had collected his cases, which were 70 in number, and of these 38 per cent. were males and 61 per cent.

females. He had also made a comparative study of malignant disease in connexion with its sex incidence in the alimentary canal in immediate continuity with the œsophagus, namely, the fauces and oro-pharynx on the one hand, and the stomach on the other; and he had made a similar inquiry in connexion with the larynx. In all these regions the disease was of much more frequent occurrence in the male sex. He was not prepared to offer any explanation of the greater frequency of carcinoma in the post-cricoid region in women. In regard to the age incidence also, the disease occurred much earlier in women than in men, and in the post-cricoid region it showed itself at even an earlier age than elsewhere in the œsophagus. The duration of the disease was shorter in the lower pharynx than in the œsophagus.

As to the treatment of cases of carcinoma behind the cricoid, he was in favour of operation by which the pharynx and œsophagus were exposed through an incision on the left side of the neck. In this way, if the disease was not too extensive, it could be removed: if this was impossible, an œsophagostomy was done, and a tube was stitched into the lower end of the wound. Gastrostomy was not a very satisfactory procedure in these cases.

Dr. H. MARSCHIK (Wien). Die Resultate der Wiener Klinik werden auf der Naturforscherversammlung dieses Jahres eingehend besprochen werden. Gastrostomie sollte besser unterbleiben, wie Turner sagt; wir haben durch sie einen Fall verloren, der vielleicht durch einfache Radikaloperation hätte gerettet werden können. Operative Resultate sind befriedigend, über Dauerresultate bestehen noch zu wenig Erfahrungen. Unter den Fällen sind besonders interessant: (1) Eigenartiges Ulcus rodens der Hypopharynxhinterwand, ohne Drüsenmetastasen und sehr gutartig; (2) Abklatschkarzinome der Hinterwand bei einem grossen Karzinom der Larynxhinterwand; (3) Karzinom des Recessus piriformis bei einem jungen Mädchen, durch Hypopharyngoskopie entdeckt, mit grossem Drüsentumor; (4) zwei Fälle von Karzinom mit Lues kombiniert. Durch präliminare antiluetische Behandlung verschwand das, was an dem lokalen Infiltrat Syphilis war, und wurden die Patienten so gebessert, dass sie darnach nicht mehr zur Radikaloperation zu bewegen waren. (5) Ein Fall zeigt, dass man manchmal bei der Operation Ueberraschungen erlebt. Karzinom des Oesophaguseingangs wurde operiert mit umfangreicher Resektion der Halsorgane; dabei zeigte sich bereits der Oesophagus tiefer unten erkrankt. Die Sektion des Falles von Arrosion der Carotis communis durch das Karzinom ergab den ganzen Oesophagus bis zur Kardia in Neoplasma umgewandelt. Der Oesophaguskrebs hat die Tendenz, dem Schlauch entlang nach oben und unten, weniger mit Ueberschreitung der Wand, konzentrisch zu wachsen, und es kann der ganze Oesophagus vom Hypopharynxeingang bis zur Kardia in Karzinom umgewandelt sein, ohne dass die Umgebung ergriffen wäre.

Dr. William Hill (London). The speaker dealt with palliative endopharyngeal treatment. He had carried out operative removal *per vias naturales* with the help of his large operating direct pharyngoscope. This was necessarily incomplete and palliative, and would be unsurgical if the patient would consent to operation and in operable cases. Removal was followed by the application of radium—200 mg. screened by 2 mm. of lead, silver, or platinum for from twelve to twenty-four hours. Radium was an uncertain remedy and was less often effectual in the deep or post-cricoidal pharynx than in the true gullet. In the latter region he had employed radium nearly forty times with very marked improvement both subjectively and objectively. In three cases the growth had temporarily disappeared as far as naked-eye evidence was concerned. In the deep pharynx there had been very decided benefit in 3 out of 8 cases, but others failed to react. Most of his 50 cases of post-cricoidal cancer were too advanced even for radium, and he then resorted to intubation with his styletted tube, rather than to gastrostomy. The statistics had been confused in these primary pharyngeal cases by their being inaccurately labelled as either extrinsic laryngeal, or œsophageal, cancer; Waggett's terminology abolished confusion. Mikulicz's nomenclature, hypopharynx, was obviously bad, as it meant *underneath* the pharynx, i.e. in the gullet.

Sir Robert Woods (Dublin) thought that Mr. Waggett had hit the nail on the head when he said that the chief difficulty in treating post-cricoid carcinoma lay in making a sufficiently early diagnosis, for, by the time such a case developed any symptoms, it had to be regarded as already considerably advanced.

His own experience of the operation was limited to 4 cases, in all of which the disease was so far advanced as to necessitate extirpation of the larynx. He thought that when complete transverse section of the gullet was necessary for removal of the disease, very often there would be great difficulty in avoiding stricture. He had obtained much help from a rubber tube adjusted to a fistula above the trachea. The fistula was kept open by a solid rubber plug. This device enabled the patient to remove and reintroduce the pharyngeal tube without medical aid.

Professor Uchermann (Christiania). I have operated on three or four of these cases in recent years and have always tried to preserve the larynx, making a lateral pharyngotomy and removing what was diseased without doing a total laryngectomy. I do not think it necessary to go so far in cases in which the larynx has still an almost normal appearance and is only partially involved. The cases were all elderly men. The result in one case was satisfactory in so far as the man lived comfortably for one year and then died of an accidental pneumonia. No signs of carcinoma were found. In two other cases there was fatal recurrence after a few months. My last case, which was operated upon recently,

was that of a man of about 60 years, a ship's engineer. A year previously he had had anterior pharyngotomy performed by a surgeon for a tumour in the pharynx. When I saw him, a flat, greyish, non-ulcerating tumour was present in the left sinus pyriformis and in contact with the posterior surface of the cricoid. In this case I did the operation in two steps, first making the ordinary incision through the skin and muscles, and three days later the incision of the mucous membrane under local anæsthesia. One gets in this way no infection of the wound and no fever. The tumour, as large as an oblong penny-piece, was fairly easily removed, an œsophageal tube was put in and the wound partly filled with iodoform gauze. The last application unfortunately had the effect of making the man quite maniacal for a whole week after the removal of the gauze. I had the same experience once before after packing a big wound with this gauze and shall not repeat it. After three months the man went back to his ship, but three months later there was recurrence at nearly the same place, which I removed with forceps (endopharyngeal). Afterwards I applied radium, being of the same opinion as Dr. Hill, that the time for treatment with radium is directly after the radical operation. The result has yet to be seen. I have had good results with similar treatment in malignant tumours of the upper jaw.

Reply

Mr. E. B. Waggett (London) trusted that the interest evinced by the meeting in the subject would result in the systematic and habitual examination of the lower pharynx in all suspicious cases. This would inevitably lead to the discovery of early cases and thereby remove a source of opprobrium from laryngology.

[Friday Afternoon, August 8]

SECTION XV

RHINOLOGY AND LARYNGOLOGY

INDEPENDENT PAPER

PACHYDERMIA LARYNGIS: SOME POINTS IN ITS ÆTIOLOGY AND DIAGNOSIS

By JOBSON HORNE, M.D., Surgeon to the Metropolitan Ear, Nose, and Throat Hospital, formerly Science Research Scholar, the British Medical Association

Pachydermia laryngis has been added to the literature of this special department of practice since the Congress met in London in 1881. The morbid change in the larynx was first described by Virchow in 1887 and the term was brought into prominence at that time by an historical case. The tendency was and has been since to regard the condition as an entity and not as a manifestation of some underlying disease. The warty thickening of the larynx was originally attributed to chronic inflammation, but the cause of the inflammation and the nature of the underlying irritant had not been gone into. Hence the necessity for further inquiry into the ætiology.

In the short time available for the reading of a paper it is impossible to enter at all fully into any of the several interesting problems which the title of this paper suggests. I propose to restrict myself to one problem only—the development of the warty excrescences about the vocal processes and in the inter-arytænoid space. In the fewest words possible I will tell you the results of my researches, and after reading the paper I will demonstrate on the screen photographs of macroscopic preparations and of microscopic specimens illustrating the points I mention.

In a typical case of pachydermia laryngis the changes about the vocal processes of the cords are so striking as to be pathognomonic. From one vocal process, as you know, projects a broad-based excrescence which on phonation is received into a pouch or depression on the other vocal process. Huskiness is the main symptom. But the amount of huskiness is often very slight, and far less than one would expect in the presence of such hyperplastic changes about the vocal processes. In fact, the slightness of the vocal symptoms has always been a matter for clinical comment and surprise.

Different explanations have been offered for the development of these changes about the vocal processes. Virchow in 1887 described the

occurrence of symmetrical oval swellings in the region of the vocal processes, the centre of each swelling being slightly depressed; this depression he attributed to the firmer fixation of the mucous membrane to the underlying connective tissue. Fraenkel, writing in 1889, did not accept this explanation of the depression, but attributed it to pressure exerted by the tumour of one cord upon the corresponding tumour of the other cord. Semon, writing in 1897, was of the same opinion as Fraenkel, namely, that this unilateral crateriform depression was probably the result of pressure by the opposite elevation, and not of the firmer fixation of the mucous membrane to the connective tissue; for if the latter were correct, he argued, the depression would not be invariably unilateral.

Clinically, that is to say, from the appearances presented in the laryngeal mirror, the explanation offered by Fraenkel and Semon seemed to be a very natural one; at the same time it did not harmonize with the remarkable absence of loss of voice which one would have expected to result from the pressure required to bring about the condition met with.

With a view of arriving at a solution of this difference of opinion, I investigated the point in its pathological as well as its clinical aspect.

The results of my investigations were first communicated to the British Medical Association at Portsmouth in 1899. The following is a brief summary of those results:

Firstly, in all the specimens examined the depression or pouches about the vocal processes were found to be bilateral; the depression was usually more marked on one side than on the other, but it was present on both cords.

Secondly, in no case did the pouch or depression, when examined under the microscope in serial sections, present evidence of pressure or attrition. On the contrary, a greater degree of hyperplasia was found. The theory of the formation of the depression by pressure was therefore entirely refuted by histological evidence.

Thirdly, there was histological evidence to show that at certain spots the mucous membrane below the vocal process was, as described by Virchow, more intimately adherent to the underlying cartilage.

Based upon those pathological findings the following is the explanation of the whole matter which I offer:

1. In the normal larynx a line formed by a ridge of mucous membrane starts from behind the vocal process of each cord and passing downwards and forwards, takes a crescentic course running immediately below the process and parallel to and below the vocal cord. This line is most marked at the vocal process and then thins off. It is more apparent in the male sex, and pachydermia laryngis is more frequently met with in the male sex. The mucous membrane immediately above and below this ridge and on either side is more intimately adherent to the submucosa, hence the formation of the ridge itself. The ridge may be duplicated, and occasionally it may be so pronounced as to suggest in the mirror a duplication of the vocal cord itself.

The vocal process may be looked upon as a *point d'appui* and this ridge represents and may be conveniently spoken of as a line of traction.

2. In pachydermia laryngis the changes in the region of the vocal process—certainly as observed at an autopsy—are, as one would naturally expect, symmetrical and bilateral.

3. When, in the later stage of pachydermia, a warty growth appears in the region of the vocal processes it is not strictly a neoplasm. It is only a localized hyperplasia, or exaggeration of a pre-existent structure, namely, the ridge or line of mucous membrane already referred to.

4. The mucous membrane immediately above and below that part of the ridge which has become exaggerated is more intimately adherent, hence the furrow or depression above and below the excrescence. In this way there is formed at least one depression on both cords. For this reason the unilateral theory of the pouch again must fall.

5. These furrows and excrescences about the vocal processes, being developed at symmetrical spots and in the same plane, presumably would interfere with the apposition of the cords and the production of voice; but, clinically, as we know, this apposition is not interfered with, at least not sufficiently to prevent phonation. The solution of this problem of the preservation of the voice in spite of the bilateral hyperplasia of the vocal processes rests on the following observation.

6. By the time the warty condition about the vocal processes is established the cords, I found, were no longer on the same plane. An alteration in the plane of the cords, however slight, at once explains the preservation of the voice. For one cord has to be only on a level very slightly higher than its fellow to permit of a dovetailing of the excrescences and depressions and of the preservation of the voice. This dovetailing may be spoken of as a vertical adaptation of the cords. The alteration in the plane of the cords I found out in the following way. The entire larynx from a case of pachydermia laryngis was hardened without being cut open so that the cords were preserved in the same position as at the time of death. The larynx was then set in hard paraffin and vertical sections were cut at right angles to the vocal processes and the relative positions occupied by the cords were demonstrated as I have mentioned to you.

Another striking feature of pachydermia verrucosa, but not so frequent in my experience as the one I have just treated of, is (as you know) a warty excrescence in the inter-arytænoid space; this, in pachydermia simplex, is placed centrally and is usually bilobed, these two points serve to distinguish it from the tuberculous nodules or granulations so frequent in this space, which are placed more to one or the other side of the middle line, are less uniform, and their epithelial covering is crenated, thinned, and, perhaps, ulcerated. The bilobing is accounted for by the excrescence being merely a hyperplasia of pre-existing tissue, and so retaining the central fissure commonly present in the inter-arytænoid space.

[Saturday Morning, August 9]

SECTION XV

RHINOLOGY AND LARYNGOLOGY

DISCUSSION No. 3

INDICATIONS FOR AND RELATIVE VALUES OF TONSILLOTOMY AND TONSILLECTOMY

(For Reports by Prof. Dr. H. Burger and Dr. J. L. Goodale, see Part I, pp. 111–33)

Additional Remarks by Prof. Dr. H. BURGER, Amsterdam

Messieurs, — Vous avez lu mon rapport sur la question de l'opération des amygdales. J'ai dressé ce rapport en forme de thèses, forme qui permet de préciser le mieux les points à discuter.

Aujourd'hui je ne parlerai que de deux états morbides, exigeant une intervention chirurgicale, c'est-à-dire l'hypertrophie simple et l'inflammation chronique.

Plusieurs collègues de l'Amérique du Nord recommandent l'ablation radicale, toujours quand il y a une indication à opérer. Pour eux l'amygdale est un organe sans fonction et sans utilité, produisant fréquemment des affections graves, même mortelles.

Dans le cas d'hypertrophie simple, occasionnant des troubles de la respiration ou de la déglutition, ils ne se bornent pas à la simple amygdalotomie, mais procèdent systématiquement à l'ablation totale de l'organe hypertrophié. A plus forte raison ils considèrent cette opération indiquée quand l'amygdale se trouve dans un état morbide chronique, ou quand elle sert de point de départ à des inflammations amygdaliennes ou périamygdaliennes, s'accompagnant ou non de complications éloignées ou générales.

Pour ma part, je ne saurais nullement accepter ce point de vue. Ne pas connaître le rôle physiologique des amygdales, voilà ce qui ne donne pas encore le droit d'en nier l'importance. Je partage l'opinion du docteur Broeckaert qui dans un excellent rapport s'oppose à la suppression systématique des amygdales comme des organes nuisibles et dangereux. Quoiqu'on puisse très bien vivre sans ces organes, les preuves de leur non-utilité nous font défaut.

Mais je vais plus loin que mon ami belge. Je me rallie absolument à l'opinion de notre collègue américain Mackenzie qui dans un article des plus sympathiques vient de protester contre le ' massacre ' des amygdales.

Tout en admettant l'indication de l'amygdalectomie dans maints cas de tonsillite chronique, je considère la tendance moderne d'opérer de cette manière toutes les hypertrophies amygdaliennes de l'enfance comme une exagération dangereuse et nullement justifiée.

Voici les considérations principales en faveur de cette manière de voir :

1° L'ablation de l'amygdale est une opération plus difficile, plus sérieuse, plus dangereuse que la simple tonsillotomie.

2° Chez les enfants elle exige la narcose générale, tandis que la tonsillotomie peut toujours être faite à l'aide de l'anesthésie locale, dans la majorité des cas même sans aucune anesthésie.

3° Le danger dans les opérations amygdaliennes dépend en tout premier lieu de la narcose.

Il résulte de ces considérations que dans la jeunesse l'amygdalectomie ne doit jamais être faite sans indication précise et urgente. Et voilà la première thèse que je voudrais bien souligner :

A la jeunesse l'opération de choix c'est la tonsillotomie ; l'ablation radicale doit être réservée à des cas très exceptionnels.

Quels sont ces cas exceptionnels ? Je n'en peut pas trouver d'autres que l'inflammation aiguë d'une certaine importance, se répétant fréquemment, et touchant des amygdales de petit volume non aptes à la tonsillotomie.

Chez les personnes adultes le terrain de l'exérèse complète est plus étendu à cause de deux raisons : D'abord parce que chez eux l'opération peut être faite dans l'anesthésie locale, et puis parce que les tonsillites chroniques à cet âge sont de beaucoup plus fréquentes qu'à la jeunesse.

Cependant je ne suis pas d'avis que chez l'adulte toute amygdale malade doit être enlevée. A cet âge également je préfère la tonsillotomie dans la grande majorité des cas.

Je n'attends que très peu des méthodes conservatrices : le lavage des cryptes, la médication désinfectante et astringente, méthodes qui ont trouvé, l'année passée, plusieurs défendants dans l'Association allemande de Laryngologie.

La tonsillotomie classique, au contraire, donne des résultats bien satisfaisants. J'insiste à comprendre dans ce cadre non seulement l'opération avec l'amygdalotome, mais également celle avec l'anse froide ou galvanique et celle avec la pince de Ruault. C'est à tort, je crois, que Broeckaert compte toutes ces interventions au domaine de l'amygdalectomie. Je ne peux pas admettre qu'à l'aide d'une de ces méthodes il soit possible d'évider complètement la loge amygdalienne.

Broeckaert — c'était en 1910 — avouait qu'il n'avait pas encore pu se décider à l'énucléation de l'amygdale. Mais, pour exclure tout malentendu, il faut bien préciser que l'amygdalectomie n'est pas autre chose que l'énucléation, opération qui se fait dans le tissu qui sépare l'amygdale de sa loge. Quand on a vu la profondeur inattendue de cette loge après l'énucléation, il n'y a plus de doute qu'il soit tout à fait impossible de faire l'amygdalectomie par morcellement.

En résumant, je considère la tonsillotomie comme l'opération par excellence, aussi bien dans l'enfance et l'adolescence qu'à l'âge adulte.

Mais il reste, surtout chez les adultes, un groupe bien distinct de malades, pour lesquels l'amygdalectomie est un véritable bienfait. Qui de nous n'a pas rencontré dans sa pratique ces cas de tonsillites ou péritonsillites se répétant sans cesse et s'opposant hardiment à toute intervention : amputation à l'amygdalotome, morcellement d'après Ruault, cautérisations répétées, instillations infinies !

Je me souviens de deux malades, sujets à ce traitement infructueux pendant des années. La vie de ces deux personnes était troublée par des angines fréquentes, s'accompagnant chaque fois d'un abattement général d'une si longue durée que, à peine rétablis, ils furent survenus par une nouvelle poussée inflammatoire. Ces deux personnes vinrent me consulter avec une longue histoire de traitements divers. Ils devinrent mes premiers cas d'amygdalectomie et je puis ajouter que le succès en a été complet et définitif jusqu'à ce jour.

Depuis ce temps j'ai pratiqué l'opération dans des cas pareils, en somme pas très fréquemment, grâce à mon indication restreinte. Sur ce petit nombre de cas je n'ai eu ni d'insuccès ni d'accidents. Une seule fois une hémorragie, survenue quelques heures après l'opération, fut un peu inquiétante, mais pour la combattre il suffit la simple compression.

En somme je considère l'amygdalectomie comme une acquisition précieuse de la chirurgie de l'amygdale. Elle s'applique surtout aux angines et péritonsillites à répétition qui ont pour point de départ des amygdales petites ou enchatonnées. Mais, assurément, on n'a pas le droit de vouloir la substituer à la tonsillotomie classique qui reste le moyen sûr et puissant contre l'hypertrophie amygdalienne de l'enfance.

DISCUSSION

Dr. Jacques (Nancy). J'ai été très satisfait de voir poser devant une assemblée comme la nôtre la question de la légitimité de l'ablation des amygdales palatines et de l'opportunité des opérations totales ou partielles. Cette question me paraît d'autant plus brûlante qu'elle est de celles, trop nombreuses aujourd'hui, où le public prétend prendre parti et où notre division n'est que plus regrettable. De cette discussion je veux espérer qu'il sortira une doctrine générale, sinon un consensus unanime.

Je me félicite d'abord de voir définitivement jugé, je pense, le point si controversé de savoir si la suppression totale des tonsilles est de nature à compromettre l'équilibre organique par la suppression d'une fonction hypothétique propre. Les arguments cliniques négatifs se voient confirmés par les recherches histologiques et physiologiques les plus récentes. Les travaux de Retterer, de Paris, sur l'histogénèse du tissu lymphoïde de la gorge et la toute récente monographie de Caldera, de Turin, sur la physiologie des amygdales s'accordent à démontrer que ces organes font

parti d'un appareil très étendu et que leur suppression ne saurait avoir plus d'influence sur le métabolisme que la perte de quelques ganglions infectés. Pour ma part, je serais assez disposé à assimiler les amygdales à la dent de six ans dont la vulnérabilité bien connue aux agents infectieux exige souvent le sacrifice précoce, sans qu'il en résulte aucun dommage appréciable pour la nutrition.

Ceci dit, je me déclare partisan de l'amygdalectomie totale, que je considère comme nécessaire — dans la grande majorité des cas — à la guérison radicale des accidents infectieux et qui ne doit pas être considérée comme une opération dangereuse, si l'on veut bien prendre certaines précautions, trop souvent négligées.

Les amygdales palatines dégénérées le sont dans toute leur profondeur et la conservation de la moitié inférieure de l'organe ne fait que réduire la source de l'infection.

L'ablation radicale des amygdales doit être regardée comme une opération relativement sérieuse, mais justifiée par ses résultats. Je n'hésite pas à employer la narcose chloroformique chez les enfants pour supprimer amygdales et adénoïdes infectées. J'utilise l'anse galvanique et la considère comme le procédé le plus parfait pour évider une loge amygdalienne sans léser les piliers, à condition d'agir avec précautions. Je touche la plaie avec une solution forte de glycérine iodée. Enfin je garde en surveillance à l'hôpital ou à la maison de santé mes opérés pendant 5 à 7 jours.

Mr. E. D. Davis (London) said that his remarks were based on experience gained from about 5,700 operations. The majority of the cases were operated on at Charing Cross Hospital, where it was the custom to see them two hours after operation, again one week later, and finally at the end of six months.

The notes of 1,520 of these cases had been analysed, and, in addition, the results of 156 tonsillectomy and tonsillotomy operations had been carefully examined more than six months after operation. Statistical tables had been attempted, but were found to be incomplete. Inefficient operations had been omitted.

Tonsillotomy. Method of operation. Number of cases examined, 78. The tonsils were ' chopped ', so to speak, by a sharp Mackenzie or by a French or Mathieu guillotine.

Recurrence. Of 1,520 cases operated on 41 had had tonsillotomy previously performed elsewhere and had to be done again, owing to recurrence of tonsillitis, adenitis, or other symptoms. Again, in 78 of the cases actually examined after operation 39 were failures and in 36 the operation was repeated.

Hæmorrhage following tonsillotomy was undoubtedly more than that following enucleation by the blunt guillotine or snare. The amount was increased when the tonsils were incompletely removed, or if the palate, pillars of the fauces, or base of tongue were injured, a rare occurrence.

Scarring and deformity. In tonsillotomy it was frequent to find the anterior pillar of the fauces adherent to the surface of the stump of the tonsil, but otherwise deformity did not exist.

Adenitis followed a large number of the failures of tonsillotomy.

Convalescence was similar, and of equal duration, to that of tonsillectomy.

Complications occurred quite as frequently as in tonsillectomy, but in both they were rare.

Tonsillectomy. Method of operation. Number of cases examined, 78. In children. The tonsils with the capsules were evulsed by a blunt guillotine. Anæsthetic, ethyl chloride. Patient sent home two hours after operation.

Adults. Deliberate and certain method of 'snare and forceps'. A simple minor operation followed by a minimum amount of trauma.

Recurrence. In the 78 cases examined more than six months after operation the results were extremely good. No repetition of the operation was necessary. In fact *no* recurrence existed.

Scarring and deformity. In the 78 cases in which the snare and guillotine enucleation had been performed, 3 showed deviation of the uvula and slight scarring of the anterior pillar. This deformity had no functional effect and was only revealed on a careful examination of the pharynx.

He had not seen a single case in which food or débris collected in the fossa left between the pillars of the fauces and above the base of the tongue.

Voice. Enucleation had been performed in a number of singers, and these patients were repeatedly seen more than six months after the operation. *All* the singers stated that the voice had improved. In the case of a young actress of 17, a few weeks after enucleation, the lingual tonsil appeared to be slightly enlarged, but afterwards subsided. This is the only case in which enlargement of the lingual tonsil was seen, though it was frequently looked for.

Adenitis. Tonsillectomy was performed in a number of cases for enlargement of the cervical lymphatic glands. The glands slowly and gradually diminished in size, and usually took more than six months to entirely disappear. The gland at the angle of the jaw was the last to go. He had not seen adenitis follow enucleation, but in *tonsillotomy* adenitis occurred in a number of cases.

Convalescence was not longer than that of a clean tonsillotomy. Tonsillotomy and tonsillectomy cases were treated in precisely the same manner.

Complications. Great care was taken to eliminate all septic foci, such as oral sepsis, before operation. Otitis media, an uncommon event, occurred with equal frequency in both operations.

The defensive action of the tonsil was frequently quoted as a contra-indication to tonsillectomy. In all the cases he had examined there was

no evidence to show that patients with no tonsils were more susceptible to local or general infection of any kind.

Conclusions. He was firmly convinced that tonsillectomy should be performed in every case. It was the operation he had carried out during the past four years.

In children tonsillectomy should be performed with the blunt guillotine.

In adults the more deliberate and certain operation of the 'snare' was necessary, more particularly: (1) when adenitis was present; (2) when frequent attacks of tonsillitis with or without suppuration had occurred; (3) when for mechanical reasons the guillotine operation was difficult, e.g. in cases in which tonsillotomy had been performed more than once.

Dr. Trétrôp (Anvers, Belgique) rend hommage au consciencieux travail des rapporteurs. Il est d'avis qu'il faut être éclectique, que la tonsillotomie et la tonsillectomie, opérations toutes différentes, ont chacune leurs indications.

La première est plus indiquée chez les enfants dans les cas d'hypertrophie persistante, suite d'inflammation chronique; la seconde surtout chez les adultes et tout spécialement en cas d'amygdales enchatonnées, impossibles à opérer par tonsillotomie. Les dangers de ces deux opérations sont nuls à condition de faire une préparation opératoire, consistant à traiter et faire disparaître toute inflammation avant d'opérer, à ne jamais employer de chloroforme, enfin à faire le traitement post-opératoire et suivre l'opéré jusqu'à guérison.

Pour la narcose le chlorure d'éthyle chez les enfants à raison de 1 cm^3 jusqu'à 5 ou 6 ans n'offre aucun danger.

Comme méthode opératoire, M. Trétrôp emploie l'anse froide et le morcelleur et, grâce aux soins préalables et consécutifs, il n'a jamais observé de complications immédiates ni tardives, pas même d'hémorragie.

M. Trétrôp s'associe aux conclusions des rapporteurs qui considèrent l'amygdalectomie comme une excellente opération de la pharyngologie moderne. Il convient d'en user comme de toute opération utile, mais de ne jamais en abuser.

Mr. John F. O'Malley (London). Before proceeding to discuss this question I would like to say a few words as to what I understand by the terms tonsillectomy and tonsillotomy.

To me tonsillectomy, or cutting out of tonsils, means the removal, anatomically complete, of the circumscribed mass of lymphoid tissue known as the tonsil with its capsule, by any instrument, sharp or blunt, capable of cutting or dissecting; while tonsillotomy is the incomplete removal by the same means.

Practically all the indications for any tonsil operation are included in a list of ailments which I shall mention, many of them are directly due to tonsillar disease, others are predominantly, if not exclusively, the

result of adenoid trouble, and still others are due to the combined injurious effects of both tonsils and adenoids. I am in the habit of grouping them for clinical use under the headings of ear, nose, throat, chest, and general affections, which are directly or indirectly the result of diseased tonsils and adenoids, and the question of operation in every case is considered accordingly. The following is the list referred to :—

Ear

Acute suppurative otitis media.
Chronic ditto.
Tympanic and Eustachian catarrh { Deafness { Persistent. / Recurring. / Earache—recurring.

Nose

Respiratory obstruction.
Rhinitis { Simple { Persistent discharge. / Recurring ditto. / Hypertrophic.
Epistaxis.

Throat

Tonsils { Recurring simple tonsillitis. / Follicular ditto. / Suppurative ditto. / Hypertrophied tonsils.
Pharynx { Pharyngitis. / Difficulty in swallowing.
Larynx. Laryngitis { Alteration of speech. / Recurring hoarseness.

Chest

Respiratory obstruction { Pigeon chest. / Narrow chest.
Bronchitis—recurring after head colds.
Asthma (?).

General

Recurring colds { Head (nose). / Throat. / Chest.
Enlarged cervical glands.
Poor health—child pale, pasty, peevish, and no other cause for it.
Rheumatism (?).
Chorea (?).
Epilepsy (?).
Nocturnal enuresis (?).

There is only one type of case in which I consider that tonsillotomy may conceivably be as effective as tonsillectomy. It is the case in which, on examination, the tonsils are seen to be very prominent and almost or completely meeting in the middle line. In children under ten years of age, these tonsils cause some mechanical obstruction to respiration, swallowing, and speech, but otherwise the patient is in excellent health. In older patients these tonsils are occasionally attacked by a parenchymatous inflammation. Anatomically, the lymphoid tissue or free surface is much more extensive than the attached or capsular aspect, the former being as it were mushroomed out from a narrow base, the crypts tend to be superficial, the gland feels firm to touch after removal, and owing to the limited capsule there is little or no tonsillar recess, so that this type is practically never sodden with infection nor the seat of a quinsy, and with the exception of its size it is a harmless tonsil.

In every other type of tonsil needing operative interference, tonsillectomy is indicated.

Tonsillotomy has been the operation of election for many years and it is claimed that the results were good. I am in full agreement with this view, but venture to think that they could have been better. Personally I have ceased to do tonsillotomy, and now resort to complete removal in every case for the following reasons.

The very word tonsillectomy implies in itself a definite standard of operation, and the examination of the removed tonsil provides a constant control over the efficiency of the performance, but there is no such guide by which to fix a standard of efficiency in tonsillotomy, and it is in consequence impossible to judge accurately of its merits as an alternative operation.

By this method two surgeons of decidedly different manipulative skill remove relatively different amounts of tonsils in two similar cases, with the hope of obtaining in each instance the best result. Will the result be greater or less in proportion to the amount removed, or is it immaterial how much we may take away so long as the tonsil is incised, or how can we expect definite results as a sequence of operations which depend on so many uncertain factors ?

After pondering on these questions I came to the conclusion that a tonsil should not be touched unless it is in itself diseased, or a source of, or a portal for, disease elsewhere, and therefore when interference becomes necessary, total removal is indicated.

It was this unavoidable lack of standard, and the consequent reproach of doubtful value which grew up around the uncertain results of tonsillotomy, that was largely responsible for bringing the surgery of the tonsils into some measure of ill repute. Partly for this reason the custom arose of relegating tonsillotomy to the most junior and inexperienced medical officer in the hospital, as his special surgical perquisite, a proceeding which was not calculated to improve its position.

Tonsillectomy, with a guillotine, can be successfully performed in every

case in children under fourteen years of age, and in over 90 per cent. of patients above this age who may require operation; the remaining 10 per cent. I do by Mr. Waugh's dissection method. The shortest and safest anæsthetic, namely, nitrous oxide gas, affords ample time for its performance by guillotine; ten to twelve seconds suffice for the removal of both tonsils. The affected gland is totally extirpated, and any disease dependent on it is cut off from its source of infection, the amount of the hæmorrhage is negligible, being only 3 drachms on an average in 500 cases tested, and there are no injurious sequelæ.

When a tonsil, whether enlarged or not, is the seat of infection, or is prone to recurring attacks of simple or lacunar inflammation, or when cervical adenitis or middle-ear disease is associated with it, it is manifestly bad surgery to add a further traumatic tonsillitis, to seal up infected crypts, and leave behind in the depths of the tonsillar fossa the diseased portion which is at the greatest disadvantage from the standpoint of drainage.

Dr. Harold Whale (London) discussed the remote results of tonsillotomy and tonsillectomy, based on an analytical scrutiny of 220 unselected cases. All the patients had been operated on in Mr. W. D. Harmer's clinic at St. Bartholomew's Hospital. There were 110 cases of tonsillotomy and 110 of tonsillectomy. The sexes were about equally divided; the ages at operation ranged from 3 to 43. The earliest of these operations occurred in 1908, the latest in 1912; 70 per cent. of the patients were examined at an average period of two years after operation, and all of them since May 1912. Case-cards were made recording in detail their complaints before and after the operation, the conditions found at subsequent examination, and the results of all previous operations.

Inference from figures obtained. For tonsillitis, tonsillectomy was the operation of choice; because if as much tonsillar tissue as possible was removed complete in its capsule, there was a greater prospect of permanent cure, and there was no probability of initiating an infection.

Laryngitis and functional voice troubles. Cases: tonsillotomy, 26. Result: cured, 15 (58 per cent.); not cured, 6 (23 per cent.); uncertain, 5 (19 per cent.). Cases: tonsillectomy, 29. Result: cured, 14 (48 per cent.); not cured, 14 (48 per cent.); uncertain, 1 (4 per cent.). As far as these cases were concerned it appeared from the gross figures that tonsillotomy had proved the operation of choice, and tonsillectomy was much less suitable. The failure to cure, or fresh supervention of, voice troubles after operation was in some cases referable to deformities produced by the operation. But the cases where this failure or sequela occurred without any demonstrable deformity were, after tonsillotomy 27 per cent., and after tonsillectomy 48 per cent.

Lymphadenitis. For the cure or avoidance of lymphadenitis tonsillectomy was the operation of choice. Occasionally glands were infected from a tonsillar fossa which was to all appearance quite empty, whereas the other side exhibited tonsillar remains but no glands, and this occurred

practically only after tonsillotomy. Wherefore it appeared that, if tonsillotomy were practised for the cure of lymphadenitis, it was a lesser evil to leave remains than to cut so deeply as either to leave a clean capsule or to cut into the areolar tissue beyond. In other words, tonsillotomy, if practised, should be designedly only a very partial operation. If the view were adopted that tonsillar remains tended to cause lymphadenitis, it was more important to eradicate the lower pole (with the plica triangularis) than the upper pole.

Hæmorrhage. An investigation of the records of 3,697 tonsil operations afforded strong evidence that dangerous hæmorrhage was more than twice as probable after tonsillectomy as after tonsillotomy.

Deformity and its responsibility for failure to cure the patient's complaints or for the supervention of fresh complaints unknown prior to operation. The deformities found comprised thickening, scarring, kinking, or notching of the palate, with or without lateral deviation of the uvula; overgrowth of the plica triangularis, sometimes imprisoning a tonsillar remnant; adhesion of the two tonsillar pillars together on one side, or of the posterior pillar to the posterior pharyngeal wall.

Conclusions

The disadvantages of tonsillotomy were: (1) The initiation of an infection, whether manifesting itself as tonsillitis, lymphadenitis, or both. Such sequelæ were more likely if a very free and deep removal had been performed than if only a moderate amount of the tonsil had been removed. (2) Recurrence of the trouble for which the operation was performed; except in the case of voice troubles, when recurrence was no more probable than after tonsillectomy.

The disadvantages of tonsillectomy were: (1) A risk of serious or even dangerous hæmorrhage at operation. (2) A risk of harmful deformity supervening later; the deformities most likely to cause harm were adhesion of the posterior faucial pillar to the posterior pharyngeal wall, and overgrowth of the plica triangularis. (3) A risk of voice troubles supervening later, even without any causative deformity. To any patient who required operation for some other reason, who had never had any voice trouble, and whose livelihood or occupation depended on the use of the voice, tonsillectomy should be urged with caution.

Thus it would appear that tonsillectomy was the more dangerous operation. But with the single exception of voice troubles, it was more likely to permanently cure the complaint for which advice was sought; notably so in the case of tonsillitis or lymphadenitis; moreover, these two infections were never initiated by tonsillectomy. It was important to eradicate the lower pole.

Mr. T. Jefferson Faulder (London). So far as I can gather from a rapid perusal of the reports of Professor Burger and Dr. Goodale, a strong

case is made out for tonsillectomy in practically all cases where interference with the tonsil is indicated. The arguments in favour of tonsillotomy amount to very little, and it does not require much of an enthusiast to reduce that little to nothing.

(1) With regard to the physiology of the tonsil, as Professor Burger says, 'Unsere Kenntnis ist eine ungenügende,' but a useful analogy can be drawn with the appendix, both as regards its development and its liability to infection. Surgeons are not deterred from removal of the appendix by considerations of physiological function.

(2) One of Dr. Goodale's conclusions (No. 6) is: 'The indications for operation should be determined by the pathological changes of the tonsils which are actually a detriment to the individual.'

Exactly so, and if we consider seriatim the possible indications for interference with the tonsils it will appear in most cases that we are not doing the best for the patient if we leave any part of the condemned tonsil behind. For example, Professor Burger says (No. 11), 'Bei chronischer Tonsillitis ist in der Regel die obere, im Gaumen eingebettete Mandelpartie am schwersten erkrankt.' Now the vast majority of tonsils have this upper embedded part more or less developed. Again, assuming that the tonsil is a source of general infection, such as rheumatism, the same conclusion is, as it were of necessity, reached.

The case of cervical adenitis, where due to the tonsil, is another striking example of the futility of tonsillotomy, and so on through all the lists of indications.

(3) An exception is made for tonsils which are hypertrophied, but causing no symptoms beyond those of mechanical obstruction. Here, it is said, partial removal is sufficient. So it ought to be. But unfortunately partial removal may be followed by unfavourable consequences. These tonsils, not infrequently, grow to or beyond their original size. In some of the cases also they now for the first time become liable to attacks of inflammation. In this connexion both reporters allude to the possibility of sealing up the so-called crypts by partial operations. It should be noted that all modifications of the original tonsillotomes were devised with the object of removing more and yet more tonsil tissue. One might fairly ask the question, 'What exactly is the amount of tonsil tissue which may be regarded as an adequate removal, and what is the right amount to leave?' This question is a fair one because some tonsils are not embedded at all, and there are all grades between this type and the entirely embedded tonsil.

(4) Partial removal is not unlikely to result in leaving behind what is in effect the small embedded tonsil, 'kleine versenkte Mandeln.' This was one of the reasons which led to the evolution of tonsillectomy.[1]

(5) In the case of a certain uncommon type of tonsil, namely, a tonsil

[1] From February to August of this year 1,252 cases were operated upon at Golden Square. Of these no less than 169, that is, about 14 per cent., gave a history of previous operations.

projecting into the fauces, not at all embedded and with a relatively small capsular area, that is to say pedunculated, skilful tonsillotomy becomes the same thing as tonsillectomy, and in this case, therefore, there is no point in arguing the relative advantages of the two procedures.

(6) Therefore, if tonsillectomy is in all, or nearly all, cases the best method it remains to discuss the means of carrying it out. I have performed about 1,500 tonsillectomies (that is about 3,000 tonsils) in the last two years. The majority of these were done with no other instrument than a guillotine. Professor Burger says, 'die Tonsillectomie ist technisch eine weit schwierigere Operation als die Tonsillotomie.' If by tonsillectomy Professor Burger means those operations involving the use of catch-forceps, knives, or scissors, snares, &c., an assistant, and possibly some length of anæsthesia, I entirely agree with him; but so far as my experience of tonsillectomy with the guillotine goes, I should like to be allowed to invert Professor Burger's dictum and say that effective tonsillotomy with the guillotine is more difficult than tonsillectomy with that instrument. 'If it were otherwise, the need for this discussion would not have arisen.'

(7) Reference has been made to damage to surrounding parts by tonsillectomy. I have examined carefully a very large number of tonsils excised with the guillotine, and, so far as this method of operation is concerned, one must attach its proper value to the fact that none of them show any trace of muscle tissue, except microscopically in a few specimens at the lower end of the capsule.

Dr. Goodale has said, with reference to tonsillotomy, that 'the ideal operation removes as much of the tonsil tissue as possible without injury to the pillars'. Therefore the ideal tonsillectomy is that which can remove all the tonsil tissue without injury to the pillars. This result can be obtained with rapidity and certainty in the majority of children with the amygdalotome.

(8) With regard to bleeding it seems to me that whatever operation we do there will occasionally be troublesome cases. I am, however, satisfied that the bleeding at the time of operation is, to say the least, no more in tonsillectomy properly performed with a guillotine than in tonsillotomy with the same instrument. So that I do not entirely agree with the statement, 'Sie geht mit grösserem Blutverlust einher.'

(9) With regard to the anæsthesia one great advantage of the guillotine excision, when that is available, is the shortness of the anæsthesia required. So that I must again, to some extent, dissent from Professor Burger when he says, 'Kurzdauernde Betäubungen sind für die Tonsillektomie nicht zu empfehlen.'

Dr. Luc (Paris). I thoroughly agree with Professor Burger that tonsillectomy should be performed only *exceptionally* in children, for two reasons: (1) because the operation is very difficult to perform *really completely* in the haste imposed by such short narcosis as is produced by

bromide of ethyl and other agents employed in those circumstances; (2) because the hæmorrhage to which undoubtedly it is more apt to give rise than simple amygdalotomy is extremely difficult to control at this age.

I think, on the contrary, that tonsillectomy is generally advisable in adults, since it is the surest means of putting an end to the recurrence of infection for which we have been consulted. Here local anæsthesia gives all its advantages, enabling us to perform the operation *without hurry* and consequently *thoroughly*.

As regards the technic of local anæsthesia, I cannot too much insist on the necessity of making the anæsthetic injections, not only into the pillars, but also through the deep wall of the tonsillar fossa where the glosso-pharyngeal nerve reaches the gland.

In considering the danger of hæmorrhage connected with the operation, I advocated, this year, at our French meeting, as a preventive measure, my practice of systematically suturing the pillars in all adults immediately after removing the tonsils. Under such conditions this suture is easily performed, and gives the operated patient and the operator complete security and tranquillity of mind.

Mr. Frederick C. Pybus (Newcastle-on-Tyne). (1) Except in rare instances, tonsillar disease is dependent on infection.

(2) Infection may cause (*a*) *hypertrophy* or (*b*) *gross disease*.

(3) Tonsillotomy may be sufficient for the *hypertrophy*.

(4) The great difficulty is in deciding as to when there is gross infection even in the large tonsils. The infection may be (*a*) in the lacunæ, (*b*) at the bases of the lacunæ, (*c*) encapsuled, or (*d*) the tonsils may be very small and embedded. In all these places infection is hidden. After tonsillotomy the most diseased part of the organ remains.

In the second class, infection may be (*a*) local (tonsillitis, recurrent diphtheria, lacunar plugs), (*b*) glandular (tuberculous or pyogenic adenitis), or (*c*) systemic (rheumatism, chorea, anæmia). For these cases tonsillectomy is essential.

Even without gross infection in the former class (hypertrophy) we have no guarantee that recurrence of growth will not occur and that sepsis will not supervene. The frequency of tonsillar infections and trouble persisting in adults emphasizes the need for complete removal in children.

In all cases I perform tonsillectomy—with a blunt guillotine under ethyl chloride anæsthesia—by a method described by Whillis and Pybus on January 10, 1910, at the Northumberland and Durham Medical Society, and in a paper published in the *Lancet*, September 1910.

After this operation there is no more trauma or hæmorrhage than after tonsillotomy and less than after removal by dissection. In adults I perform tonsillectomy by dissection under local anæsthesia.

Specimens are exhibited in the Museum.

Dr. S. S. Whillis (Newcastle-on-Tyne). My views on this question are already known to you from my previous publications in the *Lancet*, 1910, and *British Medical Journal*, 1911. I agree with the conclusions in the report of Dr. Goodale with few exceptions. I think there is less sepsis after tonsillectomy than tonsillotomy. It is difficult to know whether hyperplasia of the tonsils is not due to sepsis. I consider that if a tonsil has to be removed this should be done completely and not by tonsillotomy, for the whole of the disease must be got rid of.

There is no more hæmorrhage following tonsillectomy than tonsillotomy and after the former it is more readily controlled. The worst hæmorrhage is from a branch of the facial. The operation of tonsillectomy has been performed for a sufficient length of time to show any injurious effect it might have on the human economy if such were likely.

Mr. Herbert Tilley (London) said that cases of recurrence after tonsillotomy—often after an interval of ten years—had led him to adopt tonsillectomy in 95 per cent. of his cases. It was impossible to foretell such a recurrence when the case was first seen, even in simple enlargement of the tonsils without apparent disease.

He had met with many recurrences in his own practice as well as in the patients of others who could not be accused of having performed the tonsillotomy inefficiently.

He did not think that post-operative hæmorrhage was more common in a well-executed tonsillectomy than in tonsillotomy, and especially if the surgeon took care to see that any free bleeding had ceased before the patient was allowed to recover from the anæsthesia. Possibly tonsillectomy required a few days longer convalescence, but surely that time was well spent if a certain and permanent cure could be assured.

Dr. W. S. Syme (Glasgow) said a strong argument in favour of tonsillectomy was that since its introduction more and more cases on which tonsillotomy had been performed were being sent back for the complete operation. The general practitioner had come to look upon tonsillotomy as necessarily more or less unsatisfactory. The question was not between tonsillectomy and tonsillotomy but between tonsillectomy and leaving the tonsil alone. When, however, one tried to decide what tonsils might safely be left one was met with much difficulty, and this especially arose when one operated on cases of adenoids where the tonsils were little, if at all, changed in appearance. He used chloride of ethyl anæsthesia in the majority of cases. When he used chloroform he did not look upon the deep and dangerous anæsthesia, which some insisted on, as necessary.

He did not think that the enucleation of the tonsil by some form of guillotine was a satisfactory general procedure, as he considered it not sufficiently definite, for one could not be sure that in all cases the aim of the operation could be accomplished. If one determined to remove the

tonsil completely a definite method should be adopted and this meant some form of dissection.

Sir StClair Thomson (London) felt that he must protest against the suggestion made by Dr. Syme that complete enucleation of the tonsils could only be carried out by a method of dissection. In his own text-book, published two years ago, Sir StClair had demonstrated that complete enucleation of the tonsil in its uninjured capsule could be rapidly accomplished with the tonsillotome. This chapter had been written a year or two previously. In fact, he had been carrying out complete enucleation of the tonsils with the guillotine for the last five years. The writings of Sluder, Pybus, and others, had abundantly proved the same thing. He had no objection to enucleation by other methods, but he very much doubted if the operation could be performed as rapidly, safely, and with as little traumatism as it could be with the ordinary tonsil guillotine.

Dr. Broeckaert (Gand). Tout en n'étant pas un massacreur d'amygdales, il faut rendre hommage aux efforts des Américains qui ont montré que la vieille amygdalotomie est souvent insuffisante quand il s'agit d'*amygdales infectantes*. Toutefois l'amygdalotomie faite avec *libération préalable de l'amygdale*, surtout au niveau du pôle supérieur, devient souvent, même chez les enfants, une véritable amygdalectomie L'amygdalectomie à l'américaine, opération sanglante et nullement inoffensive, doit être réservée à des cas tout à fait spéciaux et seulement quand l'amygdalectomie faite d'après des procédés moins radicaux n'a pas atteint son but.

Mr. W. H. Kelson (London). In each case one must determine by careful examination whether simple hypertrophy or disease is present. *Tonsillotomy* must still be considered the best operation for children in whom simple enlargement causes symptoms by interfering with breathing and deglutition or produces deafness. These form the bulk of the patients, and experience shows that in the vast majority of them a permanent cure is obtained by tonsillotomy.

Tonsillectomy is clearly indicated in cases of diseased tonsils, large or small, but it must be considered the more severe operation because (1) deeper anæsthesia is used, and (2) the convalescent period is longer.

Professor Kubo (Japan). Die Tonsillektomie wird heute auch in Japan von manchen Spezialisten so gerne ausgeführt, wie man früher Turbinektomie statt Conchotomia partialis machte, was aber heutzutage fast gänzlich ausser Mode geworden ist. Obgleich deutliche nachteilige Folgen nach Tonsillektomie in Bezug auf den ganzen Organismus noch nicht beobachtet wurden, so ist diese Operation nicht als ungleichgültig zu betrachten, da sie zeitweise heftige Nachblutung mit sich bringt, trotzdem wichtige voroperative Untersuchungen vorgenommen worden

sind. Ausserdem ist die Operation nicht so leicht wie die Tonsillotomie (bei uns ohne Anästhesierung operiert). Infektionsgefahr nach Operation ist hierbei grösser. Man muss sehr vorsichtig sein, bei solchen Kranken Tonsillektomie vorzunehmen, denen die Stimme beruflich wichtig ist. Tonsillektomie ist bei gewissen Fällen unvermeidlich, wie z. B. bei rezidivierender Tonsillitis, oder im Anfangstadium von primären malignen Tumoren der Tonsille.

Ich möchte zum Schlusse über einen interessanten Fall berichten, der einen 14 jährigen Knaben betraf. Derselbe wurde wegen Nephritis subacuta von einem Kollegen der Kinderheilkunde sechs Monate lang ohne Erfolg behandelt, trotzdem alle Massregeln, z. B. absolute Ruhe, Diät, Arzneimittel etc., berücksichtigt worden waren. Der Fall wurde anfangs von heftiger Epistaxis begleitet. Die Eiweissmenge im Harn nahm manchmal zu, trotz absoluter Ruhe, wenn einmal leichte entzündliche Erscheinungen an den Gaumentonsillen vorkamen. Aber die Entzündung der Tonsillen war dabei gar nicht hochgradig. Da die Beziehung zwischen Eiweissharn und Gaumentonsillen nicht mehr zu bezweifeln war, empfahl ich den Eltern Tonsillektomie, die aber von ihnen nicht bewilligt wurde. Nach einer Tonsillotomie mit Schlitzer und Schlinge in zwei Sitzungen wurde die Eiweissmenge deutlich vermindert und die Zylinder waren nicht mehr zu konstatieren. Zehn Tage nach der Operation war Eiweiss nicht mehr im Harn zu sehen und der Knabe so weit gesund, dass er die Schule besuchen konnte. In solch einem Falle wäre eine totale Ausrottung der Gaumentonsillen sehr geeignet. Es ist aber sehr bemerkenswert, dass der betreffende Fall durch Tonsillotomie doch zur Heilung kam. Daher möchte ich die Tonsillektomie besonders bei einfacher Hypertrophie bis dahin ersparen, wo die Tonsillotomie erfolglos ist.

Dr. P. Watson-Williams (Bristol). That healthy tonsils are protective is suggested by the relative infrequency with which those who have tuberculous glands in the neck develop pulmonary tubercle. I believe that acute systemic rheumatic infection is sometimes actually prevented by the occurrence of acute rheumatic tonsillitis, and that absence of healthy tonsils renders the patient more open to systemic infection. On the other hand, when the lymphoid tissue aggregations are so profoundly infected that they become themselves hotbeds of recurring infection, it is much better to remove *in toto* what is worse than useless. But the routine complete removal of the tonsils, even if not definitely pathological, is surely to be deprecated. I do not find tonsillectomy more difficult, or more liable to result in traumatic deformity or in post-operative complications than the 'so-called' simpler procedure of tonsillotomy. And if we, as a rule, remove the supra-tonsillar fossa, we, at any rate, remove the upper pole and the body of the tonsil.

In young children then, I would reserve tonsillectomy for definitely infected tonsils, or where systemic infection has arisen; in adults tonsil-

lectomy is to be preferred when there is occasion to operate at all. By the method of Whillis and Pybus, and of Sluder, in America, tonsillectomy in children can be performed under nitrous oxide gas, or ethyl chloride anæsthesia. In adults local anæsthesia suffices though ether has advantages, since a more deliberate dissection is often necessary.

Dr. Wilhelm Sobernheim (Berlin). Meine Stellung zur Tonsillektomiefrage gestatte ich mir folgendermassen zu präzisieren : Ein maligner Tumor der Tonsille verlangt die völlige Entfernung der Mandel ; auch die Tuberkulose, soweit sie nicht das Endstadium einer allgemeinen Tuberkulose darstellt, und sofern sie in den oberen Luftwegen auf die Tonsille beschränkt ist, erfordert die Enukleation, ohne dass man die Verschleppung des tuberkulösen Virus befürchten müsse. Die schleunige Entfernung bei luetischem Primäraffekt kann die Ausbreitung der Krankheit verhindern. Bei habitueller Angina und rezidivierender Peritonsillitis ist bei dauernder Störung des Allgemeinbefindens die Tonsillektomie den weniger radikalen Methoden vorzuziehen. Bei Hyperplasie der Tonsille, sofern sie ein Atemhindernis darstellt, genügt die Tonsillotomie ; im Kindesalter ist diese prinzipiell vorzunehmen. Bei der Tonsillitis chronica wird man mit Schlitzung oder Entfernung des oberen Pols auskommen, wenn nicht Komplikationen, wie Gelenkrheumatismus, Endokarditis etc., die Tonsillektomie nötig machen.

Dr. Friedrich (Kiel). Die innere Medizin gibt dem Laryngologen häufig die Indikation zur Tonsillotomie und Tonsillektomie auf der Grundlage von allgemeinen infektiösen Krankheiten, wie Nephritis, Myokarditis, Drüsenfieber u.s.w. Der Halsarzt soll prüfen, ob in der Tat die Beziehungen zwischen allgemeinen Infektionen und chronischen Tonsillitiden so häufig sind, wie behauptet wird. Die Indikation zur Art der Entfernung ist verschieden bei Kindern und bei Erwachsenen. Bei den ersteren handelt es sich gewöhnlich um hyperplastische grosse Tonsillen, hier ist die Exstirpation — gewöhnlich in Verbindung mit der für Infektionen noch wichtigeren Rachentonsille — indiziert. Bei Erwachsenen erhalten wir in den unserer Therapie überwiesenen Fällen, ausser den hyperplastischen Fällen, in denen die Tonsillotomie indiziert ist, häufig Kranke mit kleinen derben, flachen, kaum über die Gaumenbogen hervorragenden Tonsillenresten. Hier ist die Tonsillektomie indiziert. Massgebend ist das Aussehen, die Zerklüftung und Narbenbildung für die Indikation zu jeder Art der Exstirpation. Die Indikation muss vom Laryngologen, nicht vom Internisten gegeben werden.

Dr. H. Marschik (Wien). Die theoretischen Erörterungen über den Wert und die Indikation von Tonsillotomie und Tonsillektomie sollten nur den Referenten reserviert bleiben und die einzelnen Diskussionsredner sich auf persönliche Erfahrungen und einzelne bemerkenswerte Fälle beschränken. Der Standpunkt der Klinik Chiari in Wien hat

sich in letzter Zeit mehr zugunsten der Tonsillektomie verschoben. Dagegen ist das Morcellement wegen der Blutungen und wegen des unsicheren Erfolges nicht beliebt. In einer Anzahl von Fällen bewies die Entdeckung von kleinen eiter- und streptokokkenhaltigen Abszessen dicht unter der Kapsel die Wichtigkeit radikalen Vorgehens bei erkrankten Tonsillen. Auch mehrere Fälle von geheilter Nephritis und Albuminurie zeigen den unzweifelhaften Zusammenhang zwischen diesen Erkrankungen und den Tonsillen. Drei Fälle von merkwürdigen endokraniellen Erkrankungen, den Bildern von Poliomyelitis oder Meningitis basalis, die gleichzeitig starke Eiterung der Tonsillen hatten, wurden durch Tonsillektomie zur Heilung gebracht. Desgleichen drei Fälle eigenartiger rezidivierender Halsphlegmone, von denen zwei durch konservative Therapie stets wieder gebessert werden konnten, einer aber mehrfache Incisionen erdulden musste, wurden durch Tonsillektomie endgiltig geheilt.

Dr. W. M. Stookey (Salt Lake City, U.S.A.). In America to-day the consensus of opinion is very largely in favour of tonsillectomy, and the results obtained from this operation are vastly better than those obtained from tonsillotomy.

This discussion has brought out the following points :

(1) The functions of the tonsil are still unknown.

(2) Tonsillectomy gives vastly better results than tonsillotomy.

(3) The dangers of tonsillectomy in skilled hands are scarcely more than those of tonsillotomy.

(4) Statistics given in this section of the Congress to-day show that after tonsillotomy there are 25 to 50 per cent. of recurrences in which further work must be done, while following tonsillectomy there is very seldom a recurrence.

(5) Up to the present date no detrimental conditions have been found to follow the complete removal of tonsils at any age.

(6) Tonsillectomy may not be indicated in all cases in small children, but when tonsillotomy is done parents should be given to understand that the more radical operation may become necessary.

(7) Only a greater knowledge of the function of the tonsil can determine the tonsillar surgery of the future. At the present day, being guided by results to the patient, we must say that if there is to be any surgical interference with the tonsil, tonsillectomy should be preferred in practically all cases.

Dr. M. Halle (Berlin). Es wird viel zu wenig Wert auf strikte Indikation gelegt. Sicherlich ist es nicht schwer, eine Tonsillektomie auch bei Kindern in jedem Falle auszuführen. Aber ich halte eine solche Operation für absolut kontraindiziert so lange nicht *besondere* Indikationen dafür vorliegen. Wenn immer wir eine funktionelle Bedeutung der Tonsille anerkennen, — und es ist kein Zweifel, dass sie eine solche

haben, — dann haben sie diese besonders in der Kindheit. Es geht nicht an, hier bei einer einfachen Tonsillenhypertrophie die Tonsillenexstirpation zu machen. Das heisst, meines Erachtens, nicht mehr Arzt, sondern Techniker sein ; das heisst, die Tonsillen als maligne Tumoren anzusehen. Bei meinen Fällen, die ich sorgfältig kontrolliere, sehe ich in nur wenigen Prozent der Fälle spätere Erkrankungen, Anginen u. dgl., und solche habe ich auch nach Tonsillenexstirpation gesehen. Kommen solche Rezidive in der Kindheit vor, so kann man den Eltern ruhig sagen, dass man gehofft hat, mit der Tonsillotomie auszukommen, dass es aber sich gezeigt hat, dass eine weitere radikale Operation unvermeidlich ist. Die Eltern werden es durchaus verstehen, dass man zu Anfang konservativ vorging, und es dem Arzt danken. Deswegen aber, weil einige erkranken, bei allen Kindern die Tonsillenexstirpation zu machen, halte ich für absurd. Ausgenommen hiervon sind alle Fälle von Sekundärerkrankungen durch Affektion der Tonsillen, besonders bei eingebetteten Tonsillen. Hier kann nur die Exstirpation in Frage kommen.

Ebenso sind die chronischen Erkrankungen der Tonsillen bei Erwachsenen zu behandeln. Hier führt keine konservative Behandlung zum Ziel, auch nicht das Morcellement. Und das wird leicht zu verstehen sein, wenn man sieht, dass in vielen Fällen dicht an der Kapsel Abszess an Abszess sitzt. Auch hier kann nur die Exstirpation indiziert sein, die ich immer unter lokaler Anästhesie und schmerzlos ausführe.

Professor Uchermann (Christiania). I only want to add a few remarks to what Dr. Halle has said. I cannot see the sound principle of removing *in toto* an organ that is not infected, from fear of its becoming so ; such cases are always in a minority. It would be wiser then, in common cases of hyperplastic tonsils, to remove only what is too much and to run the risk of recurrence. I also think that most patients would prefer this plan.

Reply

Professor H. Burger (Amsterdam) insisted upon the danger of anæsthesia in tonsil operations. He always performed tonsillotomy and adenotomy without narcosis. Tonsillectomy, however, required narcosis, and he thought this a strong argument in favour of tonsillotomy as the general operation in childhood. They were not fully informed as to the physiological function of tonsils ; was it therefore right to systematically remove them in children ? He was gaining the impression that the laryngologists claimed to be wiser than the Lord.

SECTION XV

RHINOLOGY AND LARYNGOLOGY

INDEPENDENT PAPER

OPERATIVE BEHANDLUNG DER HYPOPHYSEN-TUMOREN

Von Dr. OSKAR HIRSCH, Wien

Mein Vortrag beschäftigt sich mit der operativen Behandlung der Hypophysentumoren nach endonasalen Methoden. Ich würde Ihnen gern einige einleitende Worte über die Funktion der normalen und über die Symptome der pathologischen Hypophyse sagen, muss aber wegen der Kürze der Zeit *in medias res* eingehen.

Ich halte es für angezeigt, Ihnen zuerst die Frage zu beantworten: *Wie sehen die Tumoren aus, die wir zu behandeln haben?*

Vom operativen Standpunkte können wir die Hypophysentumoren in drei Gruppen einteilen:

I. Gruppe: intrasellare oder vorwiegend intrasellare *solide* Tumoren. Das sind diejenigen Tumoren, welche ausschliesslich oder vorwiegend auf die Sella beschränkt sind, diese gegen die Keilbeinhöhle vorwölben, dagegen über das Diaphragma nur wenig oder gar nicht emporwachsen. (Bild 1 und 2.)

II. Gruppe: vorwiegend intracranielle, *solide* Tumoren. Das sind solche Tumoren, welche die Sella mehr oder minder gegen die Keilbeinhöhle vertiefen, ausserdem aber aus der Sella hinauswachsen und sich in die Gehirnbasis eingraben oder in diese hineinwachsen. (Bild 3 und 4.)

III. Gruppe: das sind solche Tumoren, welche aus einer einzigen grossen Cyste bestehen oder eine Cyste — meist ist es eine Erweichungscyste — enthalten. Die cystischen Tumoren können intrasellare oder intracranielle Geschwülste sein.

Können wir in vivo diagnostizieren, welcher von diesen Gruppen ein Hypophysentumor angehört?

Am Lebenden können wir die Entscheidung, ob es sich um einen soliden oder cystischen Tumor handelt, nicht treffen. Diese kann erst durch die Operation gebracht werden. Dagegen können wir mit grosser

Wahrscheinlichkeit sagen, ob es sich um einen intrasellaren oder intracraniellen Tumor handelt. Ich habe zur Entscheidung dieser Frage die Topographie der Hypophyse herangezogen (Bild 5). Sie sehen auf diesem Bilde, dass das Chiasma opticum von der Hypophyse durch einen tiefen Subarachnoidalraum getrennt ist, der sog. Cisterna chiasmatis, worauf bereits Zander hingewiesen hat. Damit ein Hypophysentumor Sehstörungen hervorrufe, muss er über den Sellaeingang hinauswachsen, die Cisterna chiasmatis ausfüllen und das Chiasma gegen die Gefässe drücken und es komprimieren. Wir können daher mit grosser Wahrscheinlichkeit annehmen, dass die Tumoren, die keine Sehstörungen verursachen, intrasellar oder vorwiegend intrasellar entwickelt sein werden, während diejenigen Tumoren, die Sehstörungen hervorrufen, mit einem Abschnitt in die Schädelhöhle hineinragen, das heisst intracraniell sitzen müssen.

Auch das Röntgenbild gestattet, auf den Sitz des Tumors zu schliessen, indem bei den intrasellaren Tumoren die Sella gegen die Keilbeinhöhle stark vorgewölbt, ihr Eingang aber nicht erweitert erscheint (Bild 6), während bei den intracraniellen Tumoren neben einer mehr oder minder starken Ausbuchtung der Sella auch noch eine Erweiterung des Sellaeinganges festzustellen ist (Bild 7).

Nachdem ich Ihnen die Beschaffenheit der zu behandelnden Tumoren gezeigt habe, will ich nun die *Methoden demonstrieren, die zur Behandlung dieser Geschwülste bisher angewendet wurden.*

Ueber jene Methoden, die am Lebenden noch nicht erprobt worden sind, will ich diesmal nicht sprechen.

Die älteste Methode ist die von Horsley (Bild 8).

Horsley sucht die Hypophyse von der mittleren Schädelgrube durch Emporheben des Temporallappens zu erreichen.

Die zweite Methode ist die von Krause. Sie führt durch die vordere Schädelgrube (Bild 9).

Solange die Hypophysenchirurgie nur über *diese beiden intracraniellen Methoden verfügte,* wurden nur wenige Fälle operiert.

Einen Aufschwung erhielt die Hypophysenchirurgie durch die Methode Schloffers (Bild 10).

Nach Aufklappung der Nase werden das Septum und die Muscheln exzidiert und die Siebbeinhöhlen beiderseits ausgeräumt. Hierauf wird die auf diese Weise freigelegte Keilbeinhöhle eröffnet, die nun sichtbar gewordene Sattelgrube aufgemeisselt und der Tumor mit scharfem Löffel excochleiert. Um Vervollkommnung und Verbreitung dieser Methode haben sich insbesondere v. Eiselsberg, Hochenegg und Kocher verdient gemacht.

Diese Methode wurde von Kanavel dahin modifiziert, dass die Nase mittels eines U-förmigen Schnittes nach oben geklappt wird, worauf nach partieller Resektion des Septums die Keilbeinwände freigelegt und eröffnet werden (Bild 11). Diese Methode wurde wiederum von Halstead verbessert, indem dieser Autor den Schnitt unter die Lippe verlegt

(Bild 12). Eine weitere Modifikation der Schloffer'schen Methode rührt von Chiari her. Dieser räumt von einem äusseren Schnitt in der Gegend des medialen Augenwinkels das Siebbein aus und legt nach partieller Septumresektion die vorderen Keilbeinwände frei (Bild 13).

Gleich nach den ersten bekannt gewordenen Hypophysenoperationen, welche nach der Methode Schloffers ausgeführt worden sind, schlug ich vor, die Hypophysenoperation auf endonasalem Wege vorzunehmen. Ich gab zwei Methoden an, von denen ich die erste die ethmoidale, die zweite die septale Methode nannte (Bild 14).

Die ethmoidale Methode beruht auf der Eröffnung *einer* Keilbeinhöhle nach vorausgegangener Ausräumung der Siebbeinzellen derselben Seite. Diese Methode wird in Lokalanästhesie in 3–4 Sitzungen ausgeführt.

In der ersten Sitzung wird die mittlere Muschel entfernt, in einer zweiten Sitzung werden die Siebbeinzellen derselben Seite ausgeräumt, worauf dann in einer 3. Sitzung die vordere Keilbeinwand abgetragen und die ausgeweitete Sattelgrube eröffnet wird (Bild 15).

Bei der septalen Methode, welche gleichfalls in Lokalanästhesie ausgeführt wird, erfolgt die Freilegung der *beiden* vorderen Keilbeinwände durch die submuköse Resektion des Septums. Diese Methode wandte ich bis auf zwei Fälle in allen übrigen an. Der Verlauf der Operation ist kurz skizziert folgender :.

Die mittleren Muscheln werden beiderseits in einer Sitzung reseziert. Nach ca. einer Woche nehme ich die eigentliche Operation vor. Die Schleimhäute des Septums werden beiderseits mit 20 Prozent Cocainlösung anästhesiert, dann wird die ganze Schleimhaut des Septums beiderseits mit Schleich'scher Lösung infiltriert. Zum Schluss werden auch die Recessus sphenoethmoidales mittels in Cocain getauchter Watteträger cocainisiert. Von einem Schnitt am vorderen Knorpelrand wird das Septum bis an die vorderen Keilbeinwände reseziert, hierauf die Keilbeinwände mit Knochenstanzen abgetragen, bis die erweiterte Sella sichtbar wird. Schliesslich wird auch diese aufgemeisselt und der Tumor excochleiert.

Eine meiner Methode sehr ähnliche ist die von Cushing, welche die vorderen Keilbeinhöhlen gleichfalls auf dem Wege der submukösen Septumresektion eröffnet, doch geschieht dieser Eingriff von einem Schnitt unterhalb der Oberlippe und in Narkose.

Sie sehen, dass reichlich Methoden vorhanden sind, um an die Hypophysentumoren heranzukommen.

Welches ist nun die Methode, die wir als die derzeit beste anzusehen haben ?

Allgemein beantwortet ist es jene Methode, die bei geringster Mortalität die radikalste Entfernung der Tumoren gestattet. Wir müssen nun sämtliche Methoden nach diesen beiden Richtungen hin prüfen.

Ueber die Mortalität bei den einzelnen Methoden soll Ihnen die folgende Tabelle Aufschluss geben :

Methode.	*Zahl der Fälle.*	*Todesfälle.*	*Mortalität.*
Horsley	17 (5 nicht beendet)	5	41 %
Krause	5 (2 nicht beendet)	—	Undurchführbar 40 %
Schloffer	49	17	34·7 %
Kanavel-Halstead .	11	4	36 %
Cushing	29 (10 sellare Decompress.)	4	13·7 %
Hirsch	37 (50 Operationen)	5	13·6 % (10 %)

Sie sehen also, dass die endonasale Methode betreffs Mortalität die günstigsten Resultate liefert und dass in dieser Hinsicht nur die auf denselben Prinzipien basierende Methode von Cushing ihr gleichkommt.

Jetzt haben wir die Methoden nach der zweiten Richtung hin zu betrachten, *welche dieser Methoden die radikalste Entfernung gestattet.*

Um diese Frage zu beantworten, muss ich auf die anfangs gegebene Einteilung der Hypophysentumoren zurückkommen. Die intracraniell oder vorwiegend intracraniell gelegenen Tumoren sind für keine der genannten Methoden radikal entfernbar, für die nasalen Methoden deshalb nicht, weil der in der Gehirnbasis liegende Teil bei den nasalen Operationen nicht erreicht werden kann, für die intracraniellen Methoden (Horsley und Krause) sind diese Tumoren deshalb nicht entfernbar, weil sie vom Boden des III. Ventrikels nicht ablösbar sind. Meist handelt es sich um maligne Tumoren. Eine radikale Entfernung gestatten nur die intrasellaren und cystischen Tumoren.

Da also die intracraniellen Hypophysentumoren für keine Methode radikal entfernbar sind, die intrasellaren und cystischen Tumoren für alle Methoden gleich leicht entfernbar sind, wird für die Wahl der Methode deren relative Ungefährlichkeit massgebend sein.

Von diesem Standpunkte aus müssen wir die in Lokalanästhesie ausführbare endonasale Methode als die empfehlenswerteste betrachten. Neben dieser kommt nur noch die ihr verwandte Methode Cushings in Betracht.

Welches sind die Erfolge der endonasalen Hypophysenoperation?

Von den 37 auf endonasalem Wege operierten Fällen — in dieser Zahl sind die beiden Fälle von Holmgren und Spiess mitgezählt — *starben* 5 *Patienten*, zwei an Meningitis, einer an Pneumonie, einer an Blutung in dem III. Ventrikel und einer im Coma diabeticum. 4 Fälle wurden obduziert. Es fanden sich überaus grosse, intracraniell liegende, maligne Geschwülste.

Bei *vier Patienten* blieb die Operation ohne Einfluss, bei *fünf Patienten* wurden vorübergehende 2–6 Monate anhaltende Erfolge erzielt.

Bei den übrigen 21 *Patienten* wurden zum Teil sehr bedeutende und lang andauernde Besserungen erzielt.

Unter diesen gebesserten Patienten befinden sich solche, welche der Erblindung nahe waren, durch die Operation aber wieder berufsfähig wurden, ferner Patienten mit Störungen der Intelligenz, des Gedächtnisses und sonstigen psychischen Anomalien, welche geistig wieder leistungs-

fähig wurden, und schliesslich zwei Patientinnen mit Verlust der Menses, bei denen nach der Operation die Menses wiederkehrten.

Ich demonstriere Ihnen ein Resultat für viele.

Die Heilungsdauer beträgt bei den ersten von mir operierten Fällen nahezu 3 Jahre.

Wovon hängt der Erfolg der Hypophysenoperation ab?

Der Erfolg hängt zum grossen Teil von der Art und Grösse des Tumors ab, zum anderen Teil von der Erholungsfähigkeit der Sehbahnen und der Rückbildungsmöglichkeit der trophischen Veränderungen.

Für den Erfolg der Operation ist es von grosser Wichtigkeit, ob es sich um einen intracraniell-soliden oder um einen intrasellaren oder um einen cystischen Tumor handelt. Die *intrasellaren und cystischen Tumoren* geben die besten Chancen für eine ausgiebige und andauernde Besserung. Leider werden die Hypophysentumoren im Stadium des intrasellaren Sitzes ziemlich selten diagnostiziert. Ich habe die Gründe in meiner letzten Arbeit auseinandergesetzt. Unter meinen Fällen waren zwei intrasellare Tumoren, beide waren Fälle von Akromegalie. Die Zahl meiner Fälle mit cystischen Tumoren betrug sechs.

Bei den *intracraniell gelegenen soliden* Tumoren kann durch die Entfernung des Sellabodens und des intrasellaren Tumorabschnittes nur eine Druckentlastung herbeigeführt werden. Diese genügt jedoch in der Mehrzahl der Fälle, um eine oft bedeutende Besserung der Lokalsymptome, d. i. des Visus, nicht selten auch einzelner Allgemeinsymptome herbeizuführen. Der *Grad der Besserung* hängt vom Grad der Druckentlastung, die *Dauer des Erfolges* von der Wachstumsgeschwindigkeit des Tumors ab. Maligne und rasch wachsende Geschwülste werden den Effekt der Operation rascher aufheben als gutartige und langsam wachsende Geschwülste. Sie sehen, dass wir die endonasale Hypophysenoperation als eine erfolgreiche Methode ansehen können, sofern wir von ihr nicht das verlangen, was keine Methode bisher leisten kann, die Heilung maligner Geschwülste.

DISCUSSION

Dr. W. Hill (London). Diagrams of the following commoner types of per-nasal operations on the pituitary fossa were thrown on the screen.

(1) Schloffer's operation with v. Bruns' displacement of the external nose after v. Eiselsberg.

(2) Schloffer's operation with Ollier's displacement after Proust.

(3) The same according to Kocher.

(4) The same according to Dialti.

(5) Septal route operation with v. Bruns' displacement.

(6) Kanavel's method commencing with Lariche's naso-labial incision.

(7) Halstead's operation commencing with Rouge's infra-labial incision —favoured by Cushing.

(8) Hirsch's endo-nasal septal method.

(9) Graham's minor rhinotomic incisions through top of nose or through the ala, auxiliary to Hirsch's operation.

Enlargement of the ala upwards and forwards was not required in acromegalic subjects, but where the nostrils were small it gave all the extra access required, and rendered the various larger displacement rhinotomies unnecessary in most cases, and one was actually nearer the fossa than in the favoured method of Cushing (Halstead's operation). The septal route was not always bloodless, as there might be troublesome bleeding in acromegalic patients as he had experienced in a case of his own. The latter died from increased intra-cranial pressure owing to the formation of a large post-operative hæmatoma beneath the capsule of the supra-sellar part of the tumour. (Slide shown.)

Skiagrams of three patients with pituitary tumours were also exhibited.

Dr. Marschik (Wien). Nach Chiari's Methode wurden in Wien bis jetzt drei Fälle von Hypophysentumor operiert. Diese Methode hat nach der Angabe Chiaris den Vorteil des kürzern Weges zur Sella turcica und des grösseren Operationsfeldes. Alle drei Fälle wurden wegen hochgradiger Sehstörungen operiert und durch die Operation im Sehvermögen wesentlich gebessert. Unmittelbaren Todesfall nach der Operation hatten wir *keinen*. Ein Fall starb sechs Monate später an der Ausbreitung des malignen inoperablen Tumors in die Umgebung und das Gehirn, zwei Fälle leben noch in gutem Zustande ein und zwei Jahre nach der Operation.

Dr. J. Broeckaert (Gand). Je tiens à rendre hommage à l'habileté opératoire du collègue Hirsch, qui a dans le domaine de la chirurgie de l'hypophyse une expérience en quelque sorte unique. Qu'il me soit permis toutefois de faire quelques remarques à propos de son intéressante communication. Si l'on étudie avec soin les nombreux cas qu'il a relatés on arrive à la conclusion que l'opération, telle qu'elle a été pratiquée, n'a pas toujours répondu au but, soit 1° à cause de difficultés techniques, 2° parce que la tumeur était devenue inopérable, 3° parce que la voie suivie n'a permis qu'une exérèse absolument illusoire. C'est pour ces motifs qu'il importe de faire toujours au préalable une étude attentive de l'image radioscopique et de voir quelle est la meilleure voie à suivre. Dans un certain nombre de ces cas, où la réussite a été incomplète ou nulle, la *voie transpalatine*, que j'ai décrite depuis longtemps, et qui a été suivie avec succès dans nombre de cas, entre autres par Freysing, de Cologne, aurait été toute indiquée : par ce procédé on arrive sur l'hypophyse par la voie la plus courte, non pas suivant une ligne pour ainsi dire *parallèle* à la selle turcique, mais perpendiculairement, avec possibilité d'explorer au doigt la cavité hypophysaire.

Je conclus donc en disant que la méthode de Hirsch est incontestablement une excellente méthode pour les rhinologistes, mais qu'il ne faut pas systématiquement rejeter les autres méthodes dont celle par voie transpalatine peut rendre les plus grands services.

Dr. Halle (Berlin). Mir scheint, dass für den Rhinologen die Operation nach Cushing kaum Vorteile hat. Wir können die Keilbeinhöhle von der Nase genau so gut übersehen, wie von der Mundöffnung her. Der grosse Vorzug bei der nasalen Operation ist, dass man in lokaler Anästhesie operieren kann, und dass man jederzeit im Stande ist, an das Operationsfeld wieder heranzukommen. Das zeigte sich auch von grösstem Wert bei einem Falle von Hypophysenerkrankung (Cyste), die ich vor kurzem operiert habe. Während der ersten acht Tage nach der Operation hatte der Patient nicht einmal höhere Temperatur als 37°. Als ich dann aber den Tampon fortliess, traten zwei Tage später schwere Allgemeinerscheinungen auf mit 40,2°. Ich öffnete die beiden Mucosalappen wieder und fand dazwischen reichliches Sekret, das aus dem Operationsfeld kam. Durch neue Drainierung gelang es, das Fieber bald zum Sinken zu bringen. Bei anderer Methodik dürfte es nicht immer leicht sein, das Operationsfeld zu beherrschen.

Dr. F. H. Westmacott (Manchester). The transnasal route was difficult for the general surgeon unaccustomed to the anatomy of the internal nose. The route of operation had to be determined, however, by the character of the tumour. The only case he had seen operated on was one in which he assisted Mr. Thorburn of Manchester. In this, a fluctuant swelling the size of a grape presented in the sphenoidal sinus. The symptoms were relieved, but on removal of the projecting mass, covered with granulations, by means of a nasal snare, death took place from encephalitis. Post-mortem, a large glioma was found to occupy the major part of the base of the skull.

Dr. John M. West (Berlin). My experience with tumours of the hypophysis has been extremely limited. I have only had an opportunity of operating upon three cases. The first was a case of acromegaly and I found a cyst. The second was one of the dystrophia genitalis type. At the operation I found a fairly large tumour, which I considered best not to attempt to remove. The third case was also one of tumour, with only blindness as a symptom, and again I did a decompression operation of the sella turcica but did not remove the tumour. In all the cases the hæmorrhage was very scanty. There was no mortality.

All three cases were operated on by an intra-nasal method which I had worked out on the cadaver and published in the *Archiv f. Laryng.* and the *Journal of the American Medical Association.* The method consists in removing a window from the posterior part of the septum. Through this window one can see into both sphenoidal sinuses after removing their anterior walls.

Dr. Holmgren (Stockholm). I have operated in only one case by Hirsch's method. This patient's vision a few days before the operation was only $\frac{1}{10}$ and it diminished rapidly. Some days after the operation

he was absolutely blind, and did not see a big electric light near his eyes; fourteen days later he could walk about alone, and one month later he could do his ordinary work.

Reply

Dr. O. Hirsch (Wien). Mit grosser Genugtuung entnehme ich aus der Diskussion, dass das Interesse der Rhinologen für die Hypophysenchirurgie zunimmt. Einzelne Redner haben andere Wege als die von mir besprochenen empfohlen. Ohne auf Einzelheiten einzugehen, möchte ich darauf hinweisen, dass es uns an Methoden nicht gebricht, insbesonders nicht an solchen, welche die Hypophysentumoren von der Keilbeinhöhle aus zu erreichen suchen. Allen diesen Methoden sind in Bezug auf ihre Leistungsfähigkeit jene Grenzen gezogen, die ich in meinem Bericht gezogen habe. Wir werden also bei der Wahl der Methode *jenen Operationen den Vorzug geben müssen, welche sich in der Praxis bereits als die am wenigsten gefährlichen erwiesen haben.* Dies sind zweifellos die von mir angegebenen endonasalen Methoden. Um üble Zufälle bei den Hypophysenoperationen möglichst zu vermeiden, ist nicht nur das Studium der normalen Anatomie der Hypophysengegend empfehlenswert, sondern auch das Studium der anatomischen Verhältnisse dieser Gegend, wie sie durch die Hypophysentumoren geschaffen werden, die ganz verschieden sind von der normalen Anatomie.

SECTION XV

RHINOLOGY AND LARYNGOLOGY

INDEPENDENT PAPER

DIE AKUT-INFEKTIÖSEN HALSENTZÜNDUNGEN

Von Professor A. KUTTNER, Berlin

Jahrhunderte lang sind die allerverschiedensten Erkrankungen des Rachens, des Schlundes und des Kehlkopfes unter dem Sammelbegriff der Synanche (Angina) zusammengefasst worden. Die Untersuchungsmethoden waren noch nicht genügend ausgebildet, um in der Mannigfaltigkeit der Erscheinungen das Wesentliche vom Unwesentlichen zu scheiden und danach die verschiedenen Krankheitsformen zu gruppieren und einzurangieren. So hielt man sich an das nächstliegende, an das am meisten in die Augen fallende Symptom, an die Schmerzhaftigkeit und fasste alle Affektionen, die Schmerzen in der Halsgegend auslösten, unter dem Sammelbegriff 'Synanche, Angina' (συνάγχειν = zusammenpressen) zusammen.

Als man später, dank der Verbesserung der Untersuchungsmethoden und dank vor allem der Kontrolle der Leichenuntersuchung erkannte, dass es doch recht verschiedenartige Affektionen sind, die man so kurzerhand zusammengeworfen hatte, da ging man daran, die einzelnen Krankheitsbilder zu trennen und voneinander zu scheiden. Aber in dem Bestreben, den früheren Fehler gut zu machen, fiel man in das andere Extrem: man trennte und schied so gründlich, dass jede noch so geringe Verschiebung des Ausgangspunktes oder des Sitzes der Erkrankung, jede klinische und anatomische Begleiterscheinung, mochte sie noch so nebensächlich sein, als essentielles Unterscheidungsmerkmal angesehen wurde. Bald gab es ungezählte Arten und Unterarten von Halsaffektionen, und da jede von ihnen einen besonderen, möglichst klangvollen Namen erhielt, so war das Ende wieder eine heillose Verwirrung. Die kritiklose Scheidung hatte zu demselben Effekt geführt wie die kritiklose Zusammenfassung.[1]

Geschichtliches. — Erst als das vorige Jahrhundert, dank der Errungenschaften der pathologischen Anatomie und Bakteriologie, den Boden für ein besseres Verständnis pathologischer Vorgänge genügend vorbereitet hatte, war auch für unsere Disciplin die Möglichkeit zu einer Neuordnung dieses so verworrenen Kapitels gegeben. Und so habe ich im Jahre 1895, gestützt auf zahlreiche klinische, pathologische, histologische und bakteriologische Untersuchungen und Tierversuche in einer ein-

[1] Näheres hierüber siehe in meiner Monographie: *Larynxödem und submuköse Laryngitis*, Georg Reimer, Berlin, 1895.

gehenden Monographie zeigen können, dass eine Reihe von Halserkrankungen, die bis dahin als grundverschieden aufgefasst worden waren — das entzündliche Oedem, das Erysipel, die Phlegmone und der Abscess — *ätiologisch, pathologisch-anatomisch* und *klinisch* zusammengehören.

Die ganze Gruppe hatte ich damals zusammengefasst unter der Bezeichnung *Laryngitis*, bezw. *Pharyngitis submucosa* und sie je nach ihrer pathologischen Besonderheit geschieden in ein :

1. *Stadium oedematosum* (Erysipelas).
2. *Stadium plasticum*
3. *Stadium suppuratium* } akut-infektiöse Phlegmone und Abscesse.

Dreiviertel Jahre nach dem Erscheinen meiner Monographie hielt Felix Semon in der Royal Medical and Chirurgical Society in London einen Vortrag,[1] in dem er, gestützt auf 14 klinische Beobachtungen, für die von mir vertretene Auffassung eintrat und diese dahin erweiterte, dass die ' akut septischen Entzündungen des inneren und äusseren Halses, die bisher als akutes Kehlkopfoedem, oedematöse Laryngitis, Erysipelas des Pharynx und Larynx, Phlegmone des Pharynx und Larynx und Angina Ludovici beschrieben worden sind, pathologisch identisch seien '.

Diese Ansichten wurden, wie Semon später in seinen gesammelten Abhandlungen erzählt,[2] in der an seinen Vortrag sich anschliessenden Diskussion ' von der grossen Mehrzahl der Redner, teilweise mit einer in dieser hochakademischen Gesellschaft ganz ungewöhnlichen Heftigkeit bekämpft '. Hauptsächlich war es wohl der Ausdruck ' pathologische Identität ', an dem man Anstoss nahm.

Sieben bis acht Jahre später aber, im Jahre 1902, so berichtet Semon weiter, als Philip de Santi in derselben Gesellschaft, *mit ausgesprochener Anlehnung an die Semon'sche Arbeit*, einen Vortrag über dasselbe Thema hielt und in diesem genau dieselben Anschauungen zum Ausdruck brachte, da fanden die Diskussionsredner seine Ansichten fast ausnahmslos ganz selbstverständlich.

Dass aber Semons erste Anregung in der Zeit, die zwischen seinem eigenen und Philip de Santis Vortrag lag, auch in England doch nicht wirkungslos geblieben ist, dafür zeugt die im Jahre 1901 erschienene 3. Auflage des vortrefflichen Watson Williams'schen Lehrbuches, die sich die Semon'schen Ansichten in allen wesentlichen Punkten zu eigen gemacht hatte.

In Frankreich, wo sich schon in der vorlaryngoskopischen Zeit eine ganze Reihe hervorragender Aerzte (Bayle, Bouillaud, Brichetau, Cruveilhier, Sestier, Trousseau u. a.) in hervorragender Weise mit dieser Frage beschäftigt hatten, fand die moderne Auffassung ziemlich schnell Anerkennung und ging in alle besseren Lehrbücher über.[3] Ebenso in Italien und in den Vereinigten Staaten Amerikas.

[1] Felix Semon, *Forschungen und Erfahrungen* Bd. ii, p. 375, Berlin, 1912.
[2] pp. 421–2.
[3] Eine besonders klare und überzeugende Darstellung lese ich in dem Sammelwerk

In Deutschland war meine Auffassung der akut-infektiösen submukösen Halsentzündungen sehr schnell Allgemeingut geworden. In zahlreichen Aufsätzen und in fast allen Lehrbüchern (Bruck, Chiari, Gerber, Hajek, P. Heymann, und A. Meyer, Kronenberg, Edmund Meyer, Moritz Schmidt, Rupprecht, Schech u.s.w.) wurden sie glatt und fast ohne Vorbehalt akzeptiert.

Nicht ganz so rückhaltlos war man mit der von Semon gewählten Fassung einverstanden: man nahm Anstoss an der Einbeziehung der Angina Ludovici und an der Bezeichnung ' pathologische Identität '.

So standen die Dinge, als mir ca. 18 Jahre nach der Veröffentlichung meiner ersten Anregung von Seiten des Präsidiums der Rhino-Laryngologischen Sektion des XVII. Internationalen Medizinischen Kongresses in London der Wunsch ausgesprochen wurde, ich sollte noch einmal hier in einem offiziellen Referat zu diesem Thema Stellung nehmen. Dieser ehrenvollen Aufforderung habe ich gern Folge geleistet, denn seit dem Erscheinen meiner ersten Arbeit war meine Aufmerksamkeit unausgesetzt auf diese Fragen gerichtet, und neue eigene Beobachtungen, ebenso wie die inzwischen erfolgten Fortschritte auf bakteriologischem Gebiet haben mich überzeugt, dass der damals gelegte Grund sich in allen wesentlichen Punkten gut und tragkräftig bewährt hat, so dass wir auf den alten Fundamenten getrost weiter bauen können. So hat sich denn in logischer Weiterentwicklung meiner Ideen von 1895 nachfolgende Auffassung *der akut-infektiösen Halsentzündungen* allmählich bei mir herangebildet:

Alle *akut-entzündlichen Erkrankungen des Rachens, des Schlundes und Kehlkopfes* sind zurückzuführen entweder auf:

1. Mechanische, chemische, thermische Verletzungen, oder auf
2. Infektiöse Prozesse, d. h. auf die Einwirkung pathogener Mikroorganismen.

Diese *infektiösen* Halserkrankungen zerfallen wieder in verschiedene Unterabteilungen. Sie treten auf als Vorläufer oder Teilerscheinungen:

(*a*) Bei *konstitutionellen* Infektionskrankheiten, wie Lues, Tuberkulose u.s.w.

(*b*) Bei allgemeinen Infektionskrankheiten (Masern, Scharlach, Typhus, Pneumonie, Influenza u.s.w.) oder

(*c*) Als *selbständige* Erkrankungsformen der einzelnen in Rachen, Schlund oder Kehlkopf gelegenen Gebilde.

Hierher gehören alle die verschiedenen Halsaffektionen, die man je nach ihrem Sitz, ihren klinischen und pathologischen Erscheinungen oder auch nach ihrem Entdecker bezeichnet hat als Angina, Angina Ludovici, Angina Plaut-Vincenti, Tonsillitis (erythematosa, lacunaris, fossularis, follicularis, parenchymatosa, exulcerans benigna, oedematosa, erysipela-

von P. Brouardel und A. Gilbert (Paris, 1906), in dem von G. Roque und L. Galliard bearbeiteten Bd. XV (*Malad. de la bouche, du pharynx*, etc.).

tosa, phlegmonosa, abscedens), Peritonsillitis, Pharyngitis et Laryngitis acuta, erysipelatosa, Laryngitis submucosa acuta, akut-entzündliches Larynxoedem etc. und die pseudomembranöse Beläge bildenden Affektionen, die genuine Diphtherie und die verschiedenen Arten der pseudodiphtherischen Erkrankungen.

Alle die unter diesen und ähnlichen Namen laufenden, früher als essentiell verschiedenartig angesehenen Erkrankungen sind zusammenzufassen unter der Bezeichnung ' Akut-infektiöse Entzündung der Halsorgane ' und gehören ätiologisch, pathologisch und klinisch eng zusammen.[1]

Unter den, diese Erkrankung hervorrufenden Mikroorganismen sind einige wenige spezifische, d. h. solche, deren Einwirkung jedesmal dieselben typischen Krankheitserscheinungen auslöst : der Loeffler'sche Bacillus, der die genuine Diphtherie, und die Spirochaete, welche die Plaut-Vincenti'sche Angina hervorruft. Bei all den anderen Halsaffektionen, die eben aufgezählt wurden — und nur diese sind Gegenstand unserer Betrachtungen — hat sich bisher eine Spezifität nicht nachweisen lassen. Wohl wissen wir, dass diesem oder jenem Infektionskeim in der Aetiologie dieser oder jener Erkrankungsform eine besonders wichtige Rolle zukommt, aber eine wirkliche Spezifität in dem Sinne, dass dieser Mikroorganismus immer dieselbe Affektion auslöst, und dass diese Affektion von keinem anderen Mikroorganismus ausgelöst werden kann, konnte bisher nicht erwiesen werden.

In der Mund-Rachenhöhle eines jeden gesunden Menschen finden sich zahlreiche Mikroorganismen verschiedenster Art. Bei ganz normalen Halsorganen hat man hier nachweisen können : Streptokokken, Staphylokokken, Diplokokken, Diphtherie-Bacillen, Pneumokokken, Meningokokken, das Bacterium Coli, Schimmelpilze u.s.w. Meist finden sich zwei, drei, oder noch mehr verschiedene Arten nebeneinander. Diese Keime können avirulent sein, sie sind aber, wie wiederholentlich gezeigt wurde, oft genug auch hochvirulent. Wir stehen also vor der auffallenden Tatsache, dass Keime, die bei ihrem Träger auch nicht die geringsten Krankheitserscheinungen auslösten, bei der Ueberimpfung auf Tiere schwere und schwerste Erkrankungen hervorrufen. Zur Erklärung für dieses überraschende Verhalten nimmt man an, dass in diesen Fällen die

[1] Es besteht zwischen der deutschen und englischen Terminologie eine Differenz, die leicht zu Missverständnissen führen kann. In England bezeichnet man Prozesse, die in Deutschland ' akut-infektiös ' genannt werden, als ' acute septic ' und weist damit auf die mit den akut-infektiösen Prozessen gewöhnlich verbundenen Allgemeinerscheinungen hin. Wir dagegen verbinden mit der Bezeichnung ' septisch ' einen ganz besonderen Begriff. Wir scheiden die akut-infektiösen Prozesse in solche, die vorwiegend lokale Veränderungen auslösen und in solche, bei denen neben den örtlichen mehr oder weniger schwere Allgemeinerscheinungen auftreten, die unter Umständen das ganze Krankheitsbild beherrschen. Aber durchaus nicht jede, sondern nur eine ganz bestimmte, später noch näher zu charakterisierende Form dieser Allgemeinerkrankung wird bei uns ' septisch ' genannt. Das englische ' septic ' deckt sich also ungefähr mit dem deutschen Begriff ' infektiös ', ist aber wesentlich weiter gefasst, als unser ' septisch '.

pathogenen Kräfte der Mikroorganismen, die Toxine und Endotoxine (Aggressine — Kruse, Bail) von den natürlichen bakterienfeindlichen Widerstandskräften des Organismus (Alexine — Buchner) in Schach gehalten werden.

Unter gewissen Umständen aber, infolge von mancherlei Gelegenheitsursachen, reicht die Kraft der natürlichen Abwehrmittel nicht mehr aus, um die Giftwirkung der Mikroorganismen zu parallelisieren. Mechanische, chemische, thermische Verletzungen zerstören die schützende Decke und ermöglichen den Infektionskeimen, die bis dahin harmlos in der Mund- und Rachenhöhle lagerten, das Eindringen in die Tiefe der Gewebe, wo sie dann ihre verderbliche Wirkung entfalten. Oder aber es haben Unterernährung, Blutverluste, interkurrierende Erkrankungen und ähnliche Zwischenfälle die *allgemeine* Widerstandskraft des Organismus, Erkältungen auf reflektorischem Wege die *lokale* Widerstandskraft der Halsorgane so weit geschwächt, dass die Gewebe nun nicht mehr imstande sind, die Lebensenergie der bereits an Ort und Stelle lagernden oder frisch eindringenden Infektionskeime zu parallelisieren. Nun erst ist der Weg in die nicht mehr genügend verteidigten Organe frei, und durch die Lymph- und Gewebsspalten, die sogenannten ‘ physiologischen Wunden ’, dringen die pathogenen Keime in das Innere der Gewebe und entwickeln dort ihre ‘ Virulenz ’, d. h. die Gesamtheit ihrer organfeindlichen Kräfte.

Die Folgen dieser Schädigungen können sein : 1. lokaler, 2. regionärer, 3. allgemeiner Art.

I. Lokale Schädigungen

Das Eindringen der pathogenen Mikroorganismen in die Gewebe selbst ist die unerlässliche Vorbedingung für die Entfaltung ihrer Giftwirkung. Hier entwickeln und vermehren sie sich manchmal so stark, dass sie wie Fremdkörper schon durch ihre Masse die Gewebe rein mechanisch schädigen, wie beim Erysipel und bei der tropischen Malaria, oder sie wirken, was weit verhängnisvoller ist, durch die von ihnen produzierten Stoffe. Das sind entweder Stoffwechselprodukte des lebenden Mikroorganismus, Toxine genannt, z. B. beim Tetanus, oder durch ihre Endotoxine, das sind Giftstoffe, die erst beim Zerfall der Bakterien frei werden, z. B. beim Typhusbacillus und bei den Choleravibrionen.

Nicht jeder Infektionskeim kann an jedem beliebigen Ort in den Organismus eindringen, aber die Schleimhaut der oberen Luftwege ist so reich an ‘ physiologischen Wunden ’, dass jede Abart der ganzen Fauna hier eine passende Einfallspforte findet. Diese Vulnerabilität der Halsschleimhaut ist gewiss mit Schuld daran, dass ausser den typischen Infektionskeimen noch so viele andere zufällige Schmarotzer der Mund- und Rachenhöhle in den Halsorganen ihre Wirksamkeit entfalten und von hier aus tiefer in den Organismus eindringen.

Nicht jeder Infektionserreger ruft gleich an der Eintrittspforte

nachweisbare pathologische Veränderungen hervor ; der Tuberkelbacillus z. B. kann die Schleimhaut des Rachens und der Tonsille, ja selbst weite Strecken des Organparenchyms und der Lymphwege durchwandern, ohne erkennbare Krankheitserscheinungen hervorzurufen. Aber das ist nur eine Ausnahme, die meisten Infektionsträger rufen bereits an der Stelle, wo sie in das Gewebe eintreten, eine mehr oder weniger energische Reaktion hervor, gewöhnlich nicht sofort, meist erst nach einer gewissen Frist, in der sie durch die immer lebhafter werdende Produktion von Giftstoffen den Widerstand der natürlichen bakteriziden Schutzkräfte überwinden (Inkubationszeit).

Die erste Reaktion auf den Angriff körperfeindlicher Bakterien, welcher Art sie auch sein mögen, ist immer und überall, in allen Halsorganen dieselbe, ist im vollen Sinne des Semon'schen Wortes ' identisch ' : immer sind es die typischen klassischen Symptome der Entzündung — Erweiterung der Gefässe, vorübergehende Beschleunigung und darauf folgende Verlangsamung des Blutstromes, Randstellung der weissen Blutkörperchen, Austritt seröser Flüssigkeit und zelliger Elemente aus den Gefässen, Wucherungsvorgänge an den Gewebszellen — durch die sich die angegriffenen Organe dieser Invasion zu erwehren suchen.

In diesem Anfangsstadium ist die Erkrankung klinisch schwer zu erkennen, wenn sie sich in der Tiefe der Gewebe entwickelt, aber wohlbekannt und gut charakterisiert, wenn sie sich über die Oberfläche hin ausbreitet : es ist das *Schleimhaut-Erythem* mit seiner Rötung, Schwellung und Auflockerung der obersten Zellschichten, das zuweilen als selbständiges Krankheitsbild, viel häufiger aber noch als Anfangsstadium tiefer greifender Prozesse auftritt.

Aus dieser einheitlichen, identischen Primärreaktion entwickeln sich bei weiterem Fortschreiten des Krankheitsprozesses recht verschiedenartige pathologisch-anatomische Bilder, indem bald dieses, bald jenes der ursprünglichen Symptome so in den Vordergrund tritt, dass die übrigen fast ganz dagegen verschwinden. So entstehen aus der Primärreaktion, wie aus einer gemeinsamen Wurzel, die verschiedenartigsten Formen der Entzündung, von denen wir hier nur die uns interessierenden Varietäten anführen wollen :

1. *Die hämorrhagische Entzündung.* — Wenn die Blutgefässe durch die Noxe besonders stark in Mitleidenschaft gezogen werden, wenn sie durch Tromben oder Embolien verlegt werden oder schon vorher erkrankt sind (Arteriosklerose), dann treten oft neben den Leukocyten grössere Mengen von roten Blutkörperchen aus der Blutbahn aus. Die hierdurch bedingte Rotfärbung der Entzündungsprodukte gibt dem Krankheitsbild ein besonderes Gepräge.

2. *Die katarrhalische Entzündung.* — Wenn die Entzündung sich in den obersten Schleimhautschichten abspielt, so wird die Schleimhautproduktion alteriert : die Absonderung ist meist vermehrt, die chemische Zusammensetzung des Sekretes verändert. Zahlreiche verschleimte Deckepithelien, Leukocyten und Lymphocyten sind ihm beigemengt.

Je nach seiner chemischen Beschaffenheit und nach seinem Gehalt an zelligen Elementen bezeichnet man diese Form der Oberflächenentzündung als serösen, als schleimigen oder als schleimig-eitrigen Katarrh.

3. *Die pseudomembranöse (fibrinöse, croupöse) Entzündung.* — Bei sehr vielen Entzündungsvorgängen, die sich in oder dicht unter der Schleimhautoberfläche der Halsorgane abspielen, zeigt das Exsudat eine Neigung zur Gerinnung, ganz besonders wenn das Deckepithel an einzelnen Stellen zugrunde gegangen ist. Dann tritt die Ausschwitzungsflüssigkeit durch diese Wunden an die Oberfläche und unter dem Einfluss des nekrotisierenden Gewebes bilden sich Fibrinmassen, deren Form und Ausdehnung von der Heftigkeit des Entzündungsprozesses und von dem Umfang der Nekrotisierung abhängt.

Bei leichten Verletzungen, bei denen nur die obersten Deckzellen in geringen Ausdehnung geschädigt sind, ist die Wundfläche nur mit einem zarten, leicht abziehbaren Fibringerinnsel bedeckt (fibrinöse oder croupöse Entzündung). Ist der Substanzdefekt grösser, so dass die ganze Epitheldecke, die subepitheliale Schicht und womöglich noch das tiefer liegenden Bindegewebe in weiterem Umfange nekrotisch geworden sind, dann bildet die Pseudomembran ein netzartiges Gefüge aus derberen und feineren Fibrinfasern und hyalinen Balken und Schollen, denen sich Leukocyten, rote Blutkörperchen und nekrotische Massen eingelagert finden. Diese Massen haften der Unterlage fest an, so dass sie oft nur mit Gewalt abgezogen werden können. Diese Form der Entzündung hat man, je nach dem Umfang und der Schwere der Erscheinungen, als diphtheroide (pseudodiphtherische) oder als diphtherische bezeichnet. Da diese Namen vielfach zu Missverständnissen geführt haben, hat Aschoff vorgeschlagen, sie als ‘ verschorfende ’ zu bezeichnen.

4. *Die serös-exsudative Entzündung.* — Der Austritt *seröser* Blutbestandteile überwiegt alle anderen Entzündungserscheinungen. Die Beimischung zelliger Elemente, die Wucherungsvorgänge an den fixen Gewebselementen treten so zurück, dass die Flüssigkeitsansammlung im Gewebe die Szene beherrscht. Erfolgt diese in eine vorgebildete Höhle, so spricht man von ‘ entzündlichen Hydrops ’, erfolgt sie diffus in das Gewebe hinein, so bildet sich ein ‘ entzündliches Oedem ’. Verbreitet sich das Oedem dicht unter der Oberfläche, so wird die Schleimhaut an den Stellen, wo sie locker an ihre Unterlage angeheftet ist, in glasig durchscheinenden, hellgrauen oder goldgelben Blasen abgehoben. Entwickelt sich das Oedem mehr in der Tiefe, so quellen die einzelnen Gewebselemente auf und das ganze Organ erscheint gequollen und vergrössert, die Oberflächenschleimhaut glasig durchscheinend.

5. *Die zellig-exsudative oder infiltrierende Entzündung.* — Diese Form der Entzündung steht in einem ausgesprochenen Gegensatz zu der serös-exsudativen Entzündung. Ueberwog dort die *Flüssigkeitsausscheidung*, so beherrscht hier die Durchsetzung des erkrankten Gewebes mit *körperlichen* Elementen die Szene.

Leukocyten, Lymphocyten, histogene Wanderzellen, von der positiv

chemotaktischen Kraft der Infektionserreger angezogen, erfüllen alle Maschen des Entzündungsgebietes, das sich je nach der Masse des Infiltrationsmaterials derb oder bretthart anfühlt. Unter dem fermentativen Einfluss der Infektionsgifte (Lysin, Leukocidin — van de Velde), kommt es zu einer Erweichung des Infiltrationsmaterials. Die harte Masse wird teigig und allmählich von einer rahmigen undurchsichtigen Flüssigkeit durchsetzt. Diese breitet sich in den Gewebsspalten aus und bewirkt durch ihre eigene fermentative Kraft selbst wieder eine Einschmelzung und Nekrotisierung der Intercellularsubstanz und der fixen Gewebszellen (Eiterbildung).

Ist diese Eiterbildung eine circumscripte, so dass sich der Eiter nicht diffus im Gewebe, sondern in einer abgeschlossenen Höhle bildet, so liegt ein Abscess vor.

6. Bei der *parenchymatösen* oder *alterativ-degenerativen* Form der Entzündung überwiegen die alterativ-degenerativen Veränderungen der Gewebszellen den exsudativen Vorgängen gegenüber (Lubarsch).

Dieses sind die verschiedenen Formen der entzündlichen Reaktion, die in den Halsorganen durch den Angriff pathogener, akut wirkender Infektionskeime hervorgerufen werden. Aus einer einheitlichen Primärreaktion, wie aus einer gemeinsamen Wurzel hervorgegangen und dann nach verschiedenen Richtungen sich entwickelnd, bildet jede von ihnen das pathologisch-anatomische Substrat für eines oder auch für mehrere der bisher streng geschiedenen Krankheitsbilder.

Den ursprünglichen Typus der Entzündung sehen wir beim Erythem: seröse, schleimig-seröse, schleimige und schleimig-eitrige Katarrhe, die diphtherischen und pseudodiphtherischen Erkrankungen im Schlund, im Rachen und Kehlkopf, ebenso die hämorrhagische Laryngitis und Pharyngitis sind bekannt; es decken sich diese Krankheitsbilder mit den entsprechenden Entzündungsformen. Die serös-exsudative Form der Entzündung sehen wir beim typischen Erysipel, die verschiedenen Typen der phlegmonösen Entzündung bei der Angina Ludovici, bei der eitrigen Phlegmone und beim Abscess des Rachens und Kehlkopfes. Die alterativ-degenerativen Veränderungen des Parenchyms sind bei den akut-infektiösen Halserkrankungen kaum je so heftig, dass sie dem ganzen Krankheitsbild ihren Stempel aufdrücken. Oefters aber bilden sie die Grundlage gewisser, besonders auffallender Erscheinungen in der Oberflächenschicht: sie sind es, die die häufigen Trübungen und Fleckenbildungen im Epithel, die Desquamation des Epithels beim Katarrh und bei der Angina lacunaris hervorrufen oder wenigstens vorbereiten helfen.

II. Die regionäre Erkrankung

Bei all diesen Krankheitsprozessen pflegen die Entzündungsvorgänge nicht auf den ursprünglichen Angriffspunkt des Infektionskeimes beschränkt zu bleiben. Meist ziehen sie auch die Umgebung in Mitleidenschaft, indem sie entweder von Punkt zu Punkt *kontinuierlich* fortkriechen oder

indem Infektionsmaterial auf dem Lymphwege bis zur nächsten oder übernächsten Drüsenstation verschleppt wird und hier neue Krankheitserscheinungen hervorruft.

Sehr wesentlich für unsere Betrachtung ist die Tatsache, dass die *einzelnen Erkrankungsformen bei ihrem kontinuierlichen Fortschreiten ihren ursprünglichen pathologisch-anatomischen Charakter durchaus nicht immer festhalten.* Im Gegenteil, häufig sehen wir, dass eine Erkrankung, die ursprünglich als erythematöser oder katarrhalischer Prozess einsetzte, in der Umgebung des primären Herdes Phlegmonen und Abscesse hervorruft — und dass umgekehrt die primäre Phlegmone beim Uebergreifen auf die Nachbarorgane Oedem, also serös-exsudative Entzündungsvorgänge veranlasst. Dieser unmittelbare Uebergang des einen Entzündungstypus in den anderen, beweist unwiderleglich die Einheitlichkeit dieser pathologisch-anatomischen Veränderungen, zwischen denen *eine essentielle Verschiedenheit* nicht besteht und nicht bestehen kann.

III. Allgemeinerscheinungen

Neben der generellen Uebereinstimmung der *lokalen* Vorgänge, die durch die Entwicklung aus einer gemeinsamen Wurzel, und der generellen Uebereinstimmung der *regionären* Vorgänge, die durch das Uebergehen des einen Entzündungstypus in den anderen dargetan wird, gibt es noch ein drittes Moment, welches die Zusammengehörigkeit all der verschiedenen Formen der akut-infektiösen Halsentzündungen verbürgt, und das ist die *Gleichartigkeit der von ihnen ausgelösten Allgemeinerscheinungen.* Nicht etwa, dass diese nun immer ein und dieselbe Form annehmen, im Gegenteil, die Formen, in denen die konstitutionelle Erkrankung ihren Ausdruck findet, sind recht mannigfach und wechselnd. Aber ihre Zusammengehörigkeit, die prinzipielle Einheitlichkeit in der Vielheit der Erscheinungen ist trotzdem zweifellos; sie wird bewiesen durch zwei Momente: erstens durch die Gleichartigkeit des Entstehungsmodus; immer sind es dieselben Infektionskeime oder deren Giftstoffe, die erst den Primärherd und dann von hier aus nach ihrer Verschleppung durch den ganzen Organismus die Allgemeinerscheinungen auslösen; und zweitens durch die Tatsache, dass *jede akut-infectiöse Halsaffektion, in welcher Gestalt sie auch auftreten mag, diese oder jene oder auch das ganze Heer der konstitutionellen Begleit- und Folgeerscheinungen auslösen kann.*

Als solche konstitutionelle Begleit- oder Folgeerscheinungen kommen in Betracht:

1. *Das Fieber.* — Das Fieber, der Ausdruck einer Störung in der Wärmeregulierung des Körpers, fehlt fast nie bei akuten Infektionskrankheiten. Es beruht auf einer Schädigung des Wärmecentrums durch die im Blute kreisenden Gifte der Infektionserreger. Die hierdurch gesteigerte Wärmeproduktion kann durch die Wärmeabgabe des Körpers nicht genügend ausgeglichen werden.

2. *Die entzündliche Leukocytose.* — Im Verlauf der allermeisten In-

fektionskrankheiten — eine Ausnahme bilden nur Typhus, Masern, Malaria und nicht lokalisierte Sepsis — beobachtet man eine auffallende Vermehrung der Leukocyten im Blut (Hyperleukocytose). Diese wird durch chemotaktische Einflüsse des Infektionsvirus hervorgerufen, indem die in den blutbildenden Organen befindlichen Leukocyten in die Blutbahn gelockt werden (positive Chemotaxis). Ihr gegenüber steht bei den erwähnten Erkrankungen die Hypoleukocytose, bei der die im Blut kreisenden Leukocytosenmengen, von dem Infektionsgift abgestossen, vermindert sind (negative Chemotaxis).

3. *Anämie* und 4. *Milztumor*, die sich mit sehr vielen Infektionskrankheiten vergesellschaften, sind zurückzuführen auf die von vielen Infektionserregern erzeugten hämolytischen Gifte (Hämolysine), die eine starke Zerstörung der roten Blutkörperchen bewirken. Ihr Zerfall bedeutet eine entsprechende Herabsetzung des Hämoglobingehaltes (Anämie). Die Aufspeicherung ihrer Zerfallsprodukte in der Milz und die damit Hand in Hand gehende vermehrte Neubildung von Erythrocyten in diesem Organ gibt sich als Milztumor zu erkennen.

5. *Nierenschädigung* und 6. *Nervenschädigung*. — Sehr häufig werden durch die im Blutkreislauf zirkulierenden Giftstoffe Schädigungen der Nieren- und Nervensubstanz ausgelöst. Diese treten gewöhnlich in der Form einer parenchymatösen Degeneration auf (auf die durch Verschleppung von Bakterien in der Nierensubstanz entstehenden metastatischen Herde kommen wir noch bei Besprechung der Pyämie zurück), die bei Lokalisation in der Niere zu Albuminurie und Hämaturie, bei Lokalisation im Gehirn, im Rückenmark und in den peripheren Nerven zu Krampf- (Tetanus — Lyssa) oder zu Lähmungserscheinungen führt. Diese auch sonst recht oft bei akuten Infektionskrankheiten auftretenden Schädigungen der Nieren und des Nervensystems sind bei den akut-infektiösen Erkrankungen der Halsorgane besonders häufig und besonders gefürchtet. Die Geringfügigkeit der primären Halsaffektion steht oft in schreiendem Missverhältnis zu den schweren Störungen, die durch sie in den Nieren und im Nervenapparat, besonders in dem nervösen Apparat des Herzens ausgelöst werden. So mag mancher unaufgeklärte Herztod auf eine vorhergegangene, gar nicht beachtete Infektion der Halsorgane zurückzuführen sein: ähnlich wie man auch oft bei chronischen Kinder-Nephritiden beobachtet, dass übersehene Anginen erst auf dem Umwege über die Urinuntersuchung durch die Vermehrung des Eiweiss- und Blutgehaltes entdeckt werden.

7. *Bakteriämie*, 8. *Pyämie*, 9. *Septikämie*. — Wenn die Bakterien in die Blutbahnen gelangen, sei es durch Einbruch eines Krankheitsherdes in die Gefässbahn oder von Tromben oder Embolien aus oder auch durch Vermittlung der Lymphbahnen, so spricht man von einer *Bakteriämie*. Dieser Zustand braucht sich in keiner Weise durch Krankheitserscheinungen zu erkennen zu geben: es können zahlreiche Mikroorganismen in der Blutbahn kreisen, ohne dass der Organismus mit einer für uns irgendwie erkennbaren Reaktion darauf antwortet. Erst wenn sich die Infek-

tionsträger innerhalb des Gefässsystems selbst vermehren und entwickeln oder von hier aus neue Krankheitsherde etablieren, kommt es zu den schweren Allgemeinerscheinungen, die wir als *Pyämie* und *Septikämie* bezeichnen. Diese beiden Krankheitsbilder lassen sich ganz gut in der Theorie, in der Praxis aber oft nur sehr schwer, manchmal auch gar nicht auseinanderhalten.

Von *Pyämie* sprechen wir, wenn Infektionskeime vom primären Herd aus durch die Blut- oder Lymphbahn in entfernte Körperregionen verschleppt werden und überall da, wo sie günstige Entwicklungsbedingungen antreffen, neue Krankheitsherde entstehen lassen. Auf diese Weise bilden sich Metastasen in den Gelenken, in den Nieren, im Knochen, in den Lungen, im Herzmuskel u.s.w. Jeder dieser neu auftretenden Entzündungs- und Eiterherde ruft neben den lokalen Schädigungen eine frische Intoxikation durch die sich immer wieder neubildenden Giftstoffe hervor, die durch Schüttelfröste, Temperatursteigerungen u.s.w. den Verfall des Organismus beschleunigen.

Die *Septikämie* unterscheidet sich im Prinzip von der Pyämie dadurch, dass sich bei ihr die Infektionsträger vom primären Herd aus durch das ganze Gefässsystem verbreiten, sich in ihm, *das kreisende Blut gleichsam als Kulturmedium benutzend*, weiter entwickeln und, besonders in den Capillaren, enorm vermehren. Hier sind es vor allem die Bakterien*gifte*, welche die schweren Allgemeinerscheinungen, Fieber, Coyma, Lähmungen u.s.w. hervorrufen.

Diese theoretische Scheidung, nach der die *Pyämie* hauptsächlich durch Metastasenbildung, die *Septikämie* durch die rein toxische Allgemeinerkrankung charakterisiert wird, ist, wie gesagt, in der Praxis oft nur schwer durchzuführen. Denn auch bei der Pyämie können die toxischen Erscheinungen stark in den Vordergrund treten, wenn die Nachschübe von Eiterdepots ins Blut sehr stark sind, und bei der Septikämie wieder sind metastatische Herde durchaus nicht ausgeschlossen.

Im Rahmen dieser verschiedenartigen Krankheitstypen spielen sich nun all die zahlreichen Formen von akuten Halsaffektionen ab, in ihrer bunten Vielgestaltigkeit doch eine bestimmte Einheitlichkeit bewahrend: immer handelt es sich *ätiologisch* um akut-infektiöse Prozesse, *pathologisch* um wohl charakterisierte Entzündungsvorgänge in den lokalen und regionären Krankheitsherden, und das unübersehbare, anscheinend jeder Ordnung spottende Heer der Komplikationen[1] rangiert sich lückenlos

[1] Einen Fingerzeig für ein besseres Verständnis der in so mannigfachen Formen auftretenden Generalisierung dieser Erkrankungen geben vielleicht die später noch zu erwähnenden Versuche von Abrami und Richet fils. Diese Autoren spritzten Kaninchen eine Streptokokken-Aufschwemmung in eine Körpervene und bewirkten gleichzeitig durch mechanische Belastung oder durch einen Tropfen Crotonöl eine geringfügige Störung in der Zirkulation des Ohres. Bei jedem dieser Versuche stellte sich prompt an der geschädigten Stelle eine erysipelatöse Entzündung ein: es waren also immer die Infektionserreger auf hämotogenem Wege zu *allererst* dorthin verschleppt worden, wo *ein Locus minoris resistentiae geschaffen worden war*. Dieser

in die Reihe der konstitutionellen Symptome, wie sie uns als Begleit- und Folgeerscheinungen anderer akuter Infektionskrankheiten längst vertraut sind. So lösen sich in dieser Einheitlichkeit restlos all die ungezählten Halsaffektionen, von denen man jeder einen besonderen Namen zuzubilligen für notwendig hielt.

Die einzelnen Krankheitsbilder und ihre Therapie

Auf eine eingehende Erörterung der einzelnen Krankheitsbilder, die ja hinlänglich bekannt sind, darf ich verzichten. Verschiedene eigene Beobachtungen und die zahlreichen einschlägigen Krankheitsfälle der chirurgischen Abteilung des Städtischen Krankenhauses am Urban zu Berlin, die mir Herr Direktor Koerte gütigst zur Verfügung stellte — wofür ich ihm auch an dieser Stelle gern meinen Dank ausspreche — haben neue Gesichtspunkte nicht ergeben. Ich begnüge mich mit einigen generellen Bemerkungen.

Die *erythemartigen* Erkrankungen (Pharyngitis, Tonsillitis, Uvulitis, Laryngitis simplex etc.) sind unkomplizierte Entzündungen der Oberfläche, bei denen die vier Kardinalsymptome der Entzündung ungefähr gleichmässig entwickelt sind.

Beim *Katarrh* tritt hierzu eine lebhaftere Betätigung der schleimbildenden Organe und eine stärkere Betonung der alterativ-degenerativen Vorgänge, die zu einer Abstossung der obersten Epithelschichten (Rachen-Kehlkopfkatarrh) führt. Die katarrhalischen Prozesse pflegen meist aber auch noch etwas tiefer zu greifen und im submukösen Gewebe und im Parenchym exsudative Vorgänge auszulösen, die eine Volumenzunahme des erkrankten Organes veranlassen. So ist beim Rachen- und Kehlkopfkatarrh oft die ganze Schleimhaut leicht verdickt, der Umriss der Epiglottis, der Aryknorpel, der ary-epiglottischen Falten weniger scharf als unter normalen Verhältnissen. Auf derselben Basis beruht die Vergrösserung der Tonsillen bei katarrhalischen Erkrankungen. Die gelben Punkte und Flecken in den Lacunen verdanken ihr Entstehen den Sekretmassen, die aus der Tiefe der Crypten herausquellen und durch die Beimischung grösserer oder geringerer Mengen von Leuko- und Lymphocyten, von degenerierten und abgestossenen Epithelien, von Spalt- und Schimmelpilzen ihre jeweilige Farbe und Konsistenz erhalten. Die fibrinösen Ausscheidungen, die das in ihr Netzwerk eingelagerte Sekret festhalten, bilden sich zumeist bei Oberflächenläsionen.

Bei der *Pharyngitis* und *Laryngitis haemorrhagica* tritt die Alteration der Gefässwände besonders in den Vordergrund.

Die echte Diphtherie und all die anderen mit pseudomembranösen Auflagerungen einhergehenden Erkrankungen dokumentieren sich als

Versuch, dessen Ergebnisse ich aus eigener Erfahrung bestätigen kann, könnte zum Ausgangspunkt weiterer Untersuchungen dienen, ob vielleicht auch sonst beim Einbruch von Infektionsherden in den Kreislauf diejenigen Organe zunächst geschädigt werden, deren Widerstandsfähigkeit aus irgendeinem Grunde herabgesetzt ist.

Entzündungsvorgänge, bei denen die Nekrotisierung der Oberfläche und die Ausscheidung fibrinogener Substanzen in den Vordergrund tritt.

Bläschenbildung entsteht durch circumscripte Oberflächenentzündung bei lebhafter Exsudation seröser oder eitriger Massen.

Erosions- und Geschwürbildung tritt ein, wenn die Entzündungserscheinungen einen mehr alterativ-degenerativen Charakter annehmen, der schliesslich zu oberflächlichen oder tiefer greifenden Substanzverlusten führt.

Das *Erysipel* zeigt in seiner charakteristischen Form den Typus der rein serösen, das *harte Infiltrat*, die *Phlegmone* und der *Abscess* den Typus der infiltrierenden und erweichenden Entzündung.[1]

Dass zwischen diesen Grundtypen zahlreiche Uebergänge und Kombinationen vorkommen, dass die Heftigkeit der lokalen und regionären Erscheinungen, die Tendenz zur Generalisierung von den verschiedensten Umständen — Menge und Virulenz des Infektionsstoffes, allgemeine und lokale Widerstandsfähigkeit des Patienten, Lokalisation des primären Herdes, u.s.w. abhängig sind, bedarf ja keiner weiteren Erörterung.

Auch die Therapie hat in der Zwischenzeit nennenswerte Veränderungen nicht erfahren, bis auf die Serotherapie, über deren Wirksamkeit eine weitere Klärung recht erwünscht wäre. Der augenblickliche Stand der Dinge ist ungefähr folgender:

Bei Staphylo- und Pneumokokken-Affektionen hat man durch Serumbehandlung bisher nirgends einen Erfolg erzielt.

Ueber die Wirkung des Streptokokken-Serums bei Streptokokken-Erkrankungen sind die Meinungen geteilt. Zahlreiche Autoren melden, dass sie bei wiederholentlichen Versuchen niemals einen Erfolg zu verzeichnen hatten. Andere, darunter Semon und Ph. de Santi, haben durch Serum-Injektionen in manchen Fällen einen ganz überraschenden Umschwung erzielt. Einen derartig verblüffenden Erfolg habe ich auch erlebt.

Am 7. Mai 1910, Vormittags, wurde ich zu einem 17 jährigen Patienten gerufen, der seit ca. 8 Tagen an einer sehr schweren Phlegmone des Rachens und Kehlkopfes erkrankt war. Die Tonsillen und die Pharynxschleimhaut waren mit übelriechenden, schmutziggelben Belägen bedeckt; die vielfach ulcerierte Schleimhaut des ganzen Pharynx und Larynx war in eine derbe, rötliche, graue Infiltrationsmasse verwandelt. Temperatur 39,8–40,1°; Puls 120–130; Urin enthält Blutkörperchen, Albumen, Cylinder; Bewusstsein benommen.

Das ganze Krankheitsbild war ein so schweres, dass mir der Patient, sonst ein überaus kräftiger, ganz gesunder Bursche, verloren

[1] Der Meinung Rupprechts, dass Semon nicht berechtigt war, die Angina Ludovici in den Kreis der akut-infektiösen Halserkrankungen mit einzubeziehen, kann ich nicht zustimmen. Wenn auch die Einfallspforte hier gewöhnlich eine andere ist, als bei den übrigen akut-infektiösen Halsentzündungen, so gehört dieses Krankheitsbild doch durch seine Aetiologie, durch seine pathologisch-anatomischen Veränderungen und seinen klinischen Verlauf zweifellos hierher.

schien. An demselben Tage noch wurde eine Serum-Injektion (polyvalentes Streptokokkenserum) gemacht, bei der aber nur eine halbe Dosis verabreicht wurde. Am nächsten Morgen war die Temperatur auf 39° gesunken, gegen 39,8° Tags vorher. Abends stieg die Temperatur nur bis 39,2 gegen 40,1° Tags zuvor. Jetzt nochmals Injektion einer vollen Dosis; am nächsten Tage war Temperatur 38,4°, Abends 38,5°, Puls 110; am 2. Tage Temperatur 37,6–37,8°; am 3. Tage 36,9–37,5°; vom 4. Tage an Temperatur dauernd normal. Der lokale Befund, Puls und Urin zeigten vom Tage der 1. Injektion an eine geradezu verblüffende Besserung. Vollkommene Heilung.

Ein derartiger Fall würde natürlich wenig oder gar nichts besagen. Da solche Fälle aber doch wiederholentlich beobachtet worden sind — auch Herr Kollege Ed. Meyer, Berlin, war so freundlich, mir einige ähnliche Erfahrungen zur Verfügung zu stellen — so scheint mir das zu beweisen, dass doch unter gewissen Bedingungen das Streptokokken-Serum auf die Streptokokken-Infektion von entscheidendem Einfluss ist. Leider wissen wir noch gar zu wenig darüber, welcher Art diese Bedingungen sind. Man nimmt an, und wohl nicht mit Unrecht, dass der Erfolg der Serum-Injektion davon abhängt, ob die für die Serumbeschaffung verwandte Kultur dem Infektionsstamm nahe stand.

Können wir auch die Zusammenhänge, auf die es hier ankommt, noch nicht klar überblicken, so halte ich doch diesen Weg für aussichtsreich, und ich würde es bei so schweren Fällen immer für angezeigt halten, die Injektion eines polyvalenten oder autogenen Serums zu versuchen.

Bakteriologie

Nachdem die Zusammengehörigkeit der akut-infektiösen Halsentzündungen aus der generellen Einheitlichkeit ihrer pathologisch-anatomischen und ihrer klinischen Erscheinungen erwiesen worden ist, gilt es noch, die *ätiologischen* Bedingungen der einzelnen Krankheitsformen zu untersuchen — möglich, dass sich hier ein Fingerzeig für die aus einer so einheitlichen Wurzel sich entwickelnde Mannigfaltigkeit der Krankheitsbilder ergibt.

Zwei Fragen sind es hier, von deren Beantwortung vor allem die fernere Gestaltung der Lehre von den akut-infektiösen Erkrankungen der Halsorgane abhängt:

1. Wann und wo sind es *spezifisch* wirksame Keime, die für eine bestimmte Erkrankungsform verantwortlich zu machen sind, und

2. Lassen sich bei Erkrankungen, die nicht durch spezifische, sondern durch andere Infektionserreger hervorgerufen werden, die Bedingungen feststellen, die das eine Mal zu der einen, das andere Mal zu einer anderen Erkrankungsform führen?

Die Beantwortung dieser Fragen, die auch an anderen Körperregionen nicht leicht ist, ist bei den Halsorganen mit ganz besonderen Schwierigkeiten verknüpft. Durch den dauernden, unmittelbaren Kontakt mit der

atmosphärischen Luft, durch die Nahrungsaufnahme, durch die im Munde zurückbleibenden Speisereste, durch gelegentliche Zahnerkrankungen, durch die crypten- und buchtenreiche Gestalt des lymphatischen Rachenringes wird die Mund-Rachenhöhle zu einem Sammelplatz zahlloser Mikroben. Unter diesen finden sich, wie die Untersuchungen der verschiedensten Autoren ergeben haben, selbst unter ganz normalen Verhältnissen neben harmlosen Saprophyten auch hochvirulente Bakterien. Diese Fauna des *gesunden* Halses macht das Studium des *kranken* Halses zu einem ganz besonders schwierigen, so dass nur eine grosse Erfahrung und allersorgsamste Technik vor Trugschlüssen schützt. Von den früheren Untersuchungen, bei denen man weniger skrupulös zu Werke ging, ist deshalb vieles wertlos geworden.

Schon eine brauchbare Materialentnahme ist, wie Rupprecht in einer sehr gründlichen Arbeit[1] gezeigt hat, mit grossen Schwierigkeiten verknüpft. Dieser Autor hat eine grössere Reihe von Untersuchungen in der Weise angestellt, dass er bei einem vorher gut geschulten Material einen leicht zugänglichen Punkt der ganz gesunden Halsorgane mit einer ausgeglühten Platinnadel berührte. Der Platindraht wurde dann auf Bouillon abgeimpft, und *ausnahmslos* wurde auf diese Weise die Anwesenheit zahlreicher Mikroorganismen (Pneumokokken, Staphylokokken, Streptokokken, Stäbchen, Hefe, Sarcine), meist 2–3 Arten kombiniert, festgestellt. Wenn die subtilste Technik unter so günstigen Untersuchungsbedingungen eine so vielgestaltige Zufallsfauna ergibt, dann kann ein aus einem kranken Halse gewonnenes Abstrichpräparat nur dann Bedeutung haben, wenn es irgend einen Mikroorganismus in Reinkultur oder doch wenigstens nahezu in Reinkultur zeigt. Aber selbst ein derartiger Befund ist durchaus noch nicht ohne weiteres als bündiger Beweis anzusehen, muss man doch auch jetzt noch mit der Möglichkeit rechnen, dass der primäre Infektionserreger von einem sekundären überwuchert worden ist.

Etwas günstiger liegen die Chancen, wenn man bei serösen oder eitrigen Prozessen das Untersuchungsmaterial durch Punktion aus der Tiefe der Gewebe gewinnt. Bei diesem Vorgehen ist die Wahrscheinlichkeit, den Erreger der Erkrankung in Reinkultur zu erhalten, wesentlich grösser; eine *ganz sichere Gewähr* gibt aber auch dieses Verfahren nicht. Zwar halte ich die Gefahr einer Mischinfektion, d. h. einer Infektion, bei der von vornherein mehrere Infektionskeime in die Gewebe eindringen und dort in Wirksamkeit treten, nicht für so gross, als sie gewöhnlich angenommen wird. Dagegen muss man, meines Erachtens, gerade bei Halserkrankungen mit *Sekundärinfektionen*, d. h. mit der Infizierung des bereits erkrankten Gewebes durch das Hinzutreten eines zweiten wirksamen Infektionskeimes desto häufiger rechnen. Die Aggressivkraft der verschiedenen Bakterienarten ist ja nicht die gleiche; individuelle Unterschiede und die Besonderheiten der jeweiligen Situation spielen hierbei

[1] 'Zur Kenntnis der Laryngitis submucosa,' *Monatsschr. f. Ohrenheilkunde*, 1905, No. 2.

gewiss eine wesentliche Rolle. So überwindet der eine Krankheitserreger die lokalen wie die allgemeinen Widerstände schneller als der andere. Sind diese aber erst einmal durch einen besonders energischen Infektionskeim überwunden, dann gelingt es wohl auch den weniger aktiven Keimen, gelegentlich in die Tiefe des Gewebes vorzudringen und dort ihre verhängnisvolle Wirksamkeit zu entfalten.

Diese kann dann unter Umständen so stark werden, dass der primäre Infektionserreger vollständig in den Hintergrund gedrängt wird. Sehr schön wird diese Tatsache durch eine Beobachtung von Henke[1] illustriert.

Die Punktion eines peritonsillären Abscesses am 4. Tage der Erkrankung ergab Reinkultur von Streptokokken. Ein zweite Punktion, 24 Stunden später, ergab ' fast nur Staphylokokken '. Ein Zeitraum von 24 Stunden hatte also genügt, um den primären Infektionserreger, den Streptokokkus, durch die Sekundärinfektion des Staphylokokkus nahezu vollständig zu verdrängen. Gewiss mag in diesem Fall der Stichkanal der ersten Punktion die Chancen für die Sekundärinfektion besonders günstig gestaltet haben, es unterliegt aber keinem Zweifel und ist überdies durch zahlreiche Versuche bewiesen, dass es einer Verletzung hierzu nicht erst bedarf, die ' physiologischen Wunden ' der normalen und noch mehr die der bereits erkrankten Schleimhaut genügen vollauf.

Die Erfahrung Henkes, die durch ähnliche Beobachtungen an anderen Organen bestätigt wird, beweist also, dass die Mikroorganismen, die man bei längerem Bestehen einer Erkrankung durch Punktion, Excision oder auch durch Autopsie in der Tiefe der Gewebe findet, nicht *ohne weiteres* als das die vorliegende Erkrankung auslösende Agens anzusehen sind, selbst dann nicht, wenn sie in Reinkultur vorhanden sind und ihre Virulenz durch den Tierversuch erwiesen ist.

Weitere Anhaltspunkte für die Pathogenität der einzelnen Infektionserreger können sich aus der *Blutuntersuchung* ergeben ; oft lässt aber auch diese im Stich. Zuvörderst ist zu bedenken, dass die Bakterien nur bei besonders *schweren* Erkrankungen im kreisenden Blute nachgewiesen werden können. So hatte beispielsweise P. Lehmann[2] bei 75 Blutentnahmen von schweren Erysipelfällen nur dreimal einen positiven Befund ; und in 2 von diesen 3 Fällen war dieser Befund noch dazu durch eine Komplikation (Pneumonie), nicht durch die primäre Erysipel-Erkrankung bedingt. Ob es die Gefässwand des Lebenden ist, die, so lange sie durch die Erkrankung nicht allzu schweren Schaden genommen hat, durch ihre bakteriziden Kräfte die Entwicklung der Keime hemmt (Bertelsmann, P. Lehmann), oder ob die bei der Blutentnahme sich bildenden Komplemente zu viele Bakterienleiber zerstören, oder ob nur unter bestimmten Vorausbedingungen genügend grosse Bakterienmengen

[1] ' Ueber die phlegmonösen Entzündungen der Gaumenmandeln,' *B. Fränkels Arch.*, Bd. xxvii, p. 308.

[2] ' Beiträge zur Klinik des Erysipels mit besonderer Berücksichtigung des bakteriologischen Blutbefundes,' *Jahrb. d. Hamb. Staatskrankenanstalten*, Bd. xv, 1910, p. 28.

in die Blutbahn gelangen, das mag dahingestellt sein — jedenfalls müssen wir uns damit abfinden, dass mit unseren heutigen Hilfsmitteln im Blut des Lebenden nur unter gewissen Bedingungen pathogene Keime nachzuweisen sind. Wesentlich anders liegen die Verhältnisse bei postmortalen Blutuntersuchungen. Diese ergaben demselben Autor bei 11 Proben 8 positive und nur 3 negative Befunde.

Die Immunitätsforschung, die anderweitig durch die Untersuchung der jeweilig sich bildenden Antitoxine, Bakteriolysine, Agglutine und Präzipitine die Lehre von der Spezifität der einzelnen Krankheitserreger vielfach sehr geklärt hat, hat unsere Untersuchungen bisher noch nicht sehr gefördert. Wir dürfen aber wohl hoffen, dass die modernen Bestrebungen, welche durch das Studium der Fermentwirkungen in das Wesen und Wirken der Bakterien einen besseren Einblick zu gewinnen suchen, auch uns noch zugute kommen werden.

Es schien mir unerlässlich, in dem Augenblick, wo wir unsere Erkenntnis von dem Wesen dieser Erkrankungen mit den Ergebnissen der modernen Forschung in Einklang zu bringen versuchen, auf all die Schwierigkeiten hinzuweisen, mit denen eine Neuordnung dieser Materie auf Grund ihrer ätiologischen Faktoren zu rechnen hat. Haben sich doch von den früheren Untersuchungsergebnissen viele als unbrauchbar erwiesen, weil die Cautelen, die man damals für ausreichend hielt, sich im Laufe der Zeiten als ungenügend erwiesen haben, und es hat mühevolle Arbeit genug gekostet, die Trugschlüsse, die auf diesen irrtümlichen Voraussetzungen aufgebaut waren, wieder auszuschalten.

Von den bisher bekannten Mikroorganismen kommen als Erreger von akut-entzündlichen Halserkrankungen folgende in Betracht:

1. Der Loeffler'sche Diphtheriebacillus.
2. Der Bacillus fusiformis (eventuell in seinem Zusammenwirken mit Spirochaeten).
3. Der Bacillus mucosus.
4. Das Bakterium Coli.
5. Der Pneumokokkus.
6. Der Staphylokokkus.
7. Der Streptokokkus.
8. Der Bacillus tetraginus und megatorium.

1. *Der Loeffler'sche Diphtheriebacillus.* — Dieser ist der einzige von den eben aufgezählten Krankheitserregern, der nach dem Stand unseres heutigen Wissens mit Sicherheit als ein ' spezifischer ' zu bezeichnen ist. Denn immer löst er dasselbe Krankheitsbild aus, und dieses wird von keinem anderen Infektionserreger ausgelöst.

Diese Spezifität der Krankheitserscheinung findet aber nicht, wie man früher allgemein annahm, ihren Ausdruck in der Bildung der pseudomembranösen, diphtherischen Beläge. Wir wissen heute vielmehr, dass die Halsorgane durchaus nicht immer nur durch die pseudomembranösen,

sondern auch durch andere Entzündungsformen auf den Angriff des Diphtheriebacillus reagieren, und dass andererseits pseudomembranöse Beläge, die von den echt diphtherischen weder makroskopisch noch mikroskopisch zu unterscheiden sind, auch durch andere Infektionserreger hervorgerufen werden können.

Es gehört also der pseudomembranöse Belag, der früher als Charakteristikum der Diphtherie angesehen wurde, nicht zu ihren ' wesentlichen ' Merkmalen.

2. *Bacillus fusiformis.* — Ob die Plaut-Vincenti'sche Angina als eine spezifische Erkrankung anzusehen ist, die durch den Bacillus fusiformis mit oder ohne Beihilfe von Spirochaeten hervorgerufen wird, ist heute noch nicht sicher erwiesen. Gerade diese Erkrankung legt Zeugnis ab für die grossen Schwierigkeiten, mit denen diese Untersuchungen zu kämpfen haben. Durchaus nicht selten, von vielen sorgsamen Autoren bearbeitet, kann man doch nicht recht dahinterkommen, ob es sich hier wirklich um eine spezifische Erkrankung im engeren Sinne handelt und ob das Plaut'sche Bakteriengemisch oder der Bacillus fusiformis allein Träger dieser Spezifität ist.[1]

Dass aber die Plaut-Vincenti'sche Angina hierher gehört, dafür sprechen neben der Uniformität der lokalen und regionären Prozesse und die in ihrem Verlauf auftretenden Allgemeinerscheinungen, auf die besonders Reiche in seiner letzten Arbeit hinweist : Temperatursteigerung, Leukocytose, Milzschwellung, gelegentlich auch Nephritiden, Lähmungen und myokarditische Störungen.

3. *Der Bacillus mucosus.* — Unsere Kenntnisse über die Pathogenität des Bacillus mucosus sind gering und nicht sehr zuverlässig. Man hat ihn nur bei leichten oberflächlichen Schleimhauterkrankungen erythematöser und katarrhalischer Natur gefunden. Ob ihm bei diesen Erkrankungen eine ursächliche Wirkung zukommt, oder ob er nur ein harmloser Saprophyt ist, ist zur Zeit noch nicht ganz sicher gestellt.

4. *Das Bakterium Coli.* — Die verschiedenen Typen dieser Bakteriengruppen gehören zwar nicht zu den regelmässigen Bewohnern der Mund- und Rachenhöhle, werden aber hier auch unter normalen Verhältnissen des öfteren als unschuldige Schmarotzer gefunden.

Aber nicht immer ist die Rolle, die sie spielen, eine so harmlose. Unter gewissen Bedingungen entfalten sie eine energische organfeindliche Tätigkeit und rufen dann entweder durch Verschleppung von Bakterien sekundäre oder auch ohne solche Vorläufer direkte primäre Affektionen der Halsorgane hervor. Von den diesbezüglichen Mitteilungen[2] mögen

[1] Arth. Meyer, *Samml. klin. Vortr.*, Serie 12, Hefte 26 und 27, 1908 ; F. Reiche, *Jahrb. d. Hamb. Staatskrank.*, 1905 und 1910.

[2] Lermoyez, Helme et Barbier, ' Un cas d'amygd. chron. coli bacill.,' *Ann. des mal. de l'or.*, 1894, n° 8, p. 787. Chaillon et Martin, ' Nouv. études sur la dipht.,' *Ann. de l'Institut Pasteur*, 1894, n° 7. Widal, ' Sur un cas d'ang. phleg. à coli bacille,' *Bull. et Mém. de la Soc. méd. des Hôpit. de Paris*, 1894, 15 févr. Maurel, *De l'infect. et de la contag. de l'amygd. aig.*, Thèse de Paris, 1895. Bourges,

einige, die zeitlich ziemlich weit zurückliegen, nicht ganz einwandfrei sein. Es bleiben aber doch noch eine ganze Reihe zuverlässiger Beobachtungen übrig, die an der pathogenen Bedeutung des Bakterium Coli keinen Zweifel lassen.

So berichten Lermoyez, Helme und Barbier über einen Anginafall, bei dem das Colibakterium nicht nur durch Abstrich in Reinkultur gewonnen wurde, sondern sich auch in der Tiefe des Gewebes in grossen Mengen nachweisen liess. Ist es auch nicht ganz sicher, dass die Affektion in diesem Falle von Anfang an eine typische Coli-Erkrankung war — der Patient litt häufig an Anginen und war diesmal schon 2 Monate krank, als die bakteriologische Untersuchung vorgenommen wurde — so ist es doch angesichts des Befundes höchst wahrscheinlich, dass zur Zeit der bakteriologischen Untersuchung das Bakterium Coli für das Krankheitsbild verantwortlich zu machen war. Wenn nun aber die Autoren aus dem Verlauf dieses Falles eine Reihe von Erscheinungen herausheben, die sie als charakteristisch für die Coli-Angina bezeichnen: chronischer Verlauf, Hartnäckigkeit, Geringfügigkeit der lokalen Erscheinungen, die in keinem rechten Verhältnis zu den schweren Allgemeinerscheinungen stehen, die etwas abweichende Form des lokalen Exsudates, — so zeigen andere Beobachtungen, dass die hier gesehene Form der Reaktion nicht die einzige ist, durch die der Organismus auf den Angriff des Coli-Bakterium antwortet. So sah Widal z. B. eine eitrige Phlegmone, aus der er das Bakterium Coli in Reinkultur gewann; nach Bourges und Houdelo dürfte das Bakterium Coli gelegentlich auch bei der Bildung pseudomembranöser Beläge beteiligt sein; andere Autoren sahen anginaartige Zustände, die sich von Streptokokken-Anginen klinisch nicht unterscheiden liessen.

Nach alledem besteht kein Zweifel, dass das Bakterium Coli akutentzündliche Affektionen der Halsorgane auslöst, die lokal und konstitutionell die verschiedenartigsten Formen annehmen können und klinisch von Halsaffektionen anderer Provenienz gar nicht zu unterscheiden sind.

5. *Der Pneumokokkus.*[1] — Der Pneumokokkus gehört zu den gewöhnlichen Bewohnern der Mund-Rachenhöhle. Rupprecht fand ihn bei 22 gesunden Individuen 21 mal, Ruediger in 90 Prozent seiner Untersuchungen. In all diesen Fällen ein harmloser Schmarotzer, wandelt er recht oft seine Natur und wird zu einem höchst gefährlichen Feinde des Organismus. Aber da er auch unter normalen Verhältnissen ein fast ständiger Gast in diesen Regionen ist, so ist man, nur wenn er im Blut, im Gewebe oder in ungefährer Reinkultur im Abstrich gefunden wird,

Les angines de la scarlatine, Thèse de Paris, 1891. Houdelo et Bourges, *Société de Biologie*, Séance du 27 janvier 1894. Macaigne, *Le bactérium coli commun*, Thèse de Paris, 1892. Lemoine, 'Contribut. à l'étude bactériol. des angines sur dipht.,' *Ann. de l'Institut Pasteur*, t. ix, nº 12.

[1] Ich fasste den Diplokokkus lanceolatus, den A. Fränkel'schen und Weichselbaum'schen Kokkus unter diesem Namen zusammen, da eine präzise Scheidung dieser Arten bakteriologischerseits noch nicht durchgeführt ist.

berechtigt, ihn als den tatsächlichen Erreger der vorliegenden Krankheiterscheinungen anzusehen. Und auch dann ist immer noch zu bedenken, dass der Pneumokokkus gewiss oft Sekundärinfektionen veranlasst, bei denen er dank seiner Wachstums- und Wucherungsenergie den primären Krankheitserreger leicht überwuchert. Ueber solche Fälle berichtet P. Lehmann.[1]

Erfolgreiche Agglutinationsversuche wurden angestellt von F.Bezançon und V. Griffon.[2]

Die Zahl der primären Pneumokokken-Anginen, über die in der Literatur berichten wird, ist überaus gross. Sie wurde angeblich zuerst beschrieben im Jahre 1891 von Jacoud[3]; seitdem haben die verschiedensten Autoren versucht, das Typische aus dem Krankheitsbild der Pneumokokken-Angina herauszuheben. Ich muss mich darauf beschränken, einige wenige Namen zu nennen, denen wir auf diesem Gebiet Fortschritte oder wenigstens gewisse Sicherstellungen verdanken.

Mme Sophie Weinberg[4] stellte schon im Jahre 1895 9 Fälle von Pneumokokken-Angina zusammen, auf Grund deren sie zu dem Schlusse kommt, dass der Pneumokokkus ungefähr dieselben Veränderungen hervorruft, wie der Diphtheriebacillus: feste Pseudomembranen, Drüsenschwellung, Fieber, schwere Allgemeinerscheinungen — charakteristisch sei nur der Beginn der Pneumokokken-Angina, sie setze plötzlich mit Schüttelfrost und hohem Fieber ein, ähnlich wie Pneumonie.

Fünf Jahre später veröffentlichte Emil Mayer[5] eine Reihe eigener und fremder Beobachtungen und kommt zu der Schlussfolgerung, dass die durch den Friedländer'schen Pneumobacillus bedingten Anginen subakuter oder chronischer Natur seien, dass sie in membranöser Form abblätternd und recidivierend auftreten und fast gar keine Störung verursachen, ausser vielleicht zur Zeit der Bildung des Belages.

Im Jahre 1905 schreibt Pasteur[6], dass die akute Pneumokokken-Angina bald als membranöse, diphtherieähnliche Angina, bald als erythematöse scharlachähnliche Erkrankung auftritt.

Im Jahre 1907 erschien eine sehr sorgsame Abhandlung von F. Reiche und Schomerus[7] mit zahlreichen genauen Eigenbeobachtungen und Literaturangaben. Danach tritt die Pneumokokken-Angina auf unter folgenden Erscheinungsformen:

a. Als akut entzündliche Pharyngitis s. Angina erythematosa.

b. Als Angina pseudomembranacea, exsudativa oder membranoulcerosa.

[1] Loc. cit.

[2] 'Le pneumocoque dans les angines,' *Presse méd.*, 1900, 24 octobre.

[3] *Journ. de méd. et chir. prat.*, mars 1891.

[4] *De l'angine à pneumococcus*, Thèse de Paris, 1895.

[5] 'Anginen d. Friedländer'schen Bacillus,' *B. Fränkels Arch.*, Bd. xi, p. 247.

[6] 'On Pneumococcal Throat,' &c., *Lancet*, 1905, No. 27.

[7] 'Die durch den Diplokk. lanceol. hervorgerufenen Rachen- und Kehlkopferkrankungen nebst Bemerkungen über d. Erysipelas cutis pneumococc.,' *Jahrb. d. Hamb. Staatskrank.*, 1907, p. 289.

c. Als Angina follicularis.

d. Als Angina apostematosa.

Am Kehlkopf traten die Pneumokokken-Erkrankungen unter folgenden Erscheinungsformen auf:

a. Als erythematöse Entzündungen.

b. Als pseudomembranöse Entzündungen.

c. Als phlegmonöse und erysipelatöse Entzündungen.

Also beinahe in allen nur möglichen Erscheinungsformen!

Ein besonderes Interesse verdienen die erysipelatösen Fälle.[1] Nach dieser Veröffentlichung typischer erysipelatöser Erkrankungen des Kehlkopfes durch Pneumokokken, nach dem v. Leube'schen Fall,[2] nach den höchst interessanten Versuchen von Neufeld[3], der auf hämatogenem Wege durch Pneumokokken beim Kaninchen Erysipel erzeugte, kann es keinem Zweifel unterliegen, dass auch der Pneumokokkus ebenso wie der Streptokokkus, typisches Erysipel hervorrufen kann, dass dieses *also nicht eine spezifische Streptokokken-Erkrankung* ist.

Eine gewisse Aehnlichkeit mit einigen von den früher veröffentlichten pseudomembranösen Erkrankungen, aber doch auch wieder recht wesentliche Unterschiede weisen die Fälle auf, die Semon[4] im Jahre 1907 als 'Pneumokokken-Invasion des Halses' bezeichnete. Bei diesen nicht eben häufigen Erkrankungen — auch ich hatte einen solchen Fall zu beobachten Gelegenheit — ist der Verlauf ein ausserordentlich chronischer: in meinem Falle dauerte die Erkrankung viele Monate, die Schleimhaut des Rachens und der Mundhöhle war meist mit inselartigen, weissglasigen Belägen bedeckt, die aber zeitweise ganz verschwanden. Gelegentlich kommt es zu oberflächlichen, manchmal, aber selten, auch zu tieferen Substanzverlusten. Die Temperatur ist schwankend, meist nur wenig erhöht, gelegentlich aber auch bis zu 39° und darüber ansteigend. Das Allgemeinbefinden ist zeitweise beträchtlich gestört. Meine Patientin behauptete aber recht oft, dass sie sich ganz wohl fühle. Die bakteriologische Untersuchung ergab immer unzweideutig, dass eine Pneumokokken-Erkrankung vorlag.

Weitere 3 ähnliche, aber doch wieder etwas mehr an die aphthöse Form der Stomatitis erinnernde Fälle sind von Mann[5] beschrieben worden. Auch hier war zweifelsohne der Pneumokokkus als Krankheitserreger anzusehen, und auch hier handelte es sich neben der eigenartigen entzündlichen Lokalinfektion um eine schwere Allgemeinerkrankung.

Das Gesamtergebnis der bisherigen Untersuchungen über die ätio-

[1] Verschiedene hierhergehörige Fälle mit schweren Komplikationen sind in dem Aufsatz von Liebe, 'Ueber eine Endemie v. Tonsillitis mit Sepsis,' *Med. Klinik*, 1910, p. 62, erwähnt.

[2] 'Ueber einen seltenen Fall von Pneumokokkenerysipel,' *Münch. med. Woch.*, 1906, p. 1789.

[3] 'Ueber d. Erzeugung von Erysipel am Kaninchenohr durch Pneumokokken,' *Zeitschr. f. Hyg.*, 1901, Bd. xxxvi, p. 254.

[4] Loc. cit., Bd. ii, pp. 425 u. f.

[5] *Münch. med. Woch.*, 1909, p. 73.

logische Bedeutung des Pneumokokkus für Halserkrankungen lautet also demnach:

Der Pneumokokkus verursacht recht häufig akut-infektiöse Erkrankungen der Halsorgane, die an den verschiedensten Stellen der Mund-, Rachen-, Kehlkopfschleimhaut, an den Tonsillen u.s.w. lokalisiert sein können. Die Erscheinungsform dieser Pneumokokken-Affektionen ist sehr verschiedenartig. Sie treten auf:

a. Als erythematöse Erkrankungen.

b. Als pseudomembranöse, aphthöse und ulzero-membranöse Erkrankungen.

c. Als lacunäre und folliculäre Erkrankungen an den Tonsillen.

d. Als phlegmonöse und abscedierende Erkrankungen.

e. Als erysipelatöse Erkrankungen.

6. *Der Staphylokokkus.* — Dieser Allerweltsschmarotzer fehlt auch in der gesunden Mund- und Rachenhöhle fast nie. Hier, wie auch sonst, ist und bleibt er für gewöhnlich unbegrenzte Zeit über ganz harmlos, dringt er aber in die Tiefe der Gewebe ein, so richtet er, sowohl örtlich wie durch Allgemeininfektion schwersten Schaden an, obgleich die Schleimhäute augenscheinlich einen weniger günstigen Boden für seine Entwicklung bilden, als die äussere Haut, die eigentliche Domäne der Staphylokokken-Affektionen.

Eine Abgrenzung der verschiedenen Staphylokokken-Arten (Staphylokokkus albus, aureus, citreus) nach ihrer Wirkungsweise ist zur Zeit noch nicht durchführbar.

Die Lebensenergie der Staphylokokken ist eine sehr grosse; sie wachsen und vermehren sich ungeheuer schnell und überwuchern andere Infektionserreger oft schon in wenigen Stunden so energisch, dass diese kulturell dann gar nicht mehr nachweisbar sind (Sendziak,[1] Goldscheider,[2] P. Lehmann [3]).

Nach der übereinstimmenden Ansicht aller Autoren, die sich mit dieser Frage beschäftigt haben (Sendziak,[1] Goldscheider,[2] P. Lehmann,[3] B. Fränkel,[4] Ritter,[5] Ed. Meyer,[6] M. Stoos,[7] Fürbringer [8]), kommt der Staphylokokkus nur selten als Erreger erythematöser oder katarrhalischer Erkrankungen der oberen Luftwege, insbesondere der lacunären Angina in Betracht. Ebenso scheint er bei aphthösen [9] und bei pseudomembranösen [10] Entzündungsformen nur selten eine Rolle zu spielen.

[1] Sendziak, *B. Fränkel's Arch. f. Laryng.*, Bd. ii, p. 180.
[2] Goldscheider, *Zeitschr. f. klin. Med.*, Bd. xxii, p. 534.
[3] P. Lehmann, loc. cit.
[4] B. Fränkel, *Angina lacunaris et diphtherica*, 1886.
[5] Ritter, *Berl. klin. Woch.*, 1892, 4 und 10. Disk. zu Baginskys Vortrag.
[6] Ed. Meyer, *B. Fränkel's Arch.*, Bd. iv.
[7] M. Stoos, *Zur Aetiologie und Pathol. d. Angina*, etc., 1895.
[8] Fürbringer, *Verhandl. d. Ver. f. inn. Med. zu Berlin*, 1888.
[9] Jadassohn-Baumgarten, *Jahresber*, 1895.
[10] Kolle und Wassermann, Bd. iv, p. 389.

Wesentlich bedeutsamer, wenn auch nicht zahlenmässig, so doch durch die Schwere der von ihm ausgelösten lokalen und allgemeinen Erscheinungen, ist die Rolle, die der Staphylokokkus in der Aetiologie der infiltrierenden, phlegmonösen, abscedierenden und erysipelatösen Prozesse spielt. Solche Fälle werden berichtet von Kocher, Tavel und Roos [1], Bonome-Uffreduzzi, M. Jordan, Felsenthal [2].

All diese Veröffentlichungen bestätigen den Eindruck, dem Lennhartz [3] schon Ausdruck gegeben hat, als er die Staphylokokken-Schleimhautaffektionen für relativ seltene, dafür aber recht bösartige Erkrankungen bezeichnete. Der Grund für die besondere Bösartigkeit der Staphylokokken-Infektion liegt in der Eigenart ihrer biologischen Verhältnisse.

Der Staphylokokkus ist ' der Typus der Entzündungserreger ', und ' die Entzündungen, welche er hervorruft, können vom Standpunkt des Pathologen als Typus der Entzündung betrachtet werden '.

Sind auch bei jeder lokalen Staphylokokken-Erkrankung die vier Kardinalsymptome der Entzündung deutlich ausgeprägt, so tritt doch bei weiterem Fortschreiten des Entzündungsprozesses die Auswanderung der weissen Blutkörperchen, bedingt durch die chemotaktische Wirkung der Staphylokokken, in den Vordergrund.[4] Die schnelle Erweichung dieses Infiltrates, die wieder durch Toxine der Staphylokokken veranlasst wird, und die Einwirkung ihrer verhängnisvollen Fermente, des Hämolysins und des Leukocidins, sind es, die den Staphylokokken-Erkrankungen der Halsorgane einen so bösartigen Charakter verleihen.

Noch nicht ganz geklärt scheint mir die Frage, ob der Staphylokokkus auch typisches Erysipel hervorrufen kann. In meiner früheren Publikation hatte ich auf Grund einiger älterer literarischer Angaben, von Bonome und Uffreduzzi, M. Jordan, Felsenthal, besonders aber auf Grund eigener Tierversuche und eigener klinischer Beobachtungen diese Frage bejaht. Heute stehe ich ihr etwas skeptischer gegenüber. Es lassen sich gegen diese früheren Beobachtungen (meine eigenen mit einbegriffen) doch mancherlei Einwände erheben, und auch der neue, von Jochmann mitgeteilte Fall [5] erscheint mir noch nicht ganz überzeugend. Wenn auch die Infiltration in dem Jochmann'schen Falle weit mehr im Corium als im subkutanen Gewebe gesessen hat, so vermisse ich doch die für das Erysipel charakteristische Tendenz, sich schnell über weite Strecken hin auszudehnen : am 18. September zeigte sich an der Nase der Patientin die primäre Pustel, am 20. bereits wurde die Diagnose ' Rose ' gestellt, und bis zum Tode am 24., d. h. innerhalb von 5 Tagen, hatte sich die Erkrankung in schweren, hart infiltrierenden Nachschüben im Ganzen nur bis zur behaarten Kopfhaut derselben Seite ausgedehnt, während am Ausgangspunkt der Erkrankung die Entzündungserscheinungen unverändert

[1] Siehe Kolle und Wassermann, loc. cit.

[2] Siehe meine Monographie über Larynxödem, etc.

[3] Nothnagel, *Spec. Pathol. und Therap.*, Bd. iii, pp. 3–4.

[4] Kolle und Hetsch, pp. 283–4.

[5] ' Zur Frage des Staphylokokkenerysipels,' *Mitteil. a. d. Grenzgeb. d. Med. u. Chir.*, 1906, p. 76.

geblieben waren. Dieses ganze Krankheitsbild scheint mir, trotzdem es seinen hauptsächlichen Sitz im Corium hatte, doch mehr einer langsam sich ausbreitenden Phlegmone als einem typischen Erysipel zu entsprechen.

Nun ist ja, wie ich schon im Jahre 1895 betont habe, die pathologisch-anatomische Definition des Erysipels eine sehr schwankende. Komplikationen durch tief greifende Infiltrate und phlegmonöse Prozesse sind so häufig, dass viele Autoren sie als zum Wesen des Erysipels zugehörig betrachten. Nun trete ich heute wie damals voll und ganz für die Zusammengehörigkeit der Phlegmone und des Erysipels ein, aber es scheint mir doch wichtig, in der Theorie wenigstens die beiden Prozesse auseinander zu halten. Das Typische beim Erysipel sehe ich nach wie vor darin, dass 1. die Haupterkrankung in der Mucosa, beziehungsweise in der Cutis sitzt, während die Phlegmone sich hauptsächlich in den tieferen Gewebeschichten abspielt, und 2. in der Flüchtigkeit seines Kommens und Gehens, während die Phlegmone nur langsam weiterkriecht und ebenso langsam verschwindet. Dass in der Praxis diese Grenzlinien unzählige Male verwischt werden, so dass man sehr oft nicht weiss, ob man den vorliegenden Krankheitsfall als ' Phlegmone erysipelatosum ' oder als ' Erysipelas phlegmonosum ' bezeichnen soll, ist ja hinreichend bekannt. Aber die Flüchtigkeit der Erscheinungen dürfte doch meist einen Anhaltspunkt für die Entscheidung geben. Diesen Kriterien aber scheinen mir die bisher veröffentlichten Staphylokokken-Erysipele — auch die von mir s. Z. experimentell erzeugten — doch nicht ganz gerecht zu werden. Deshalb meine ich, dass die Staphylokokken für erysipelatöse Erkrankungen der Haut und der Schleimhaut wenn überhaupt, dann nur recht selten in Frage kommen, jedenfalls weit seltener, als die Pneumo- und Streptokokken. Dafür sprechen neben diesen eben erörterten klinischen Beobachtungen auch die Ergebnisse vieler experimenteller Untersuchungen. Während Schurmayer, Eugen Fränkel, v. Lingelsheim, Fehleisen, A. Fränkel, ich selbst u. a. durch Verreibung von Streptokokken am Kaninchenohr erysipelatöse Prozesse jederzeit hervorrufen konnten, während Abrami und Richet fils[1] und ich selbst auch auf hämatogenem Wege durch Streptokokken, F. Neufeld[2] durch Pneumokokken Erysipel erzeugen konnten, blieben alle entsprechenden Versuche mit Staphylokokken bisher resultatlos

Es veranlassen also die Staphylokokken verhältnismässig selten anginöse, aphthöse, oder pseudomembranöse Entzündungen im Hals und Rachen. Dagegen bewirken sie öfters schwere infiltrierende, phlegmonöse und abscedierende Entzündungen mit ernsten Allgemeinerscheinungen. Typisches Erysipel aber dürften sie, wenn überhaupt, nur äusserst selten hervorrufen.

7. *Der Streptokokkus.* — Der Streptokokkus, dessen verschiedene Typen betreffs ihrer Pathogenität noch nicht genügend studiert sind, soll

[1] ' L'érysipèle hématog.,' *Compt. rend. de la Soc. de Biol.*, 1909, t. ii, p. 562.

[2] l. c.

hier als eine einheitliche Spezies betrachtet werden. Auch er findet sich fast ausnahmslos in der gesunden Mund- und Rachenhöhle, ohne hier irgendwelche Krankheitserscheinung auszulösen. Dabei ist er, wenn seine Virulenz die Abwehrkräfte des Organismus überwindet, der häufigste und gefährlichste, der *typische* Infektionserreger für die Schleimhäute der oberen Luftwege. Bei jeder Form der Erkrankung, beim Erythem und beim Katarrh, bei der hämorrhagischen wie bei der pseudomembranösen Entzündung, bei der Phlegmone, beim Abscess, beim typischen Erysipel — bei all diesen Erkrankungsformen hat man den Streptokokkus als unzweifelhaften Krankheitserreger so oft nachgewiesen, dass ich betreffs dieser von keiner Seite mehr bestrittenen Tatsache wohl auf die Anführung literarischer Beläge verzichten darf. Die Streptokokken-Infektion überwiegt bei den genannten Affektionen, ganz besonders aber beim Erysipel, zahlenmässig die Infektion durch alle anderen Mikroorganismen zusammen genommen

Hervorheben möchte ich nur, dass es auch 4 Streptokokken-Fälle waren, durch die mein Korreferent, Herr Dr. Ph. de Santi[1] den von mir und Felix Semon seit 1895 vertretenen Ansichten über die Zusammengehörigkeit der akut-infektiösen, submukösen Entzündungen auch in England zu endgiltiger Anerkennung verhalf.

Ebenso besteht heute nirgends mehr ein Zweifel darüber, dass die schwersten Komplikationen und Allgemeinerscheinungen — Pericarditis, Nephritis, Orchitis, Appendicitis, rheumatische Affektionen, Meningitis, Osteomyelitis, Pyämie, Sepsis, u.s.w. im Verlauf von Streptokokken-Erkrankungen der Halsorgane besonders häufig auftreten, und dass es durchaus nicht immer besonders schwerer Lokalaffektionen bedarf, um diese gefährlichen Folgeerscheinungen auszulösen. Im Gegenteil, oft genug sind die Reaktionserscheinungen an der Einfallspforte so geringfügig, dass sie ganz übersehen werden, bis die weit empfindlicheren Folgeerscheinungen, eine Polyarthritis, eine Nephritis oder Endokarditis die Aufmerksamkeit auf die Primäraffektion im Halse hinlenken.

Ueber die besonderen Bedingungen der Giftwirkung der Streptokokken sind wir noch nicht genügend aufgeklärt. Der Hämolysinwirkung, die gewissen Arten unzweifelhaft anhaftet, wird von den meisten Bakteriologen eine besondere Bedeutung nicht zugesprochen. Ob noch andere, zur Zeit unbekannte Fermente in Frage kommen, ob es Toxine oder Endotoxine sind, auf denen die Giftwirkung der Streptokokken beruht, ist ebenfalls noch nicht sicher gestellt.

8. *Weitere Mikroorganismen.* — Ausser den genannten Mikroorganismen habe ich in der Literatur noch einige andere Arten verzeichnet gefunden, die als Infektionsträger bei Halsentzündungen genannt werden, so der Mikrokokkus tetragenus, von F. Bezançon und V. Griffon,[2] Carrière,[3] Monnier[4] und Guillain und Rendu,[5] ferner ein ‘Bacillus

[1] l. c.
[2] *Presse médicale*, 1900, 24 octobre.
[3] *Revue de Médecine*, 1902, juin.
[4] *Arch. internat. de Laryngol.* 1904.
[5] *Société méd. des Hôpitaux de Paris*, 1907, 22 février.

megatorium' von H. Vincent.[1] Vielleicht kommen noch einige andere in Betracht, die mir entgangen sein mögen.

Aus dieser Betrachtung ergibt sich, dass von den Infektionserregern der akut-entzündlichen Halserkrankungen jeder einzelne — verschiedenartige — der Streptokokkus und Pneumokokkus sogar alle überhaupt in Betracht kommenden Formen lokaler und allgemeiner Reaktionserscheinungen auslösen kann. Kaum bei einem einzigen von all diesen Krankheitsbildern kann man aus dem lokalen Befunde oder dem klinischen Verlauf allein die Art des Krankheitserregers *mit Sicherheit* erkennen — *also auch in der Vielheit der Infektionserreger eine Einheitlichkeit ihrer Wirkung.*

Zusammenfassung

Die primären, akut-entzündlichen Affektionen des Schlundes, Rachens und Kehlkopfes, soweit sie nicht mechanischen, chemischen oder thermischen Ursprunges oder Teilerscheinungen konstitutioneller Erkrankungen sind, wurden bisher in zahlreiche Arten und Unterarten geschieden.

Alle diese Krankheitsformen bilden eine grosse *einheitliche ätiologisch, pathologisc-hanatomisch und klinisch eng zusammengehörige Gruppe.*

1. *Aetiologisch.* — Sie alle werden hervorgerufen durch pathogene Mikroorganismen. Nur die Diphtherie und vielleicht auch die Angina Plaut-Vincenti sind spezifischer Natur. Jede von den anderen Affektionen kann durch verschiedene Mikroorganismen — Bakterium Coli, Pneumokokkus, Staphylokokkus, Streptokokkus etc. veranlasst werden, ohne dass durch die besondere Eigenart des Infektionserregers irgend welche Besonderheiten des Krankheitsbildes bedingt würde.

2. *Pathologisch-anatomisch.* — Die durch die Einwirkung dieser Infektionskeime hervorgerufenen örtlichen Veränderungen zeigen im Anfang der Erkrankung stets und überall die vier Kardinalsymptome der Entzündung. Bei weiterem Fortschreiten des Prozesses pflegt dann bald das eine, bald das andere dieser Symptome in den Vordergrund zu treten, oft so stark, dass die anderen Symptome in den Hintergrund gedrängt werden. Diese Differenzierung in der Entwicklung der Entzündungsvorgänge ist es, die neben den Besonderheiten, die sich aus der Lokalisation des jeweiligen Angriffspunktes, aus der Virulenz und Menge des jeweiligen Infektionsgiftes, aus der jeweiligen örtlichen und allgemeinen Widerstandskraft ergeben, die Verschiedenartigkeit des klinischen Verlaufes der ganzen Erkrankung bedingen.

So verschieden aber auch das pathologisch-anatomische Bild eines Erythems, einer pseudomembranösen Entzündung, einer Phlegmone und eines Erysipels sein mögen — die zahlreichen Uebergänge von der einen Erkrankungsform zur anderen, ihr bald stadienartiges Nacheinander,

[1] *Presse méd.*, 1902, 26 juin.

bald räumlich und zeitliches Nebeneinander beweist ihre Zusammengehörigkeit, die Einheitlichkeit ihrer Wesensart.[1]

3. *Klinisch.* — Trotz all der unendlich grossen Unterschiedlichkeiten im klinischen Verlaufe der verschiedenen akut-infektiösen Halserkrankungen — man vergegenwärtige sich nur den Verlauf einer einfachen Angina und vergleiche damit das Krankheitsbild einer schweren Halsphlegmone — gehören diese doch alle zusammen in ein und dieselbe Kategorie. Denn ihnen allen liegen die prinzipiell gleichartigen Gewebsentzündungen zu Grunde, und sie alle führen zu prinzipiell gleichartigen regionären und allgemeinen Krankheitserscheinungen — die leichteste Angina, so harmlos in ihren lokalen Symptomen, kann ebenso zu einer tötlichen Septikämie führen, wie eine schwere Halsphlegmone. Deshalb sind die klinischen Differenzen zwischen all diesen Erkrankungsformen — selbst die spezifischen Erkrankungen machen in dieser Beziehung keine Ausnahme — niemals prinzipieller Natur, sondern immer nur auf graduelle und örtliche Besonderheiten zurückzuführen. Von diesen kennen wir manche: die Menge des Infektionsstoffes und seine Virulenz, die örtliche und allgemeine Widerstandsfähigkeit des Organismus sind von wesentlichster Bedeutung. Die Lokalisation des ersten Angriffspunktes kann bestimmend sein für den ganzen Verlauf der Erkrankung, denn es macht einen Unterschied, ob die Infektionskeime sich im lymphatischen Rachenring, in der Schleimhaut des Kehlkopfes, oder im Boden der Mundhöhle zuerst festsetzen. Ebenso ob der Infektionserreger in den obersten Schichten der Schleimhaut bleibt, oder ob er seinen Weg in die Tiefe der Gewebe findet. Weshalb aber derselbe Krankheitserreger das eine Mal ein Erythem oder einen Katarrh, das andere Mal eine Phlegmone oder ein Erysipel auslöst, das wissen wir nicht. Aber welchen Grad und welche Form die Entzündung, der Schmerz, das Fieber, die Komplikationen auch annehmen mögen, es sind und bleiben immer nur wechselnde Erscheinungsformen eines ätiologisch, pathologisch-anatomisch und klinisch einheitlichen Prozesses.

Demnach möchte ich vorschlagen, das Kapitel *Die akut-infektiösen Entzündungen der Halsorgane* folgendermassen zu ordnen:

A.—Erkrankungen, welche sich hauptsächlich in der *Mukosa* abspielen.

1. Erythematöse Form.

2. Katarrhalische Form (serös, schleimig, schleimig-eitrig, hämorrhagisch).

3. Degenerative Form (fettige, hyaline Degeneration der Epithelschicht, Substanzverlust, Erosion, Ulceration).

[1] In meiner früheren Arbeit habe ich die verschiedenen Erscheinungsformen als 'Stadien'— Stadium oedematosum, plasticum, suppuraticum — bezeichnet. Man hat an dem Wort 'Stadium' Anstoss genommen, weil es sich, worauf ich s. Z. schon selbst hingewiesen habe, nicht nur um zeitlich aufeinanderfolgende Phasen einer Erkrankung handelt, sondern weil diese verschiedenen Krankheitsbilder oft auch als selbständige Krankheitsformen unabhängig voneinander vorkommen. Ich bin gern damit einverstanden, wenn das Wort 'Stadium' durch 'Form' ersetzt wird.

4. Fibrinöse Form (ohne und mit gleichzeitigen nekrotischen Prozessen. Aphthöse, pseudomembranöse, verschorfende Prozesse).

B.—Erkrankungen, die sich hauptsächlich in der *Submukosa* abspielen.

1. Oedematöse Form — entzündliches Oedem, Erysipel.
2. Plastisch-infiltrierende Form.
3. Phlegmonöse und abscedierende Form.

SECTION XV

RHINO-LARYNGOLOGY

INDEPENDENT PAPER

THE PATHOLOGY OF THE VARIOUS ACUTE INFLAMMATIONS OF THE THROAT AND NECK, INCLUDING ACUTE ŒDEMA, PHLEGMON, ERYSIPELAS, AND ANGINA LUDOVICI, BUT EXCLUDING DIPHTHERIA

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Under this title is included a number of forms of acute septic inflammations of the pharynx, larynx, and cellular tissue of the neck (angina Ludovici) which up to quite recently had been looked upon as pathologically and clinically different diseases: even to the present day confusion exists and most text-books still describe 'inflammatory œdema', 'phlegmon', 'gangrene and erysipelas', &c., of the pharynx and larynx as separate diseases.

In April 1895 Sir F. Semon read a paper at the Royal Medico-Chirurgical Society of London on 'The probable pathological identity of the various forms of acute septic inflammations of the throat and neck, hitherto described as acute œdema of the larynx, œdematous laryngitis, erysipelas of the pharynx and larynx, phlegmon of the pharynx and larynx, and angina Ludovici', and advanced the view mainly on clinical grounds that these various forms of acute septic inflammation of the throat and neck should be looked upon as varying degrees of virulence of one and the same pathological process, the primary seat and subsequent development depending in all probability on accidental breaches of the protective surfaces through which the pathogenic micro-organism causing the subsequent events found an entrance.

In February 1903 I read before the same society a paper on 'Four cases of acute septic inflammation of the throat with bacteriological examination of each' as a sequel to Sir Felix Semon's communication, and advanced proofs clinically and bacteriologically confirming Sir F. Semon's conclusions.

Since reading my paper I have had experience of a few more cases, and as to-day's communication is based on these cases, I shall forthwith give as briefly as possible notes of each.

CASE I. *Erysipelas of the pharynx.*

Male, aged 48, deep-seated cervical abscess, right side of neck, due to a Bezold's perforation of mastoid ; abscess in neck opened and radical mastoid operation performed. Some three weeks later during convalescence the temperature rose to 102·4°, and cutaneous erysipelas was noted on the left cheek ; this spread to the whole of the left side of the neck and face, then across the mid-line to the face and neck on the right side ; there were patches of redness also all over the back extending to the lumbar region. Soreness of the throat was complained of and a swollen bright red condition noted of the post-pharyngeal wall and whole of soft palate and uvula.

Cultures from the throat showed mainly streptococcus pyogenes with some staphylococci.

10 c.c. of a polyvalent anti-streptococcal serum injected into the abdominal wall, followed by a second and a third injection on succeeding days. Reaction occurred at once, and shortly after the last injection all rash had gone and the throat was normal.

CASE II. *Acute œdematous tonsillitis and pharyngitis.*

Male, aged 32, history of three days' illness, i.e. headache, shivering, sickness, and for twenty-four hours pain and dryness of throat increasing rapidly.

Patient looked ill, voice a whisper, temperature 104·6°, pulse rapid, 120, enlarged and tender glands both sides of neck. Some albumin in urine.

Throat : Intense redness and œdematous swelling of right tonsil, soft palate and uvula ; dark-red, glazed, and swollen condition of post-pharyngeal wall ; slight œdema of the epiglottis.

Swab cultures from the tonsil and pharynx showed streptococcus pyogenes in numberless chains. The patient refused admission to the hospital and the further history is unknown.

CASE III. *Acute gangrenous inflammation of the throat.*

Male, aged 21, sore throat, pain and difficulty in swallowing, loss of voice, and headache of two days' duration and rapidly getting worse. Previously very healthy. Temperature 101·2°, pulse rapid and soft.

Uvula much swollen, intensely red above, but lower half quite black and gangrenous. Soft palate red and œdematous and tonsils slightly so. Epiglottis slightly swollen. Extensive œdema of left aryepiglottic fold.

On admission patient could not swallow at all. Swab cultures from the uvula and pharynx revealed streptococcus pyogenes, some staphylococci, no diphtheria bacilli.

At once 20 c.c. of anti-streptococcal serum was injected into right lumbar region, and pure carbolic acid rubbed carefully into the gangrenous uvula.

Five hours later the patient could swallow. Three days later the patient was rapidly recovering and from then an uninterrupted recovery ensued. Only the one dose of serum was injected as the improvement following was so very marked.

CASE IV. *Acute pharyngitis due to streptococcus pyogenes followed by septicæmia, deep glandular inflammation and pericarditis.*

Male, aged 21, first taken ill with sore throat and swelling in neck October 16, got worse and was admitted into hospital October 24, partly unconscious and almost moribund. Temperature 101·4°, pulse 120, low tension. Severe præcordial pain. Tongue swollen and brown, posterior pharyngeal wall very swollen, darkish brown and some sticky mucus. Mouth difficult to open; large brawny red swelling left side of neck.

Auscultation: Loud pericardial friction rub. The swelling in neck had been incised but only a little sanious fluid escaped. Mucus from throat and discharge from cervical wound showed mainly in cover-glass specimens, and upon cultivation, streptococcus pyogenes. A yeast, staphylococcus albus, and a few bacilli were also found on cultivation of the mucus from the throat.

10 c.c. of anti-streptococcal serum injected at 9.30 p.m., at 8 a.m., and 4.20 p.m., next day, and a fourth dose on the following day. Patient began to rally at once, although not expected to live through the night, and made an excellent recovery.

CASE V. *Acute suppurative inflammation of the larynx.*

Male, aged 47, a private patient in previously excellent health. Sudden onset with chill, malaise and intense pain in the throat; temperature 102·4°, pulse 110, soft; within 24 hours of onset difficulty in breathing and swallowing.

When I saw the patient 48 hours after the first onset of disease, I found him very ill with dyspnœa, constant bad cough, and bringing up a lot of mucus and pus; the temperature was 102·6°, the patient was cyanosed, pulse rapid and small, anxious look. The pharynx was slightly congested; examination of the larynx showed very considerable acute œdema of the arytenoids, inter-arytenoid space and aryepiglottic folds and all the larynx was more or less bathed in pus. No tenderness of larynx externally.

20 c.c. of polyvalent anti-streptococcal serum injected at once and preparations made for emergency tracheotomy. Within 12 hours there was a marked change for the better; temperature 100·8°; muco-pus diminishing and breathing easier; patient made a rapid recovery. The pus from the larynx on cultivation showed pure streptococcus longus.

CASE VI. *Acute œdematous septic laryngitis in a boy of* 12.

Patient was a boarder at a large private school. There had been one case of diphtheria in the school seven weeks previously. The illness began with headache, lassitude, sickness, and some sore throat, and a temperature of 101·3°.

Patient was isolated, and swabs taken from the throat and nose; no diphtheria bacilli were found, but staphylococci and streptococci were present. Twenty-four hours later patient was much worse, the throat trouble being laryngeal.

I saw the patient late in the evening and found very embarrassed breathing, recession of chest, and muco-purulent matter being coughed up.

Examination showed nil in pharynx, but considerable œdema and redness of the epiglottis and posterior region of the larynx; the swollen parts were covered with pus, no sign anywhere of membrane. Temperature 102·6°; anxious appearance; sweating; rapid pulse. 10 c.c. of polyvalent anti-streptococcal serum injected into buttock; patient easier

6 hours later, and temperature 101·4° ; a second injection given 12 hours after the first ; good recovery.

CASE VII. *Case of acute septic tonsillitis and pharyngitis.*

Male, aged 32, private patient, illness taken to be an ordinary double quinsy ; three days after commencement of attack patient seemed very ill and general condition getting worse. I saw him in consultation and found intense sloughy suppurative inflammation of left tonsil and œdematous swelling of post-pharyngeal wall and opposite tonsil ; temperature 101·4° ; painful deglutition ; anxious look ; trace of albumin in urine ; ordinary remedies had had no effect.

I advised immediate injection of anti-streptococcal serum to be followed by an autogenous vaccine, but patient refused this line of treatment. He was treated with large doses of perchloride of iron, and although his convalescence was protracted, he got quite well.

Cultures from pus over left tonsil revealed almost pure streptococcus longus.

CASE VIII. *Acute septic inflammation of pharynx, tonsils, and buccal mucous membrane, with inflammation of submaxillary cellular tissue.*

The patient was a chef and a drinker. When seen by me he had considerable swelling and œdema of the pharynx and tonsils and buccal mucous membrane on right side ; there was an abscess round a carious right upper molar tooth.

The submaxillary region of neck on both sides was swollen, hard, and tender, and the floor of the mouth œdematous and prominent ; lower and upper lip much swollen. Temperature 101·6°. Patient was admitted into hospital, the tooth seen to, and 20 c.c. of polyvalent anti-streptococcal serum injected. An autogenous vaccine was prepared from cultures taken from the throat, mixed organisms being found. He reacted within a few hours and made a rapid recovery.

The submaxillary inflammation yielding at once to the serum did not require incisions. I was prepared in this case to give citric acid internally if necessary, as recommended by Sir A. Wright.

Now, in my opinion, the history, clinical symptoms, the course of the disease, and more particularly the bacteriological examination of my cases all point decisively to their pathological identity, and to the conclusion that each case should be considered as showing merely a varying degree of virulence of one and the same pathological process.

Since the reading of Semon's paper in 1895, great strides have been made in bacteriological research, and it is mainly on bacteriological grounds that one has to discuss these acute septic inflammations.

It is now universally acknowledged that these infections are due to the invasion of the system by pathogenic organisms, of which, at all events in my experience, the streptococcus pyogenes group is far the most important and frequent. Other organisms may be and are found, especially the staphylococcus aureus, albus, and citreus, pneumococcus, bacillus coli communis, and micrococcus tenuis, and should any one of these micro-organisms multiply sufficiently, then an acute septic inflammation may be produced which can only be differentiated from a streptococcal inflammation by bacteriological examination. There is, however,

no one specific organism for any one of these various inflammations: not infrequently more than one organism is present.

Again, the same organism, especially the streptococcus pyogenes, may produce widely varying results under different circumstances, at one time a local inflammation, at another erysipelas, at another an abscess, cellulitis or a general septicæmia (Marmorek's experiments); this is well exemplified in my series of cases, in all of which the streptococcus predominated, and yet in some cases there was so-called acute phlegmon, in others suppuration, erysipelas, and gangrene.

Formerly streptococcus pyogenes and streptococcus erysipelatis were considered as two different species, but it is now universally agreed that the two organisms are one and the same, erysipelas being produced when the streptococcus pyogenes of a certain standard of virulence gains entrance to the skin lymphatics.

These different effects then are attributable partly to differences in virulence and the number of micro-organisms gaining access, partly to the seat of infection and mode of entry into the body, partly to the resistance of the individual, and partly to real differences existing between different races of streptococci. Poynton believes there is another factor, viz. a tendency inherent in micro-organisms to produce special poisons, a specific toxicity which has to be reckoned with in all investigations on the unity of the streptococcal group.

Staphylococci usually and streptococci often can be cultivated from the secretions of the mouth in normal conditions. Fraenkel's pneumococcus and the pneumobacillus of Friedländer have also been found in the mouth and nasal cavity: the entrance of these organisms into the deeper tissues when a surface lesion occurs can easily be understood. The objection urged that the different localization of these cases spoke against their being identical is wrong. The different localization of these septic inflammations depends on the resisting powers of the parts attacked, an accidental breach of surface or a pre-existing condition of catarrh rendering the part more susceptible to infection.

As Semon has pointed out, diphtheria remains diphtheria whether it primarily attacks the throat, nose, conjunctiva, or vulva, and it is the same with these acute septic inflammations of the throat.

In my experience Ludwig's angina should be included in this class of case. It is a variety of submaxillary cellulitis, an acute suppurative, sometimes gangrenous, periglandular inflammation in the region of the submaxillary glands, and the infection usually occurs from a lesion of the oral buccal mucous membrane, from a carious tooth or perhaps inflamed tonsil. It is commonest in males of 20–30.

It is always very sudden and rapid in its course, the swelling at first very hard and brawny, pushing up the tongue and causing protrusion of the submental tissue. It is a particularly dangerous variety and requires very prompt surgical measures in addition to other treatment. I might mention here the use of citric acid internally. In a case seen

by Sir A. Wright of Ludwig's angina in which multiple incisions had been made early, the patient 24 hours later was collapsed and delirious: none of the gaping incisions showed any moisture. Film preparations showed abundant streptococci; the blood taken from a vein at the elbow clotted at once. Wright prescribed 60 grs. of citric acid every 3 hours: 6 hours later lymph began to ooze from the wounds and the acid was stopped. Improvement was so rapid that no vaccine was employed and there was complete recovery; other such cases have been recorded.

The citric acid lowers the coagulability of the blood so that lymph containing large amounts of anti-bacterial and anti-tryptic bodies are freely admitted to the infected parts and the organisms thereby destroyed before the formation of pus. *Clinically* in these cases of acute septic inflammation of the throat the following points are noteworthy. That the patients are usually adults, mostly men in the prime of life, in excellent previous health, and frequently, as in Semon's cases, of the higher social scale. In my cases, however, five were hospital and three private patients of good social standing.

That it is most important to recognize early the nature of the case and whether local or constitutional treatment is the more urgent.

That, as Dundas Grant has pointed out, sore throat and general bodily illness, a voice suggesting peritonsillar abscess, but ability to open the mouth, and nothing much to be seen in the throat, are conditions dangerous to life.

Finally, the question of serum and vaccine-therapy has a considerable bearing on the pathology of these diseases.

In 1903 I advocated strongly the immediate intra-muscular injection of 10–20 c.c. of anti-streptococcal serum and pointed out that all my cases had done remarkably well under that treatment. In my subsequent cases I have had equally good results from the same treatment, my cases being, mainly at all events, of streptococcal origin.

The last few years, however, vaccine-therapy has come into fashion, and the tendency in these cases is to use an autogenous or still more recently a sensitized vaccine.

In only one of my cases was an autogenous vaccine employed, and prior to its use an anti-streptococcal vaccine was used.

These streptococcal infections are primarily local, some becoming permanently circumscribed, others giving rise to septic and pyæmic conditions.

Ball, in his lecture on 'Acute infective processes due to the streptococcus' (*Lancet*, 1912, vol. i), emphasizes the following points in regard to serum and vaccine-therapy.

That the blood should be examined.

In all cases probably of streptococcal infection a certain number of bacteria do get into the blood-stream, though their presence cannot be demonstrated by the usual methods.

In others they get the upper hand and can easily be demonstrated in the blood-stream.

A streptococcus isolated from the blood is probably more virulent than that got from a local focus and therefore of more use in preparing a vaccine. There being many types of streptococcus an autogenous vaccine is of more use than a stock vaccine.

That the object of *serum*-therapy is to neutralize the toxins produced by bacteria by providing for the human tissues anti-bodies in larger quantities than they themselves possess ; it is mostly of use in the early stages of infection.

That the object of *vaccine*-therapy is to stimulate the human tissues to produce anti-bodies for themselves.

That the value of a vaccine on the temperature is shown in favourable cases by its fall to the normal by crisis within 24 to 48 hours after a single inoculation ; in other cases with a second or third inoculation.

That in approaching a case of infection due to the streptococcus :

(*a*) The clinical diagnosis should be accurate to the smallest detail, both as regards local and general symptoms ; and the help of the bacteriologist should be sought in all cases.

(*b*) The lesion, if necessary, should be dealt with surgically and the necessity for use of artificial methods of stimulation of the machinery of resistance should be then discussed.

(*c*) A serum should be given as early as possible and a type used that most nearly approaches the autogenous variety. Following the use of the serum or in combination with it (sensitized vaccine) inject an autogenous vaccine as soon as prepared.

(*d*) Dosage : no absolute rule. If seen very early, the general condition being good and the focus of infection circumscribed, a relatively large dose of vaccine may be given, otherwise a small dose is indicated as an initial inoculation. If no effect is produced by the smaller dose gradually increase it until such an amount is reached as will produce the desired reaction. If a harmful effect follows, a smaller dose is indicated or even vaccine-therapy may be contra-indicated.

DISCUSSION

Sir Felix Semon (London). Die Verwirrung, die auch heute noch hinsichtlich der Natur dieser Leiden herrscht, lässt sich leicht heben, wenn man von den althergebrachten Lehren einen Augenblick Abstand nehmen und die vorliegenden Tatsachen vorurteilsfrei betrachten kann. Es stellt sich dann heraus, dass die ganze Gruppe der pyogenen Mikroorganismen (Streptococcus, Staphylococcus, Bacterium coli, Pneumococcus etc.), so verschieden dieselben in der Kultur und unter dem Mikroskop erscheinen, doch individuell — mit alleiniger möglicher Ausnahme des Pneumococcus, unter gewissen Verhältnissen

worüber weiterhin mehr — keinen *spezifischen*, sondern einen untereinander *identischen* Einfluss haben, nämlich den: als *Entzündungs*erreger zu wirken. — Die von ihnen hervorgebrachten Entzündungen sind *nur graduell* voneinander verschieden, wobei jedoch die *verschiedensten* Grade der Entzündung vom leichten Erythem bis zu den schwersten oedematösen, erysipelatösen, phlegmonösen, gangränösen Formen vorkommen, und *so unmerklich ineinander übergehen*, dass jeder Versuch, sie wieder weiter in Unterabteilungen zu bringen, nur zu neuer Verwirrung führen kann. Der Sprecher warnt ausdrücklich gegen solche Versuche.

Therapeutisch rät er — aber nur als einen vorläufigen *Lückenbüsser* — zur Anwendung eines polyvalenten Streptococcenserums in *frühen* Stadien, selbst wenn es nicht möglich gewesen ist, sofort den im speziellen Falle wirklich vorhandenen Mikroorganismus tatsächlich nachzuweisen.

Mit Bezug auf Pneumococcusinfektionen unterliegt es keinem Zweifel, dass der Pneumococcus ebenso wie die anderen zur Gruppe der pyogenen Bakterien gehörigen Mikroorganismen, Krankheitsbilder akut entzündlicher Natur auslösen kann. Die von dem Sprecher im Jahre 1907 geschilderten Fälle aber, in denen bakteriologisch nichts als eine Reinkultur von Pneumococcen nachgewiesen werden konnte, erweisen, dass der Pneumococcus unter gewissen uns noch nicht bekannten Umständen (? verschiedene *Arten* von Pneumococcen, ? Erwerbung *spezifischer* Eigenschaften seitens des Pneumococcus) ein Krankheitsbild auslösen kann, welches nicht in den Rahmen irgendeines bisher bekannten Symptomenkomplexes passt und dringend weiteren Studiums bedarf.

Mr. DOUGLAS HARMER (London) said that the greatest care is necessary in treatment of acute inflammations with vaccines. If a non-sensitized vaccine is used, the doses must be very small at first and may be given after the method of Fornet and Müller. Non-sensitized vaccines are not so dangerous and may be given in large doses, up to 1,000 millions. They need not be autogenous if the type of infection is known. Rapid improvement may be obtained in some cases if the vaccine contains the causative organisms. Serum should be used to counteract the toxic symptoms, but is not so valuable as sensitized vaccines for cure of the disease.

Sir STCLAIR THOMSON (London) drew attention to the fact that the subject for debate was the *pathology* of the condition. He was afraid he might wander from the subject as his experience was chiefly clinical. Still, it was of pathological interest as showing the intensity of the toxic process. He referred to the frequently rapid and unexpected cardiac failure which might arise in such cases. Laryngeal stenosis seldom led to death, and even if the stenosis were relieved by tracheotomy heart failure often supervened. This it would do in the absence of any very definite physical signs, and before cultures

could be taken, or pathological investigations made. Clinical acumen and experience were of great importance, as the patients not infrequently died before the pathologist could be consulted.

Dr. Lautmann (Paris). L'évolution rapide de l'affection en question est impressionnante. Heureusement la mort n'est pas la terminaison constante de l'affection. Dans un cas observé par l'auteur les injections intra-veineuses d'électrargol combinées à l'emploi de la pommade au collargol ont été suivies de guérison.

Mr. Alex. R. Tweedie (Nottingham) in this connexion made a brief reference to the three similar fatal cases an account of which was published by him in the *Journal of Laryngol., Rhinol., and Otol.* of the current year.

Professor Finder (Berlin) sagte, um die Richtigkeit dessen zu illustrieren, was StClair Thomson über die Nutzlosigkeit der Tracheotomie gesagt hat, dass er selbst sechs Fälle gesehen hat, von denen bei fünf die Tracheotomie gemacht wurde ; alle fünf starben ; der einzige, bei dem zufällig die Tracheotomie nicht gemacht wurde, und der übrigens mit polyvalenter Streptokokkenvaccine behandelt wurde, kam durch.

Dr. Marschik (Wien). Die Tracheotomie muss, so sehr wir von der Nutzlosigkeit derselben *quoad vitam* überzeugt sind, aus humanen Rücksichten, auf Wunsch des Patienten, oder aus andern äusseren Gründen, ausgeführt werden.

Mr. Herbert Tilley (London) agreed as to the dramatic suddenness with which these cases develop. He cited three instances.

Case I. A lady, aged 43, who was suffering from sore throat and slight difficulty in breathing of twenty-four hours' duration. The epiglottis was œdematous, and the pharyngeal and laryngeal mucosæ were extremely red. The epiglottis was scarified with relief, but the patient died next day from septic pneumonia.

Case II. An elderly lady, aged 70, who had suffered from a sore throat for two days. When he arrived at her house he was hurried to her room, but she died suddenly in bed before he had time to get instruments out of his bag. There was an extensive glandular inflammation on the right side of the neck which was deeply cyanosed, hard, and brawny ; the mucosa of the pharynx was in a sloughing condition. It is possible she died of heart failure, secondary to obstruction of respiration, because when he entered the room there was considerable stridor.

Case III. A young lady, aged 28, nearly lost her life from streptococcal inflammation (with œdema) of left tonsil, palate, and pharynx. She recovered after free incisions of the palatal mucous membrane and the tonsil. Autogenous vaccines were also given. Her aged mother, who nursed her, fell ill—the primary infection appearing in the soft palate—and died of general septicæmia in 48 hours. It is probable that the latter infected her soft palate by using a spoon from which her daughter had taken alternate spoonfuls of jelly with herself.

The speaker believed in early free incisions into the infected region and the use of an autogenous sensitized vaccine. He was perhaps old-fashioned in thinking that full doses of the liquor ferri perchloridi had a beneficial effect on the disease.

Reply

Mr. PHILIP DE SANTI (London) stated that he was well satisfied that this subject had come under discussion at the Congress, and that there was unanimity of opinion as to the clinical and pathological identity of these very grave affections. The sooner the general practitioner recognized the great importance and gravity of the cases the better. Heart failure, as pointed out by Sir StClair Thomson, Tilley, aud others, was a very real thing. He, Mr. de Santi, could well remember a relative of his own who was seen by Sir Felix Semon, the patient having apparently got a chill at the 4th of June Eton College boat procession, and who died, despite a very rapidly performed tracheotomy, within twenty-four hours of the first symptoms of illness.

Mr. de Santi's practice was to give, if the condition of the patient permitted, a polyvalent antistreptococcal serum at once; and then to have a sensitized vaccine prepared from the predominant micro-organisms found present, preferably in the blood. In his cases a polyvalent serum or vaccine had not proved dangerous in any way.

In answer to Dr. Goodall, as to how the streptococcus got there, he stated that it was not at all uncommon to find it present in the throats of quite normal people, and given an accidental breach of surface or a pre-existing state of catarrh it was not difficult to see how one of these conditions might develop.

SECTION XV

RHINOLOGY AND LARYNGOLOGY

INDEPENDENT PAPER

DIE INTRANASALE DACRYOCYSTOSTOMIE

Von Dr. L. POLYÁK, Primärarzt des St. Johannes Hospitals in Budapest

Meine Herren! Es ist mir gelungen, eine Operationsmethode auszuarbeiten, welche gestattet, den Tränensack auf intranasalem Wege mit der Nase in eine so breite Kommunikation zu bringen, wie sie früher nur von aussen durch die Toti'sche Dacryocysto-Rhinostomie erzielt wurde. Näheres hierüber, ferner die Geschichte, die Versuche von anderen Autoren, und die Beweise meiner Priorität können Sie in meinem Sammelreferate (*Centralblatt für Laryng.*, September 1912), ferner in den *Verhandlungen des Vereins deutscher Laryngologen* (Stuttgart 1913) und im *Archiv für Laryngologie*, Bd. xxvii, Heft 3, finden; diese Publikationen enthalten auch eine ausführliche Beschreibung meiner Operationstechnik.

Die Operation wird im Vorhofe des mittleren Nasenganges vor und oberhalb der Ansatzstelle der mittleren Muschel gemacht; sie ist nicht leicht, weil die anatomischen Verhältnisse nicht selten Schwierigkeiten bereiten, besonders bei schmalen Nasen mit hohem Rücken; ausserdem ist in ein Drittel der Fälle die Nasenscheidewand nach der erkrankten Seite verbogen (13 mal unter 42 Fällen); recht häufig (18 mal) musste die Ansatzstelle der mittleren Muschel zum Teil abgetragen werden, und 10 mal standen Siebbeinzellen, welche ich prälacrymale Zellen nennen möchte, im Wege, und ist ihre Abtragung notwendig geworden.

Die Technik besteht kurz darin, dass wir in Lokalanästhesie operieren, an der Operationsstelle ein Schleimhautperiostlappen bilden, welches nicht behalten wird, den aufsteigenden Ast des Oberkiefers und das Tränenbein unterhalb dem Ansatze der mittleren Muschel breit aufmeisseln und abhebeln, worauf der knöcherne Duktus und der untere Teil des Tränensackes frei vor uns liegt, wovon wir uns mit Druck mit dem Finger von aussen überzeugen können. Die knöcherne Oeffnung wird nun teils noch mit dem Meissel, teils mit meinen Knochenstanzen oder mit der Fraise erweitert, bis der Sack ganz frei gelegt ist. Nun

spalten wir den obersten Teil des Duktus, worauf gewöhnlich Eiter hervorquillt, und überzeugen uns mit einer rechtwinklig abgebogenen Sonde, ob die knöcherne Wand ganz bis zur Kuppel des Sackes entfernt ist, sollte dies noch nicht der Fall sein, so resecieren wir erst mit dem Messer und feinen Konchotomen die freiliegende membranöse Wand des Sackes, und hierauf mit meinen Knochenstanzen die restierende knöcherne Wand, bis der Sack ganz freiliegt, und seine ganze nasale Wand, bei Ektasien sogar ein Teil der facialen Wand entfernt ist. Es ist besonders dieser letzte Teil der Operation, wenn Ansatzstelle der mittleren Muschel und Siebbeinzellen im Wege stehen, und teilweise reseciert werden müssen.

Die Operation kann auch bilateral in einer Sitzung ausgeführt werden, ich habe sie bis jetzt 4 mal auf diese Weise gemacht. Die Dilatation der Tränenpunkte und Sondierung des Duktus von oben wird wohl nur bei den ersten Operationen zur Orientierung nötig gefühlt, ich verzichte auf dieselbe stets, weil damit die Funktion der Canaliculi beeinträchtigt werden kann. Eine Schlitzung des Canaliculus ist immer überflüssig und eventuell von Nachteil.

Die Operation wird immer klinisch ausgeführt, die Nase wird nicht tamponiert und die Patienten liegen 24 Stunden im Bette. Eine Nachblutung habe ich bis jetzt nicht gehabt. Die Nachbehandlung soll möglichst schonend sein, die intranasale reaktive Schwellung geht in 4–6 Tagen spontan zurück.

Ich habe diese Operation, welche ich die intranasale Dacryocystostomie nenne, bis jetzt in 42 Fällen ausgeführt. Die grösste Zahl der Fälle war eine eitrige Dacryocystitis, u. zw. ohne Dilatation des Sackes 5 Fälle (2 geheilt, 1 geheilt nach Wiederholung der Operation, 1 bedarf wegen Eversion des Tränenpunktes noch der augenärztlichen Hilfe, 1 ungeheilt, da die Oeffnung sich geschlossen hat, — wird wieder operiert); — mit sichtbarer Ektasie des Sackes 12 Fälle, u. zw. einseitig 4, bilateral 2, mit Fistel 1, bilateral mit Fistel 1 Fall, sind sämmtlich geheilt; mit wallnussgrossem ektatischem Tumor 3 Fälle, geheilt; mit nur bei der Operation festgestellten Ektasie des Sackes 7 Fälle (6 geheilt, 1 bedarf noch der augenärztlichen Hilfe); mit kleinem geschrumpften Saccus 10 Fälle; darunter 1 bilateral (geheilt 8, augenärztliche Hilfe bedürfen 2); ferner operierte ich 2 Fälle, wo früher die Extirpation des Sackes gemacht wurde, die Wunde aber nach das Auge zu weiter eiterte, ich habe in diesen Fällen die knöcherne Wand breit geöffnet, und die Wunde ausgekratzt, hierauf heilte mit Tränenableitung 1 Fall, gebessert wurde nach 2 Operationen 1 Fall, insoferne dass das Tränenträufeln sehr gering ist, und ich hoffe durch eine wiederholte Operation auch hier vollen Erfolg zu erzielen.

Ich habe ferner einen schweren Fall von Tuberkulose des Sackes operiert, und vorläufig Vernarbung erzielt, das Tränenträufeln besteht aber, weil ein starkes Ektropium vorhanden ist, welches eine augenärztliche Operation erfordert. Ausserdem hatte ich 2 Fälle von Stilli-

cidium lacrymale operiert (1 Fall geheilt, 1 Fall wegen Eversion des unteren Tränenpunktes in augenärztlicher Behandlung).

Zusammengefasst von 42 Fällen heilten sofort 33, nach wiederholter Operation 1, und mit nachfolgender augenärztlicher Hilfe 1, zusammen 35 Fälle = 83,3 Prozent; bei 3 Fällen = 7,2 Prozent muss ich wegen Verschluss der Oeffnung die Operation wiederholen; und 4 Fälle = 9,5 Prozent bedürfen der augenärztlichen Nachbehandlung.

Wenn ich nun auf Grund meiner bisherigen Erfahrung versuche, die Indikationen der intranasalen Dacryocystostomie zusammenzustellen, dann glaube ich nicht fehlzugehen mit der Behauptung, dass diese Operation indiziert ist in allen jenen Fällen, wo die Augenärzte die Toti'sche Operation oder die Exstirpation des Sackes für angezeigt halten, insoferne es sich um Sekretstauung im Sacke handelt. Ich glaube, dass wir in der Aufstellung der Indikationen nicht weitergehen dürfen, denn es handelt sich um ein Grenzgebiet, welches spezielle augenärztliche Kenntnisse erfordert. Wir dürfen uns nur auf denselben Standpunkt stellen, welchen wir bei der Behandlung der rhinogenen Sehstörungen behaupten, d. h. wir operieren nur die Fälle, welche uns von den Augenärzten zu diesem Zweck überwiesen werden. Deshalb ist West's Argumentierung [1] gegen mich, — weil ich auf die Einführung der Sonde aus guten Gründen verzichte — hinfällig; wir brauchen uns nicht um die Erkrankungen der Tränendrüse, der Lider, der Tränenpunkte und der Canaliculi zu kümmern, das ist die Aufgabe der Ophthalmologen, wir können uns darauf verlassen, dass sie uns nur Fälle überweisen, wo die Ursache der Störungen im Sacke oder in dem Duktus liegt. Es ist ja selbstredend, dass die Eröffnung des Sackes nach der Nase zu nur dann von Nutzen sein kann, wenn das Apparat oberhalb des Sackes richtig funktioniert; wir können zufrieden sein, wenn die Ophthalmologen uns nur alle die Fälle zur Operation überweisen, wo wir besseres leisten können, als die bisherigen Behandlungsmethoden dies zu tun imstande waren, und sollen uns unter keinen Umständen für Augenärzte ausspielen und damit die Methode in Misskredit bringen.

Es ist also nicht Indikationsstellung, sondern nur Aufzählung der Fälle, in welchen ich bis jetzt mit Erfolg operiert habe, der Gegenstand meiner Betrachtungen. Ich glaube das Hauptgebiet unseres Materials wird die eitrige Dacryocystitis liefern, denn diese Krankheit gefährdet zumeist das Auge, und die konservative Behandlung hat bis jetzt wenig geleistet. Die Operation ist imstande, selbst grosse, von aussen sichtbare ektatische Tumoren sofort zum Schwunde zu bringen, aber auch bei kleinen, in Folge von wiederholten Entzündungen geschrumpften Säcken, hat sie sich bewährt. Akute phlegmonöse Entzündungen des Sackes mit oder ohne Fistelbildung heilen prompt nach Herstellung der breiten, nasalen Kommunikation; Stillicidium lacrymale, wenn es durch Veränderungen am Sacke oder im Duktus bedingt ist, eignet sich ebenfalls für diese Behandlung. Auch Tuberkulose des Duktus und des

[1] *Archiv f. Laryng.*, Bd. xxvii, H. 3, S. 514.

Sackes, bis die äussere Haut nicht angegriffen ist, kann intranasal gut operiert werden, und ferner möchte ich darauf hinweisen, dass nach Exstirpation des Sackes, wenn Eiterung gegen das Auge fortbesteht, die intranasale Operation nicht nur Sistierung der Eiterung, sondern falls die Canaliculi intakt sind, eine Tränenableitung gegen die Nase, trotz Fehlen des Sackes ermöglicht.

Die Kontraindikationen sind dieselben wie bei allen intranasalen Operationen : zu niedriges Alter, grosse Schwäche des Organismus und unüberwindliche technische Schwierigkeiten. Nach gewisser Einübung bietet auch die bilaterale Operation keine Schwierigkeiten.

In Betreff meiner früheren Angaben möchte ich zufügen, dass ich jene Ansicht, welche in meiner vorläufigen Mitteilung (*Centralblatt*, 1912) steht und derartig lautet, dass von der geschaffenen Oeffnung aus das Tränenbein und die hintere (nasale) Wand des Sackes sich wegen der Raumverhältnisse nicht in dem Masse resecieren lässt, wie bei der Toti'schen Operation, auf Grund der klinischen Erfahrungen schon längst modifiziert habe. Ich muss dies hier betonen, weil West (*loc. cit.*, S. 512) auf diese Behauptung schon wiederholt zurückkommt, obwohl er Gelegenheit hatte, mich über die ganz breite Eröffnung des Sackes (intranasale Toti) in Stuttgart vortragen zu hören.

Ein auch bei anderen intranasalen Operationen brauchbarer Griff, welchen ich bei meinen Saccusoperationen mit Septumdeviation anwende, besteht darin, dass ich mir Nasenspecula mit einer kurzen und einer langen Schnabel für die rechte und die linke Seite verfertigen liess, und damit das devizierte Septum während der Operation zur Seite drücke. Der Nutzen dieses kleinen Kunstgriffes bestand darin, dass ich unter 13 Fällen von Septumdeviation nur einmal gezwungen war, eine partielle submuköse Resektion des Septums — gleichzeitig mit der Tränensackoperation — vorzunehmen.

Bezüglich der West'schen Technik (*loc. cit.*), welche ich für die breite totale Resektion der nasalen Sackwand für ungeeignet halte, möchte ich bei dieser Gelegenheit keine Diskussion beginnen. Die Kollegen, welche die intranasale Dacryocystostomie versuchen wollen, werden damit sehr schnell ins Reine kommen, welcher von uns richtiger vorgeht. Seine Argumentierung, dass eine durch den Canaliculus horizontal eingeführte Sonde der beste Beweis für die totale Resektion der nasalen Sackwand ist, beruht auf einem Irrtum, denn bekanntlich mündet der vereinte Canaliculus im unteren Drittel oder höchstens in der Mitte des Sackes, folglich kann die Sonde horizontal eingeführt werden und dabei die obere Hälfte der knöchernen Saccuswand unberührt bleiben, wie dies auch auf Fig. 2 seiner zitierten Mitteilung sichtbar ist. Der einzige Beweis für die totale Resektion der nasalen Saccuswand ist jene, wenn wir mit der rechtwinklig abgebogenen Sonde keinen Recessus nach oben mehr abtasten können.

Nun, m. H., noch ein Wort darüber, was wir Heilung nennen dürfen ? Die Operation selbst bietet einen unbehinderten Abfluss des Sackinhaltes

nach der Nase zu, aber ich hatte Gelegenheit gehabt, in einigen Fällen zu sehen, dass trotzdem das Auge trocken war und die Fluoresceinprobe sehr gut ausfiel, bei Druck auf den Saccus sich noch etwas Eiter nicht in das Auge, sondern nach der Nase zu entleerte. Eine funktionelle Heilung aus augenärztlichem Standpunkte war wohl da, sie war aber nicht vollständig, da die Eiterung weiterbestand. Ich glaube dieser Umstand soll in der Zukunft weiter kontrolliert werden, es ist möglich, dass diese Eiterungen mit der Zeit spontan aufhören, aber es ist auch möglich, dass wir gegen sie lokal vorgehen müssen.

Jedenfalls besitzen meine Operationen gegen die Operationen von aussen den grossen Vorteil, dass sie im Notfalle wiederholt, und so das mangelhafte Resultat durch weitere Eingriffe verbessert werden kann.

Zum Schlusse will ich noch die vorläufige Mitteilung machen, dass meine Methode, mit der Anlegung einer entsprechend grossen Oeffnung am Knochen und Resektion der Ansatzstelle der mittleren Muschel, nach meinem Glauben ein geeigneter Weg sein kann, um Orbitalabscesse, welche zwischen Bulbus und Nase hegen, intranasal, ohne eine äussere Wunde anzulegen, zu eröffnen und nach die Nase zu drainieren; ferner ist der Weg wohl auch zur intranasalen Resektion der Ethmoidalnerven zu gebrauchen. Es bleibt weiteren Untersuchungen vorbehalten, diese Fragen endgültig zu entscheiden.

DISCUSSION

Dr. Halle (Berlin). Ich glaube, wir haben alle Grund, Herrn Polyák und Herrn West, die beide gleichzeitig auf diesem Gebiete gearbeitet haben, dankbar zu sein für die Erweiterung unseres Wirkungskreises. Wir sind in der Tat jetzt imstande, die physiologischen Bedingungen bei Erkrankungen des Ductus oder Saccus lacrymalis wieder zu annähernd normalen zu gestalten, während früher die Exstirpation des Saccus zur Verstümmelung und zu dauernden Störungen führte. Ich habe bisher 31 Fälle von Dacryocystostomie operiert, 4 davon doppelseitig. Der älteste war 68 Jahre, der jüngste 2 Jahre 1 Monat alt. Hier habe ich die Operation nur unter Assistenz des Ophthalmologen ausgeführt, damit dieser bei dem möglichen Missglücken der Eingriffe angesichts der Kleinheit der Verhältnisse von aussen operieren könnte. Aber es ging alles gut. Während ich früher am Saccus eine Lappenbildung vornahm, habe ich das jetzt aufgegeben, da es mir überflüssig erscheint. Dagegen halte ich die Bildung des Schleimhautperiostlappens von grosser Wichtigkeit. Diesen gibt Polyák nicht korrekt wieder. West hat ihn ebenfalls früher nicht ausgeführt, später ist er meiner Anregung gefolgt, macht aber den Lappen etwas anders. Prinzipiell handelt es sich darum, dass man beim Durchmeisseln der lateralen Nasenwand von unten nicht unnütz wichtige Schleimhaut opfert. Der Schnitt wird angelegt intranasal soweit nach vorn wie möglich, erstreckt sich unten bis zum oberen

Rande der mittleren Muschel, oben so hoch wie möglich. Er geht bis auf den Knochen. Vorher wird vorn an dem Ansatz der mittleren Muschel ein kreisförmiges Stück aus der Schleimhaut ausgeschnitten, etwa entsprechend der Ausdehnung des Sackes, und völlig entfernt. Der grosse Lappen wird nun vom Knochen abgehobelt, nach hinten geschlagen, und nun der Knochen abgemeisselt, der Sack freigelegt, und die mediale Hälfte entfernt. Darauf wird der Schleimhautperiostlappen zurückgeklappt, die Schleimhaut der lateralen Wand legt sich in den kreisförmigen Defekt des Lappens, dieser wird locker antamponiert, und in zwei bis drei Tagen ist alles geheilt.

Die Vorteile dieses Vorgehens sind folgende: 1. Es wird keine Schleimhaut mehr als nötig geopfert. 2. Das Operationsfeld ist viel übersichtlicher. 3. Nach Anlegen des Lappens ist die ganze Wunde völlig gedeckt mit Ausnahme der Oeffnung für den Sack. 4. Dadurch wird die Bildung von Wundsekret, Granulationen, Narbenzeigen verhindert und die Heilung gesichert. Der etwa vorhandene geringe Defekt zwischen dem kreisrunden Defekt und dem Sackrand überheilt sehr schnell ohne Narbenbildung. Ich habe keinen Misserfolg gesehen.

Dr. Dan McKenzie (London) had had experience of the intranasal operation with a flap formation of the same type as that described by Halle, but smaller. He had found no difficulty in performing the operation, the tear-duct, sac, &c., having been opened without any trouble. In one case of lachrymal cyst, which fortunately had not been slit up, the operation had been a striking success. In opening the bone he had not required to use the gouge or chisel.

Mr. Douglas Harmer (London) said that he had operated upon five cases by Yankauer's method, but that the results had not been altogether satisfactory. In one case in which epiphora had been present for ten years a constriction was found at the upper end of the bony canal.

Reply

Dr. Polyák (Budapest). Ich muss Herrn Halle um Entschuldigung bitten, dass ich seine Lappenbildung nur kurz erwähnt habe, ich habe aber überhaupt nur sehr verkürzt in Stichwörtern über Technik gesprochen. Uebrigens ist die Art, wie er jetzt seinen Lappen macht, mit dem Fenster, ganz neu, früher hat er anders operiert. Ich will seine Modifikation gerne versuchen, sie scheint mir begründet und gut, aber ich hatte die Konservierung des Lappens bisher nicht nötig, weil mein entfernter Lappen viel kleiner (1 Quadratzentimeter) ist und dabei kann es nicht viel granulieren. Meine Fälle heilen ebenfalls sehr schön und schnell. Seine Methode, die nasale Sackwand mit Messer abzutragen, ist gut, besonders für Fälle mit Ektasie, es gibt aber auch sehr kleine, höchstens pfefferkorngrosse, geschrumpfte Säcke mit dicken Wandungen,

da ist wenig mit der Pincette zu fassen und mit dem Messer zu schneiden. Für diese aber, überhaupt für alle Fälle, ist meine Gegendruckmethode viel besser und sicherer, man kann dabei mit dem feinen Konchotom viel mehr abtragen als mit dem Messer. Dies ist besonders bei grossen ektatischen Säcken wichtig, wo man nicht nur die ganze nasale Wand, sondern auch grössere Teile der facialen Wand abtragen soll.

Mr. Douglas Harmer will ich bemerken, dass er seine Fälle nach der alten Methode von Passow oder Strazza operiert hat, dabei wird die knöcherne Sackwand nur wenig und nur im unteren Teile des Sackes eröffnet ; kein Wunder, dass diese Wunde schnell zu granulieren anfängt und die Oeffnung sich wieder schliesst. Ich rate, meine Methode der breiten Resektion à la Toti intranasal zu machen, dann wird auch das Resultat anders ausfallen.

[Monday Afternoon, August 11]

SECTION XV

RHINOLOGY AND LARYNGOLOGY

INDEPENDENT PAPER

CHRONIC HYPERPLASIA OF THE SUPERIOR MAXILLA

By FREDERIC H. WESTMACOTT, F.R.C.S. (Eng.), B.Sc. (Vict.), &c., Honorary Assistant Aural Surgeon, Manchester Royal Infirmary; Honorary Surgeon, Throat and Ear Department, Manchester Children's Hospital; Honorary Consulting Surgeon, St. John's Hospital for Diseases of the Ear, Manchester.

Unilateral enlargement of the upper jaw is not infrequently met with as a result of inflammatory changes or neoplasm, but there is a special type of enlargement to which I drew attention a few years ago,[1] and of which I have now seen eight cases. The record of these are of interest for several reasons, not the least of which is the careful histological examination which was made of the bone in four of the cases by my friend, Professor I. Walker Hall, of Bristol University.

Four of the recorded cases were under the care of my colleague, Mr. F. A. Southam, consulting surgeon to the Manchester Royal Infirmary, to whom I am indebted for placing his records and materials at my disposal.

My attention was first drawn to the benign character of the swelling of the alveolar process in 1897, when a relative of my own, a young lady of 17, was thought to be suffering from sarcoma of the left superior maxilla. As there were carious roots in the jaw, I suggested to Mr. Southam, whom she had consulted, that it might be as well to extract the teeth and explore the jaw first of all. This was done, and the bone found to be soft and vascular. A considerable portion was gouged away, and after a rapid recovery there has been no recurrence. But it was not till 1905 that the true character of this condition was discovered.

Case 2. In that year a woman, 19 years of age, came under my observation at the Manchester Dental Hospital for a swelling in the left upper jaw, and gave a history of pain and discomfort in the teeth of that side—several of which had been extracted without relief. The appearance in the mouth is shown in Fig. 1. It presented a smooth

[1] *Dreschfeld Memorial Volume*, Manchester, 1908.

swelling of both walls of the alveolus, more especially of the external wall, and was prominent in the canine fossa. It was hard and smooth on palpation, and limited by the canine tooth in front, the tuberosity of the maxilla behind, and the malar process externally. The mucous membrane appeared healthy. The orbital and nasal cavities were not encroached upon. There was also a dark shadow on the left side on transillumination.

Under a general anæsthetic an incision was made along the alveolar border, and the mucous membrane reflected inwards and outwards. A very thin, somewhat porous layer of compact bone was found, and, under this, a very vascular cancellous type of bone, which when cut with a gouge resembled the consistence of a raw potato. The whole antral cavity appeared to be filled with this material, which was removed with the gouge and bone nibblers, as far as it could be traced. As the tuberosity was not much enlarged, and appeared to be healthy, it was left. The cavity was packed with gauze till a denture could be made to cover the aperture. This was replaced by two others as shrinkage



Fig. 1. Model of upper jaw, showing swelling of alveolus with smooth surface. Case 2.

took place, and in eight weeks the cavity had healed. The patient returned almost three years after operation, with difficulty in closing the teeth, owing to swelling of the tuberosity; this caused some tenderness, but she had no return of the neuralgia. The other parts of the maxilla remained unaltered. The tuberosity was removed by an oblique section, and the bone found to be of the same cancellous condition as the other parts; the wound healed in three weeks. For three years afterwards there had been no recurrence. The histological examination was as follows:

'The tissue consists of well-formed bony tissue. The laminæ of bone are already laid down, and the bone corpuscles stain well. The interstices of the bone are occupied by a thick layer of actively proliferating osteoblasts, amongst which are a few osteoclasts. In addition, there is a rich vascular supply, and an abundance of connective tissue cells; these cells possess an unusually large amount of protoplasm. There are no collections of small cells or polynuclear leucocytes, nor are there any signs of granulomatous inflammation. The condition appears to be one of simple hyperplasia.'

I am able to record eight such cases altogether, with pathological investigation of the bone in four—seven occurred in women and one in

a man, six were on the left side, and two on the right ; in age they ranged from 17 to 30.

Case 3. A man, aged 27 years, presented a swelling of the left alveolar border, with three carious roots, which had existed with constant ' toothache ' in the bone between eighteen months and two years. The roots were extracted, but the pain and swelling remained ; five months later it was found that the width of the alveolus had not increased, yet the canine fossa was obliterated by the swelling. On removing the cancellous bone with a gouge, the almost obliterated antral cavity was found above the invading mass, which was smooth on the convex surface. The cavity healed in two months, and two and a half years later there had been no recurrence.

Case 4. A woman, aged 25 years, who had noticed a swelling of the left upper jaw for some months, which was thought to be due to a carious bicuspid tooth. On removal of the tooth the socket was found

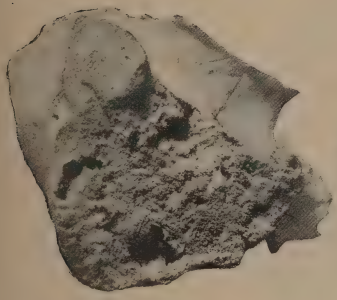


Fig. 2. Superior surface of maxilla excised, showing the antral cavity filled with cancellous bone. Case 5.

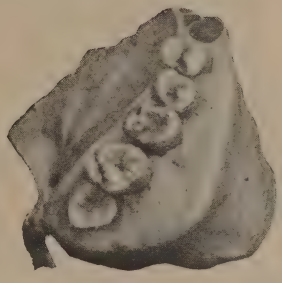


Fig. 3. Alveolar surface of excised maxilla, showing broad alveolus and porous compact bone. Case 5.



Fig. 4. Normal alveolus for comparison.

to be surrounded by spongy bone, vascular, and easily penetrated by a steel probe in all directions. The condition of the swelling remained the same for about six months, after which I did not see her again.

Case 5. A woman of 19 years received a blow on the left cheek about nine years before she was admitted to the Manchester Royal Infirmary, under the care of Mr. F. A. Southam, in February 1904. The swelling began to increase in size in February 1902, but for some weeks before her admission it progressed more rapidly. This was accompanied by severe neuralgic pain in the side of her face, for which she sought relief as well as the advancing disfigurement. The swelling was hard and solid, and transillumination revealed an opaque shadow. Mr. Southam excised the maxilla, leaving the orbital plate. The recovery was quite satisfactory, and there was no recurrence. The illustrations shown in Figs. 2 and 3 represent the upper and alveolar surfaces of the maxilla removed in this case ; and Fig. 4 shows a normal alveolus for comparison. The specimen shows the spongy nature of the bone and the porosity of the external layer of dense bone.

Case 6. A woman, aged 25 years. The right upper jaw had increased in size for three years, slow in progress, and without pain or inconvenience. All the teeth of the affected jaw were absent. Both walls of the alveolus

and the canine fossa were enlarged, and caused a prominence of the cheek, for which deformity she sought advice. The lower half of the upper jaw was removed, with good result, but the subsequent history is unknown. The bone presented an appearance resembling that in the other cases.

Case 7. A woman, aged 30, had a similar affection of the left maxilla to the previous cases, with a history of steady enlargement for four years, and with occasional neuralgic pain. The chief inconvenience was the disfigurement. The three carious roots of the twelfth year molar were present. Fig. 5 illustrates the model of the upper jaw obtained from this patient. The pathological report is as follows :—

'The sections show spongy bone containing well-staining bone corpuscles. The portions of the bone are lined by layers of active osteoblasts, and here and there are massive collections of these cells. A few osteoclasts only are present. There appears to be but little absorption

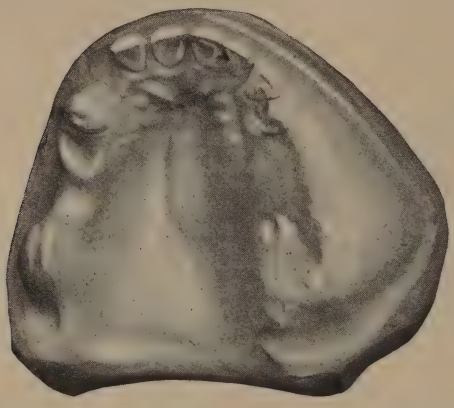


Fig. 5. Broad smooth alveolus with three carious roots, and bulging in canine fossa. Case 7.

of the newly-formed bone. The interstitial tissue is composed of connective tissue cells, among which are a number of plasma cells and some fibroblasts. The vessels are adult in type, but in some places there is considerable perivascular small-celled infiltration. Hæmorrhages are not a feature. This is all that appeared in the first lot of sections examined, but on cutting further material, areas of cell collections were found which showed little or no relation to blood-vessels. The cells themselves had large nuclei, with a small amount of protoplasm, and were of a sarcomatous type. Although the tissue seems, therefore, to be mainly hyperplastic, it must be considered as manifesting a tendency to sarcomatosis. In neither this nor in previous specimens are there any signs of suppurative or granulomatous reactions.'

The operation by gouging took place in February 1908, and the patient has been under observation ever since, and has not had any recurrence.

Case 8. A woman, aged 20 years, had suffered for three years from increasing protrusion of the right cheek and occasional 'toothache' in the whole of the bone. There were three or four carious roots in the right alveolus, which was much wider than normal. The canine fossa protruded up to the inferior orbital ridge, and outwards to the malar

process. On November 3, 1908, I removed the teeth and gouged away the whole alveolus and tumour as high as the orbital plate, and outwards to the malar-maxillary suture, together with the tuberosity. The mass extended forwards to the canine ridge, the bone being cancellous and very vascular. As in previous cases, a denture was made to fit the opening during healing, the mouth being flushed three or four times a day with a phenate of soda solution. The cavity was packed with gauze soaked in liquor hamamelidis B.P. for two days, and the bleeding gave no trouble after operation. The microscopic examination reveals well-formed osseous tissue with well-stained corpuscles. Around all the pieces of spongy bone there are layers of osteoblasts, which are proliferating. A marked feature, however, is the vigorous destruction of the spongy bone which is going on. The osteoclasts are well formed, and the areas of erosion are numerous and extensive. The interstitial tissue is richly vascular, and contains much connective tissue. Fibrous tissue is also being laid down to a considerable extent. There are not any signs of granulomatous areas or of the small-celled collections seen in some of the other preparations. Evidences of malignancy are not demonstrable. The condition appears to be one of simple hyperplasia, and the absorption of spongy bone which precedes the formation of hard bone is here well exemplified.

I have not been able to find any references to this bone affection, and from a study of these cases it appears that the process is one in which there is a reversion to the embryonic type which is present before the formation of the maxillary antrum in the original maxillary process, the remains of which are found only, in the adult, in the alveolus and malar process. It is limited in front by the premaxilla, and the new growth does not extend beyond this limit. It is evident that the alveolus is first attacked and then the outer wall of the maxilla towards the malar process, involving that part and producing the characteristic deformity. The mucous membrane not being implicated, the antral lining prevents the spreading to the internal and superior walls of the maxillary sinus, and explains the reason of the freedom of the nasal and orbital cavities from invasion, although the invasion of the sinus is early and complete. The mucous membrane covering the alveolus and outer maxillary wall strips off quite easily, leaving a vascular, porous bone exposed to view. The necessity of complete removal of the proliferating bone is exemplified by further growth in Case 2. The absence of other complications and recurrence justify the conservative method of operation in preference to a formal removal of the maxilla. No doubt many cases of excision of the upper jaw, with no recurrence of growth, have given rise to the belief that a sarcomatous condition was present, and had been cured. The naked-eye appearance is similar in many respects to that disease, and the histological appearances recorded in Case 7 give rise to the impression that a condition precedent to sarcoma may be a physiological reversal of type of bone, differing only in rate of growth ; possibly malignancy might follow continued irritation. The main feature is the large number of osteoblasts which in some cases is accompanied by a larger or smaller number of osteoclasts, in a natural

attempt to arrest the process by formation of compact bone, and may account for the rate at which the enlargement of the mass progresses. The bone is truly infantile in type, and the absence of any inflammatory or suppurative changes, in the true sense of the word, go to show that it is not a septic infection, although such a state must exist around the carious roots found in most of the cases, and may be the origin of the irritation which apparently causes the changes in the bone. In Case 5 the teeth were in good condition, but the blow some years before may have set up a passive hyperæmia around the erupting wisdom-tooth.

The diagnosis of this complaint rests upon the long history, the minor character of the pain, and the resultant deformity, together with the normal condition of mucous membrane and absence of inflammatory phenomena, especially tenderness on pressure. A dentigerous cyst would be shown by a radiogram, and would give an elastic or egg-shell crackling on palpation, with clear fluid or pus on exploring with a needle. Sarcoma would be much more rapid in growth, give rise to irregularity of outline, and the pain would be more severe. It is distinguished from leontiasis ossea by its unilateral involvement and limitation, and absence of nodular protuberances.

Lantern Slides

1. *Low power.* Hyperplastic area. Spongy bone lined by active osteoblasts, few osteoclasts only. Massive collections. Interstitial tissue of connective tissue cells. Plasma cells and fibroblasts.

2. *Low power.* Perivascular small-celled infiltration. Vessels of adult type. No hæmorrhages.

3. Areas of cell collections with no relation to blood-vessels. Cells with large nuclei and small amount of protoplasm—sarcomatous in type.

4. Same area high power.

SECTION XV

RHINOLOGY AND LARYNGOLOGY

INDEPENDENT PAPER

CONTRIBUTION A L'ÉTUDE DES RAPPORTS BACTÉRIOLOGIQUES ET HISTOLOGIQUES ENTRE LES AFFECTIONS NASALES ET LES AFFECTIONS DES VOIES LACRYMALES

Par M. le Dr. L. DEWATRIPONT, de Bruxelles

Monsieur le Professeur Kuhnt, de Bonn, déclare, dans son rapport au premier Congrès International de Laryngologie et de Rhinologie tenu à Vienne du 22 au 26 avril 1908, ' Relations entre les maladies du nez et du naso-pharynx et celles des yeux,' que dans 95,7 pour cent des cas, d'après ses statistiques, ce sont des affections nasales qui sont causes des affections des voies lacrymales. Cet énorme pourcentage cadre du reste avec les conclusions des recherches déjà faites antérieurement par maints spécialistes en renom, tant ophtalmologistes que rhinologistes, relativement aux relations qui existent entre les affections nasales et celles des voies lacrymales. Mais c'est surtout au point de vue *clinique* que la plupart des auteurs se sont placés pour établir ces relations. Or, nous avons voulu, quant à nous, les étudier particulièrement aux deux points de vue *bactériologique* et *histologique*.

Et nous avons obtenu ce résultat qu'alors que cliniquement les auteurs considèrent les divers états inflammatoires des voies lacrymales (nous laissons de côté leurs traumatismes et leurs néoplasmes) comme les modalités d'une même affection se manifestant, sous des formes plus ou moins graves, depuis les simples dacryocystites chroniques jusqu'aux péricystites phlegmoneuses, nous avons pu, grâce à nos recherches bactériologiques et histologiques, les différencier complètement en leur attribuant des origines microbiennes bien distinctes et des caractères histo-pathologiques bien déterminés.

D'autres auteurs nous ont certes montré le chemin pour ces recherches, mais nous avons surtout tenu à faire une étude systématique et à procéder avec méthode. Alors, chaque fois qu'un cas d'affection des voies lacrymales nous a été présenté, *nous avons étudié au point de vue bactériologique : d'abord* le pus ou le muco-pus prélevé à l'anse de platine stérilisée à sa sortie du canal lacrymal inférieur sous une légère pression, ou obtenu par ponction aseptique dans les cas de péricystite phlegmoneuse non

fistulisée, ou d'ectasie enkystée du sac ; *ensuite* le pus ou le muco-pus trouvé dans l'angle externe de l'œil ; *enfin* celui trouvé dans le méat inférieur.

Au point de vue histologique, chaque fois qu'un sac lacrymal a dû être extirpé, ou chaque fois qu'il a été possible de prélever des portions de tissu, par exemple après des ouvertures de phlegmons péricystiques, nous en avons pratiqué des examens au microscope.

Ainsi nous avons pu constituer des *catégories* parmi les affections inflammatoires des voies lacrymales.

Nous ne ferons pas d'historique d'ensemble, mais nous signalerons à propos de chacune de ces ' catégories ' les travaux des auteurs, principalement dans le domaine de la bactériologie et de l'histologie pathologique.

Catégorie Ire

Quatre cas de péricystite phlegmoneuse à streptocoques, d'origine nasale, chez des adultes

Cas 1er. — Emma D., vingt-sept ans. Depuis quatre ans, dacryocystite chronique bilatérale. Depuis quatre jours, gonflement et rougeur de la région du sac lacrymal gauche. Douleurs. Élévation thermique et frissons.

Le 22 février. — A gauche, tuméfaction considérable. Peau luisante, tendue, violacée. Fistule par laquelle s'écoule du pus jaune, paupières œdématiées. *A droite,* dacryocystite chronique avec ectasie légère du sac ; cependant, les injections passent. Rhinoscopie antérieure ; rhinite purulente hypertrophique. Rhinoscopie postérieure : queues des cornets inférieurs volumineuses, surtout à gauche.

Examen bactériologique direct : 1° *à gauche,* du pus recueilli par la fistule ; streptocoques ; 2° *à droite,* du muco-pus s'écoulant par le canalicule lacrymal inférieur ; pneumocoques purs, souvent intraleucocytaires ; 3° du pus nasal, streptocoques prédominants et gros diplocoques encapsulés.

Le 23 février. — Examen de cultures de vingt-quatre heures : à gauche, sur bouillon glucosé : dépôt floconneux abondant, avec trouble léger de la masse du liquide ; cocci en longues chaînes et disposés par deux. *A droite,* sur agar : nombreuses petites colonies transparentes de pneumocoques, qui sont repiquées dans bouillon peptonisé.

Inoculations. — A gauche : à un rat blanc sous la peau du ventre (trente-six heures après, mort de septicémie streptococcique). *A droite* : à une souris blanche, sous la peau du ventre (trente heures après, mort de septicémie pneumococcique).

Le 24 février. — Sous chloroforme, incision au bistouri du phlegmon péricystique. Curetage. Thermocautérisation profonde. Tamponnement à la gaze iodoformée. Pansement sec.

Le 25 février. — Bon aspect de la plaie, qui est profonde, mais commence à bourgeonner.

Le 28 février. — Cicatrisation en bonne voie.

Le 20 mars. — Guérison.

Le 27 mars. — Sous chloroforme, extirpation du sac lacrymal droit. Mèche de gaze iodoformée pour le drainage. Suture endermique, deux plombs aux extrémités du fil de soie. Pansement sec compressif.

Le 6 avril. — Guérison.

Le 15 avril. — Belle cicatrice des deux côtés.

Cas 2. — Louis Pr..., cinquante ans.

Le 22 janvier. — Depuis six à sept mois, larmoiement et écoulement de muco-pus par les points lacrymaux. *Actuellement,* dacryocystite bilatérale chronique. L'injection passe à gauche, ne passe pas à droite.

Rhinoscopie antérieure : rhinite hypertrophique. A droite, cornet moyen en contact avec grosse crête du septum. Catarrhe chronique des trompes. *Examen bactériologique direct* du muco-pus des sacs lacrymaux : pneumocoques encapsulés purs.

Le 23 janvier. — Cultures sur agar et dans bouillon peptonisé à ½ pour cent. Cultures pures de pneumocoques sans capsules.

Le 5 février. — Opération nasale, sous cocaïnisation, et après tamponnement d'adrénaline à 1 pour cent ablation au rabot de Carmalt Jones modifié de la crête osseuse du septum à droite. Pansement aseptique à la ferropyrine. Soins consécutifs.

Le 2 mars. — Poussée de péricystite phlegmoneuse gauche. Région du sac rouge tuméfiée, douloureuse à la pression. En même temps, conjonctivite aiguë. Élévation thermique (38, 6°). Insomnie.

Le 3 mars. — Examen d'une culture de vingt-quatre heures, dans bouillon, de pus ponctionné la veille. Dépôt floconneux. Liquide légèrement troublé. Streptocoques en longues chaînes.

Traitement comme pour le cas 1er.

Cas 3. — Rosalie T..., quinze ans. *Le 27 avril.* — Depuis plusieurs années, dacryocystite chronique gauche. Il y a cinq jours, poussée inflammatoire. *Actuellement,* symptômes cliniques d'une péricystite phlegmoneuse non fistulisée. Recherche des adénopathies : à gauche, un ganglion préauriculaire très net, dur, de la grosseur d'une petite noisette, et quelques ganglions rétro-maxillaires.

Rhinoscopie antérieure : rhinite purulente.

Ponction aseptique de pus phlegmoneux et inoculation sous-cutanée à un rat blanc, à onze heures du matin.

Le 28 avril à une heure du soir, vingt-six heures donc après l'inoculation, mort du rat blanc de septicémie aiguë streptococcique.

Le 29 avril. — Examen d'une culture de quarante-huit heures de pus nasal dans bouillon glucosé. Au fond du tube, flocons abondants.

L'ensemble du liquide est à peine troublé. Streptocoques en chaînes extrêmement longues. Cocci rarement disposés par deux.

Traitement comme pour le cas 1er.

Cas 4. — Adolphe de G., quatorze ans. *Le 1er mai.* — Depuis deux ans, larmoiement à droite avec pus dans l'angle interne de l'œil et paupières collées le matin. Depuis quinze jours, poussée inflammatoire, sans

cause déterminée. *Actuellement*, péricystite phlegmoneuse droite non fistulisée avec adénites concomitantes ; petits ganglions durs sous- et rétro-maxillaires ; pas de ganglion préauriculaire appréciable.

Rhinoscopie antérieure : rhinite purulente.

Pus ponctionné. Frottis sur lames. Ensemencement d'un tube d'agar et de deux tubes de bouillon glucosé. Inoculation à un rat blanc, sous la peau du ventre (à dix heures du matin).

Le 2 mai. — A quatre heures du soir, trente heures après l'inoculation, mort du rat blanc de septicémie aiguë streptococcique.

Les recherches diverses bactériologiques avaient du reste démontré du streptocoque absolument pur dans le pus phlegmoneux.

Traitement comme pour le cas 1er.

Ces quatre cas de péricystite phlegmoneuse survenue chez des adultes au cours d'une dacryocystite chronique nous ont fourni d'intéressantes notes bactériologiques.

En effet, par l'examen direct du pus, par les cultures et par les inoculations sous-cutanées au rat blanc, nous avons mis en évidence formelle dans ces quatre cas l'infection streptococcique.

Mais ce qui a surtout retenu notre attention, c'est le fait qu'alors qu'au cours de la dacryocystite chronique précédent la poussée phlegmoneuse les microorganismes en cause sont des pneumocoques purs, sitôt cette poussée phlegmoneuse déclarée, les pneumocoques cèdent le pas aux streptocoques, ou plutôt sont déplacés par eux.

Or, sous quelle influence l'infection streptococcique apparaît-elle ?

Au cours d'une dacryocystite chronique à pneumocoques, voïlà que tout à coup le caractère torpide de l'affection disparaît pour faire place à tous les symptômes graves, locaux et généraux, d'un phlegmon à streptocoques très virulents. Comment cela se produit-il ?

Naturellement il faut en tout premier lieu que des streptocoques arrivent dans le sac. Or ils ne peuvent suivre que deux routes, les canalicules lacrymaux (voie descendante) ou le canal nasal (voie ascendante).

Si nous nous souvenons qu'à l'état normal il n'y a pas de streptocoques à la surface de la conjonctive ni sur les bords palpébraux (Guénot, *Bactériologie et parasitologie clinique des paupières*, thèse de Paris, 1894 ; Bach, ' L'effet des larmes sur les germes de la conjonctive,' *Congrès de Nuremberg*, septembre 1893 ; Bernheim, ' Action bactéricide des larmes,' *Beitr. f. Augenheilk.*, fasc. viii, janvier 1893), tandis qu'il en existe une quantité dans le nez ; si nous considérons d'autre part qu'à l'état pathologique l'infection streptococcique des conjonctives, quand elle survient, est toujours consécutive à l'apparition du phlegmon péricystique, nous pourrons prévoir, à priori, que c'est par voie ascendante que survient l'envahissement du sac lacrymal par les streptocoques.

Il est toutefois possible par les canalicules lacrymaux, à la suite de manœuvres de cathétérisme pratiquées avec des sondes mal stérilisées ou après des injections faites par des embouts de seringues d'anel septiques.

Mais c'est surtout au cours d'une poussée de rhinite purulente que se fera, si, bien entendu, la perméabilité du canal nasal existe encore, l'infection streptococcique du sac.

C'est réellement ce qui s'est produit dans les 4 cas que nous avons relatés.

Voilà donc les streptocoques au niveau de la muqueuse du sac. Ils s'infiltrent ensuite jusque dans le tissu sous-muqueux, et leur pullulation donne naissance à un processus réactionnel violent. Les leucocytes organisent la défense et englobent d'abord les pneumocoques anciens à résistance moindre, d'où la progressive diminution de ceux-ci par rapport aux streptocoques envahisseurs. L'aboutissant de toute cette lutte est la péricystite phlegmoneuse à streptocoques purs.

La virulence du pus est alors considérable. Si on ponctionne aseptiquement le phlegmon et si on inocule un peu de pus sous la peau du ventre d'un rat blanc, celui-ci meurt de septicémie streptococcique en vingt-quatre à quarante-huit heures ; si c'est dans le derme de l'oreille d'un lapin, il s'y développe une vive inflammation érysipélateuse ; si on fait une inoculation intra-veineuse (dans la veine marginale de l'oreille) à un lapin, celui-ci meurt très rapidement de septicémie streptococcique ; enfin si on fait une piqûre dans la chambre antérieure de l'œil d'un lapin il survient une prompte panophtalmie.

Il nous reste à faire ressortir les différences profondes qui existent, au point de vue de leur pathogénie, entre les péricystites phlegmoneuses tuberculeuses des enfants et les péricystites phlegmoneuses à streptocoques des adultes. Dans les premières, les bacilles de Koch partent des os de la gouttière lacrymale pour envahir ensuite progressivement le tissu sous-muqueux, puis la muqueuse du sac ; dans les secondes, les streptocoques partis du nez remontent le canal nasal, arrivent dans le sac, et gagnent sa muqueuse, puis sa sous-muqueuse, puis le périoste.

Pour concrétiser notre pensée nous dirons, nous fixant en esprit comme point de repère le centre du sac lacrymal, que dans les premières l'infection, tuberculeuse, est centripète, tandis que dans les secondes l'infection, streptococcique, est centrifuge.

Poussées successives de péricystite phlegmoneuse à streptocoques dans un cas de sinusite maxillaire chronique purulente

Observation. — D..., Julien, soixante-cinq ans. *Le* 1er *mai.* — Le malade présente depuis plusieurs années de l'écoulement intermittent de pus jaunâtre par la narine gauche.

A la rhinoscopie antérieure nous trouvons de la rhinite hypertrophique avec tête des cornets inférieurs hypertrophiée. Dans le méat moyen, à gauche, du pus se renouvelle constamment après écouvillonnages à l'aide de tampons d'ouate, sitôt que le malade penche la tête en avant.

N.B. — Il n'y a donc pas oblitération complète de l'ostium maxillaire, donc pas d'empyème à proprement parler.

A la rhinoscopie postérieure, nous voyons les queues des cornets inférieurs fortement hypertrophiées.

La transillumination des sinus par la méthode de Heryng donne à gauche un éclairage beaucoup moins intense. De plus, le signe de Davidsohn existe ; la pupille gauche est obscure, tandis que la droite est nettement lumineuse.

Diagnostic. — Sinusite maxillaire chronique purulente gauche certainement consécutive (attendu qu'il n'y a pas de carie des molaires supérieures gauches) à une rhinite purulente chronique hypertrophique.

D'autre part, depuis environ deux ans, le malade est atteint, bilatéralement, mais surtout à gauche, d'écoulement de muco-pus par les points lacrymaux avec larmoiement intense. De plus, l'an dernier, au commencement d'août, il s'est produit du gonflement avec rougeur de la région du sac lacrymal droit. Et le 13 août, le malade est entré à l'hôpital dans un service d'ophtalmologie, avec, à droite, tous les symptômes d'une péricystite phlegmoneuse fistulisée. Nous avons pu prendre connaissance de sa feuille d'observations, laquelle renseignait qu'un examen bactériologique du pus du phlegmon péricystique droit avait décelé la présence des streptocoques. On n'a pas dû intervenir et après de simples pansements humides une amélioration graduelle s'est manifestée et le malade est sorti le 22 août.

Quelque temps après avoir quitté l'hôpital, il a subi une poussée de péricystite phlegmoneuse gauche et s'est soigné à domicile au moyen de pansements humides boriqués.

Actuellement, le malade présente une nouvelle poussée de péricystite phlegmoneuse gauche. Nous faisons une ponction aseptique.

Examen bactériologique du pus : streptocoques, cultures sur bouillon glucosé.

Le 2 mai. — Dépôt floconneux très abondant, le liquide lui-même restant clair ; streptocoques en longues chaînes.

Traitement. — Incision, curetage thermocautérisation, tamponnement serré à la gaze iodoformée, pansement sec.

Le 21 mai. — Guérison.

Le 27 mai. — Trocart de Krause enfoncé dans le sinus maxillaire gauche, à la partie postérieure du méat inférieur, fortement en arrière de l'orifice du canal nasal. Lavage du sinus. Il s'écoule du pus épais en quantité considérable. Lavages ultérieurs. Mais comme ils ne se font pas très bien, proposition de cure radicale. Le malade n'est plus revenu.

Cette observation de Julien D. aurait pu figurer à côté des quatre observations que nous avons précédemment analysées se rapportant aux péricystites phlegmoneuses à streptocoques des adultes d'origine nasale ; mais nous l'avons prise à part en raison de la sinusite maxillaire chronique purulente gauche concomitante.

Celle-ci est survenue au cours d'une rhinite purulente chronique. Du moins tout porte à le croire, attendu que le malade ne présentait

au niveau des molaires supérieures gauches aucune carie qui fût de nature à provoquer une périostite alvéolaire d'abord, puis une sinusite maxillaire. Mais en même temps que le processus d'hypertrophie envahissait la muqueuse du sinus maxillaire par l'ostium, il entreprenait la muqueuse du canal nasal, puis du sac lacrymal; d'où une dacryocystite purulente, et, ultérieurement, des poussées successives de péricystites phlegmoneuses, d'abord à droite, puis à gauche, puis encore à gauche.

Nous ne dirons pas que ces poussées de péricystite phlegmoneuse constituent à proprement parler des complications de la sinusite maxillaire purulente gauche, puisque précisément la première de ces poussées s'est produite à droite. Elles proviennent plutôt, comme la sinusite maxillaire elle-même, de la rhinite purulente hypertrophique. Il n'y a vraiment complication de la sinusite maxillaire du côté des voies lacrymales que quand l'infection se fait par propagation directe à travers la mince lamelle osseuse qui sépare la cavité du canal nasal (destruction de la partie spongieuse de l'os, puis du périoste du canal nasal, par un mécanisme de thromboses septiques intra-osseuses), ce qui, prétend Hajek, est beaucoup plus fréquent qu'on se l'imagine, surtout quand la sinusite maxillaire s'est organisée, par obturation complète de l'ostium, en empyème proprement dit.

Au point de vue bactériologique, l'identité des agents infectieux dans les péricystites phlegmoneuses et dans les sinusites maxillaires d'origine nasale ne laisse pas de doute sur l'origine commune de ces affections.

En effet, nous avons trouvé dans nos péricystites phlegmoneuses des quatre observations précédentes et de celle-ci des streptocoques, purs ou prédominants, avec des pneumocoques. Or les dernières recherches bactériologiques de Stanculéanu et Baup, relatives aux sinusites maxillaires purulentes, ont démontré que dans les sinusites consécutives à une rhinite purulente on trouve des streptocoques et des pneumocoques (tandis que dans celles qui sont consécutives à une périostite alvéolodentaire on trouve surtout des espèces microbiennes anaérobies, bacillus ramosus, perfringens, serpens, etc.).

Dans ces conditions la pathogénie des poussées de péricystite phlegmoneuse dans l'observation de Julien D... est évidente. Il s'est produit une affection ascendante du canal nasal et ensuite du sac par des streptocoques ayant provoqué aussi l'infection du sinus maxillaire gauche.

Quant au traitement, il doit être radical tant pour la péricystite phlegmoneuse (curetage et thermocautérisation profonde) que pour la sinusite maxillaire (intervention par la voie canine). Incision de la muqueuse buccale au niveau du sillon gingivo-labial supérieur. Décollement du périoste à hauteur de la fosse canine. Effondrement de la paroi antérieure du sinus à l'aide d'un ciseau bien tranchant. Curetage de la muqueuse hypertrophiée et des masses myxomateuses ou polypeuses.

Tamponnement serré à la gaze iodoformée. Puis contre-ouverture large du sinus au niveau du méat inférieur et par là tamponnement à la gaze iodoformée, de façon à pouvoir fermer la communication avec la bouche, qui d'une part permet l'écoulement du pus dans la bouche, et d'autre part provoque constamment une réinfection du sinus par la salive, les aliments, etc.

Catégorie II

Six cas de dacryocystite aiguë à streptocoques d'origine nasale

Cas 1er. — Femme de trente-cinq ans. Depuis quelques années, larmoiement à gauche. Depuis un mois, paupières collées chaque matin. Comme symptômes subjectifs depuis quelques jours douleurs lancinantes vers l'angle interne de l'œil gauche. Comme traitement antérieur la malade a subi deux séances de cathétérisme à gauche il y a quatre mois. En dehors de ces deux interventions elle n'a jamais eu que des lotions boriquées. *Actuellement, le 26 mars*, gonflement marqué de la région du sac, qui est rouge. Le canalicule lacrymal inférieur a été incisé par la méthode de Bowman lors du cathétérisme. Par pression, du pus sort, abondant, par les points lacrymaux. La conjonctive est fortement injectée. La cornée est intacte. Larmoiement intense. Aspect d'ensemble nettement inflammatoire aigu. Les recherches bactériologiques mettent en évidence des pneumocoques associés à des streptocoques en prédominance.

Le traitement consiste en fréquentes lotions et compresses à l'eau bouillie froide, à domicile.

Le 5 avril. — La poussée aiguë est terminée.

Le 17 avril. — La dacryocystite gauche persiste, mais a repris une allure chronique. L'examen bactériologique (examen direct et cultures) renseigne des pneumocoques purs. Ainsi il est intéressant de voir qu'une fois la régression des phénomènes inflammatoires aigus complètement terminée on ne retrouve plus que des pneumocoques dans le sac.

Cas 2. — Jeune fille de 21 ans. *Le 12 janvier.* Depuis environ un an, larmoiement à droite. Début par des douleurs au niveau de la région du sac lacrymal droit. Puis écoulement de mucus clair ; puis de pus, depuis trois mois.

Actuellement, par pression légère, fusée de pus très abondante par les points lacrymaux. Pas de gonflement de la région du sac. Caroncule lacrymale œdématiée, rouge. Conjonctive palpébrale et bulbaire, plus injectée à droite qu'à gauche. Examen d'un frottis sur lame, bacilles de Weeks et pneumocoques abondants.

Le 6 février. — La malade n'étant plus revenue depuis le 12 janvier, nouvel examen bactériologique : même résultat.

Une injection dans le sac d'eau stérile passe facilement dans le nez.

Le 10 février. — La malade revient avec du larmoiement considérable à droite, la région du sac lacrymal droit rouge et gonflée, et un état

général d'abattement et de fièvre. Elle raconte qu'elle a contracté un rhume en retournant chez elle après sa visite précédente. En effet, son nez coule abondamment (sécrétion nettement purulente). La rhinoscopie antérieure renseigne de la rhinite hypertrophique purulente. Une légère pression au niveau du sac en fait sortir du pus épais, crémeux. Frottis sur lames et ensemencements dans des tubes de bouillon glucosé de pus du sac et de pus nasal. Streptocoques. Traitement par de simples lotions et compresses à l'eau bouillie froide.

Le 2 mars. — Phase aiguë terminée. Proposition d'extirpation du sac lacrymal droit.

Le 4 avril. — Opération. Suture endermique.

Le 10 avril. — Guérison. Belle cicatrice.

Cas 3, 4, 5, 6. — Nous avons noté encore ces quatre autres cas dont les observations se ressemblent au point que nous pouvons les résumer en une seule type : La personne souffre, depuis plus ou moins longtemps, de larmoiement avec écoulement de pus par les points lacrymaux. A un moment donné, sans cause appréciable, ou sous l'influence d'un froid, de frottements, d'une irritation quelconque, la région du sac lacrymal se tuméfie, devient rouge, douloureuse.

Néanmoins, la poche de pus parvient encore à se vider par les orifices naturels, par les points lacrymaux, et chaque matin les paupières sont collées. Un processus de conjonctivite aiguë de voisinage se manifeste aussi. Et le larmoiement devient intense. Ces changements locaux s'accompagnent de phénomènes généraux plus ou moins importants, frissons, élévation thermique, accélération du pouls, insomnie, accablement général.

Si nous prélevons alors du pus du sac dès sa sortie des canalicules lacrymaux, et si nous en faisons l'examen direct (Gram), nous trouvons des pneumocoques associés à des streptocoques en prédominance. Si nous ensemençons un tube de bouillon glucosé avec une anse de platine stérilisée à la flamme, puis chargée d'un peu de ce pus, nous constatons au bout de vingt-quatre heures et, mieux, de quarante-huit heures, un dépôt floconneux, le liquide devenant, lui, légèrement troublé, et l'examen bactériologique renseigne de longues chaînes de cocci en prédominance et des cocci disposés par deux.

Or, dans trois de ces cas que nous avons observés, nous avions eu l'occasion de faire l'examen bactériologique du pus au moment de la dacryocystite chronique, et nous avions trouvé des pneumocoques absolument purs. De plus, dans ces trois cas, le canal nasal était resté perméable. Que s'était-il donc passé ? Les streptocoques venus du nez avaient envahi le sac et provoqué une poussée de dacryocystite aiguë, sans que celle-ci arrive au stade de péricystite phlegmoneuse.

Ces six cas de poussée de dacryocystite aiguë à streptocoques d'origine nasale présentent un réel intérêt, parce qu'ils constituent en quelque sorte une transition clinique et bactériologique entre la dacryocystite

chronique à pneumocoques purs et la péricystite phlegmoneuse à streptocoques, celle-ci n'étant en effet que la propagation au tissu sous-muqueux des streptocoques, agents infectieux que l'on trouve en prédominance dans les dacryocystites aiguës.

Cliniquement, ce qui différencie une poussée de dacryocystite aiguë d'une péricystite phlegmoneuse, c'est d'abord le fait que l'inflammation, moins vive, moins étendue, n'intéresse pas la sous-muqueuse, ensuite le fait que l'écoulement de pus par les canalicules lacrymaux persiste, et qu'il n'y a dès lors, la collection purulente n'étant pas close, aucune tendance à fistulisation, ce qui est la règle au contraire dans les péricystites phlegmoneuses, fermées, elles, de toutes parts, si bien que Mazet, dans sa thèse de Paris 1894 les appelait des ' empyèmes ' aigus du sac lacrymal.

Bactériologiquement, ces deux affections sont analogues. Elles sont dues toutes deux à l'envahissement des streptocoques, lesquels supplantent progressivement, de façon plus ou moins complète, les pneumocoques ; seulement, dans la péricystite phlegmoneuse, les streptocoques envahissent le tissu sous-muqueux.

Quant au traitement de la dacryocystite aiguë, il sera tout différent de celui de la péricystite phlegmoneuse. Dans celle-ci, l'existence d'une importante collection purulente qui cherche à se frayer un passage vers l'extérieur en ulcérant la peau, nécessite une prompte élimination du pus à l'aide d'une incision, puis le curetage et la thermocautérisation de la cavité, puis un bon tamponnement à la gaze iodoformée.

Dans la dacryocystite aiguë, on se contentera de mettre des compresses d'eau stérile froide souvent renouvelées. Et on évitera strictement tout cathétérisme, de crainte d'augmenter l'infection par l'irritation ou même de provoquer des égastrignures qui soient pour les streptocoques des portes d'entrée vers la sous-muqueuse.

Généralement au bout de quelques jours la régression de la poussée inflammatoire se produira, et la situation redeviendra ce qu'elle était antérieurement, à savoir de la dacryocystite chronique à pneumocoques ; on pourra pratiquer alors l'extirpation du sac lacrymal et obtenir ainsi une guérison définitive.

Si, contrairement aux prévisions, la poussée inflammatoire aboutit à une péricystite phlegmoneuse à cause de l'infection progressive du tissu sous-muqueux, il sera nécessaire de traiter ce phlegmon péricystique suivant la méthode habituelle (incision, curetage, drainage si c'est nécessaire, et pansements consécutifs faits avec soin). La guérison viendra aussi, mais beaucoup plus lentement, et avec une cicatrice plus apparente que si la poussée inflammatoire a pu être enrayée et si ultérieurement on a pu pratiquer dans de bonnes conditions l'extirpation du sac lacrymal avec suture endermique.

Catégorie III

Trois cas de péricystite phlegmoneuse tuberculeuse chez des enfants et déductions qu'ils entraînent quant à la pathogénie de cette affection

Cas 1er. — *Le* 16 *janvier.* — Van M... (Marie), six ans et demi.

Il y a une quinzaine de jours est apparu à l'angle interne de l'œil gauche un gonflement qui a progressivement augmenté. Actuellement, région du sac lacrymal gauche fortement tuméfiée, rouge, douloureuse à la pression. Fistule de ½ cm. en dedans et en dessous de l'angle interne de l'œil. Du pus s'en écoule en abondance, sale et souillé de sang.

Examen bactériologique direct de ce pus. Staphylocoques. Recherche du bacille de Koch sans résultat.

Rhinoscopie antérieure : eczéma du vestibule ; croûtes têtes cornets inférieurs.

Le 19 *janvier.* — Sous chloroforme, incision au thermocautère du phlegmon péricystique ; destruction ; tamponnement à la gaze iodoformée ; pansement sec ; bandeau. Les jours suivants, malgré les soins rigoureux apportés aux pansements, le bourgeonnement ne se fait pas.

Le 8 *février.* — La cicatrisation paraît mieux s'effectuer.

Le 15 *mars.* — Il reste un trajet fistuleux.

Le 20 *mars.* — La suppuration reparaît. Gonflement. Fluctuation.

Le 22 *mars.* — Réintervention.

Inoculation d'un cobaye, sous la peau du ventre, avec des débris de fongosités.

Le 30 *mars.* — La suppuration reste considérable. La plaie est profonde, à bords irréguliers. La sonde cannelée touche au périoste dénudé. Il apparaît à l'évidence qu'un processus d'ostéite entretient la suppuration.

Le 22 *avril.* — Guérison, apparente du moins.

Le 2 *mai.* — Mort du cobaye inoculé. Autopsie. Lésions tuberculeuses. Rate volumineuse, avec tubercules caséeux confluents et tubercules plus jeunes, isolés, encore durs. Foie : lésions analogues, moins marquées ; ganglions inguinaux caséeux contenant d'innombrables bacilles de Koch. Pas de semis apparent de granulations miliaires sur la surface des poumons, mais tubercules dans la partie corticale des reins.

Cas 2. — *Le* 10 *janvier.* — M... (Maria), six ans.

Depuis quatre semaines, épiphora très abondant accompagnant une tuméfaction douloureuse au niveau du sac lacrymal droit ; actuellement peau amincie, violacée ; fluctuation nette ; fistule par laquelle s'écoule une sérosité roussâtre. Ganglions sous-maxillaires et rétro-maxillaires fortement engorgés. Ganglion pré-auriculaire non palpable.

Examen bactériologique direct du pus. Recherche du bacille de Koch sans résultat. Ensemencement sur gélose.

Le 11 *janvier.* — Nombreuses colonies de staphylocoques dorés pyogènes.

Rhinoscopie antérieure : rhinite purulente.

Le 12 janvier. — Curetage sous chloroforme et destruction au thermocautère, tamponnement à la gaze iodoformée ; pansement sec. Bacilles de Koch trouvés dans fongosités prélevées au moment du curetage.

Le 1er février. — Aspect de la plaie mauvais. Bords fongueux. Suppuration profonde.

Le 22 février. — Cicatrisation très lente.

Le 5 mars. — Guérison apparente.

(En effet deux mois plus tard survenait une nouvelle poussée de même nature.)

Cas 3. — T..., Emma, huit ans. Péricystite phlegmoneuse gauche. Adénite sous-maxillaire suppurée. Inoculations positives quant à la tuberculose, à des cobayes, de pus prélevé par ponction du phlegmon et de pus provenant des ganglions suppurés. Même traitement que dans les deux premiers cas.

L'intérêt de ces trois cas de péricystite phlegmoneuse chez des enfants réside dans leur caractère tuberculeux.

En effet, s'il est vrai qu'à l'examen bactériologique direct du pus s'écoulant par les fistules, dans les deux premiers cas, ou ponctionné, dans le troisième, nous n'avons pas trouvé de bacilles de Koch, toutefois, dans le premier cas l'inoculation au cobaye de débris de fongosités, positive, dans le second cas la présence de bacilles de Koch dans les fongosités prélevées lors du curetage, dans le troisième cas les inoculations, positives, à des cobayes, de pus obtenu par ponction du phlegmon et de pus provenant des ganglions sous-maxillaires suppurés, et dans les trois cas l'allure clinique de l'affection, l'aspect fongueux des parois du phlegmon, la longue durée des suppurations, la tendance aux récidives et aux fistules persistantes et les fortes adénopathies concomitantes, tout cela ne laisse aucun doute quant à la nature tuberculeuse de ces phlegmons péricystiques.

Au surplus, le processus d'ostéite sous-jacente, démontré par le fait que le stylet rencontre une surface osseuse dénudée, nous permet d'affirmer que ces péricystites phlegmoneuses tuberculeuses ne sont que des manifestations d'une ostéite tuberculeuse intéressant les os de la gouttière lacrymale (branche montante du maxillaire supérieur et os unguis).

Voici donc, résumée, notre manière d'interpréter la pathogénie des péricystites phlegmoneuses tuberculeuses des enfants : ostéopériostite tuberculeuse primitive des os de la gouttière lacrymale ; envahissement par les bacilles de Koch du tissu sous-muqueux du sac, puis du sac lui-même ; finalement vaste phlegmon dont les parois tomenteuses, irrégulières, avec des fongosités farcies de follicules tuberculeux, constituent la zone active et dont le centre renferme une masse purulente caséeuse parsemée de débris dégénérés des divers tissus. Ce phlegmon corrodant progressivement la peau ne tarde pas à se faire jour vers le dehors. Au point de vue bactériologique, évidemment tant que le processus tuberculeux n'a pas dépassé le tissu sous-muqueux du sac, les bacilles de Koch seuls sont en jeu ; mais ensuite, comme il survient

au niveau de la muqueuse du sac des phénomènes réactionnels de voisinage, le mécanisme normal de l'élimination des larmes est troublé et dès lors les microorganismes latents dans le sac interviennent avec une certaine virulence et il en résulte des symptômes de dacryocystite simple, prémonitoires en quelque sorte de la péricystite phlegmoneuse ; et dans ces conditions lorsque le phlegmon sera complètement constitué, nous pourrons trouver dans son pus non seulement des bacilles de Koch, mais également des microorganismes provenant de l'infection autonome du sac. Cette infection du sac surajoutée à l'infection tuberculeuse d'abord pure du tissu péricystique est la raison de l'aspect plus ou moins aigu de l'affection. Si le sac n'était pas là et n'amenait pour son propre compte certains agents infectieux, nous aurions l'abcès froid tuberculeux à évolution torpide. Mais le contraire étant, l'abcès froid prend une allure plus ou moins aiguë, ce qui fait qu'on peut l'appeler phlegmon malgré sa nature fondamentalement tuberculeuse. D'autre part, du fait que la fistulisation se produit, une porte est ouverte aux infections secondaires (staphylocoques particulièrement, comme dans nos deux premiers cas). D'où pratiquement il résulte que si lors de l'examen bactériologique direct du pus du phlegmon péricystique l'absence de microorganismes doit faire soupçonner la tuberculose, la présence de microorganismes divers autres que les bacilles de Koch ne permet pas d'écarter d'emblée l'étiologie tuberculeuse.

En conclusion nous pensons, par l'étude de ces trois cas et les réflexions que cette étude nous suggère, que la lésion fondamentale chez les enfants est l'ostéo-périostite tuberculeuse des os de la gouttière lacrymale, dont la péricystite phlegmoneuse n'est en quelque sorte qu'un symptôme adapté à la localisation spéciale de l'affection.

Catégorie IV

La Dacryocystite tuberculeuse

Nous avons rendu compte de nos trois cas de péricystite phlegmoneuse tuberculeuse observés chez des enfants et dûs, ainsi que nous l'avons exposé, à une ostéopériostite tuberculeuse des os de la gouttière lacrymale propagée au tissu sous-muqueux du sac.

Nous allons maintenant étudier la dacryocystite tuberculeuse, c'est-à-dire l'infection de la muqueuse même du sac par les bacilles de Koch.

La dacryocystite tuberculeuse peut être primitive ou secondaire, consécutive alors soit à une tuberculose conjonctivale soit à une tuberculose nasale.

La dacryocystite tuberculeuse primitive est possible, certes, mais doit être excessivement rare. La littérature ne nous en renseigne que quelques cas, Loidholt, un (thèse Merseburg, 1889), Bock, un (*Gaz. méd. hebd. de Vienne*, 1892), et Vincent, un (thèse de Lyon, 1900). Mais comme le fait très bien remarquer Poulard dans son magnifique travail sur la tuberculose des voies lacrymales (*Arch. d'Ophtalm.*, t. xxiii,

No. 12, décembre 1903), ces trois cas sont fortement sujets à caution. En effet, dans aucun de ces cas il n'a été prélevé de fragment de la muqueuse des fosses nasales au niveau du méat inférieur en vue d'inoculation au cobaye, et par conséquent on ne sait si la tuberculose constatée au niveau du sac ne provenait pas d'une tuberculose nasale, par voie ascendante. La même remarque s'adresse à un autre cas de tuberculose prétendument primitive du sac lacrymal publié par Rollet, de Lyon (*Revue générale d'ophtalm.*, juin 1903). Il s'agit d'une jeune fille de 14 ans qui, soignée à 6½ ans pour lésions osseuses du pied, adénites suppurées du cou et gommes cutanées (cicatrices), présente du gonflement de l'angle interne de l'œil droit avec larmoiement, et des petits ganglions indolores préauriculaires, sous-maxillaires et carotidiens. Le sac lacrymal extirpé et ouvert montre des parois remplies de fongosités dans lesquelles l'examen bactériologique démontre la présence de bacilles de Koch. L'inoculation de fongosités au cobayes est du reste positive. Il s'agit donc d'un vrai cas de dacryocystite tuberculeuse. Mais celle-ci est-elle réellement primitive ? Rien à la conjonctive ni aux fosses nasales, dit l'auteur. A la conjonctive l'examen direct est probant. Mais pour ce qui concerne les fosses nasales il ne suffit pas. Il eût fallu procéder à une inoculation sous-cutanée au cobaye d'une petite parcelle de muqueuse prélevée dans le méat inférieur pour pouvoir écarter sûrement l'étiologie nasale de ce cas de dacryocystite tuberculeuse. Or, cela n'a pas été fait.

En tout état de cause la dacryocystite tuberculeuse primitive doit être excessivement rare.

Rare aussi est la dacryocystite tuberculeuse consécutive à la tuberculose de la conjonctive. Gayet en cite un cas (*Soc. fr. d'Ophtalm.*, 28 janvier 1885), mais il n'a pas fait non plus l'étude bactériologique de la muqueuse du méat inférieur, ce qui ne permet pas d'écarter l'hypothèse suivant laquelle la dacryocystite tuberculeuse dans ce cas serait consécutive à une tuberculose nasale et aurait provoqué ensuite à son tour de la tuberculose conjonctivale.

Quant aux cas de dacryocystite tuberculeuse observés consécutivement à de la tuberculose de la muqueuse nasale, ils sont nombreux. Morax par exemple en cite un cas (*Compte rendu de la Soc. fr. d'Ophtalm.*, 1908), Poulard deux cas (*Arch. d'Ophtalm.*, décembre 1903).

Dans le premier cas Poulard, bien que la rhinoscopie antérieure ne renseignât aucune lésion notable permettant d'affirmer la tuberculose des fosses nasales, prélève dans le méat inférieur une petite parcelle de muqueuse et l'inocule sous la peau de deux cobayes, lesquels deviennent rapidement tuberculeux ; dans le second cas, Poulard obtient également une inoculation positive avec du pus des ganglions suppurés.

Ainsi donc, on peut dire que la dacryocystite tuberculeuse, c'est-à-dire l'infection de la muqueuse même du sac par les bacilles de Koch, est rarement primitive, rarement aussi consécutive à une tuberculose conjonctivale, mais provient au contraire la plupart du temps d'une tuberculose des fosses nasales.

Catégorie V

Six cas de dacryocystite subaiguë à diplobacilles Morax-Axenfeld d'origine nasale

Cas 1er. — Femme de 56 ans, coiporteuse.

Épiphora depuis un an, avec issue de muco-pus abondant par les points lacrymaux, des deux côtés, sous une légère pression au niveau des sacs.

Le 4 décembre. — Un oculiste a fait une tentative d'extirpation du sac lacrymal droit, mais n'a pu terminer l'opération à cause d'une hémorragie très forte, incoercible, après l'incision cutanée.

Actuellement, le 14 décembre, accentuation de l'écoulement de muco-pus, des deux côtés, et en même temps poussée de rhinite purulente intense. Difficultés de respirer, tantôt par l'une, tantôt par l'autre narine. Mouchage fréquent.

A droite et à gauche, les injections faites dans le sac passent dans le nez, mais assez difficilement. *Examen bactériologique direct* du muco-pus des sacs : pneumocoques et diplobacilles Morax-Axenfeld.

Le 26 janvier. — Nouvel examen bactériologique : pneumocoques et diplobacilles en prédominance.

Le 31 janvier. — Rhinoscopie antérieure. Rhinite purulente hypertrophique. Cornets inférieurs fortement gonflés s'avançant vers la cloison. Pus dans le rhino-pharynx. *Examen bactériologique direct* du pus nasal : pneumocoques-diplobacilles en prédominance. — Bacilles de Weeks.

Le 1er février. — Institution du traitement au sulfate de zinc :

1° Traitement nasal : Badigeonnages avec une solution à 25 cgr. pour cent.

2° Traitement du sac : Injection d'une solution de sulfate de zinc à ½ pour cent, puis à 1 pour cent.

3° Soins conjonctivaux : Instillation d'un collyre au sulfate de zinc (2 cgr. pour 10 gr. d'eau distillée).

Jours suivants : même traitement.

Le 6 février. — La solution au sulfate de zinc à 1 pour cent injectée dans les sacs passe maintenant facilement dans les méats inférieurs et de là dans l'arrière-gorge. La perméabilité des voies lacrymales est donc intégralement rétablie. D'autre part il ne s'écoule plus de muco-pus du tout, ni à droite ni à gauche, par pression au niveau des sacs lacrymaux.

Quant au nez il est amélioré beaucoup. L'air passe plus facilement dans les narines et le mouchage est fortement amoindri.

Le 8 février. — L'amélioration persiste. L'aspect des yeux est tout différent de ce qu'il était il y a une huitaine de jours. Plus de rougeur conjonctivale. Il s'est produit une sorte d'accalmie des phénomènes inflammatoires.

Le 15 février. — La guérison est totale.

La malade a été revue depuis. Larmoiement diminué. Plus d'écoulement de pus par les points lacrymaux. Les paupières ne sont plus collées chaque matin comme avant l'institution du traitement par le sulfate de zinc.

Cas 2. — Femme de cinquante ans.

Le 3 mai. — Début il y a quatre semaines. La malade affirme n'avoir jamais eu précédemment quoi que ce soit aux yeux.

D'abord, coïncident avec des maux de tête, inflammation douloureuse au niveau de l'angle interne de l'œil droit, avec larmoiement considérable. Puis, bientôt, paupières collées chaque matin.

Actuellement la région du sac lacrymal droit est légèrement rouge et tuméfiée. Par pression il s'écoule un peu de muco-pus.

A l'examen bactériologique direct on y trouve des pneumocoques associés à des diplobacilles Morax-Axenfeld extrêmement abondants. Les injections faites à droite dans les canalicules lacrymaux passent bien dans le méat inférieur droit.

La malade présente en outre à droite tous les symptômes cliniques d'une conjonctivite diplobacillaire : conjonctivite, tarsienne et bulbaire, légèrement injectée ; vers les angles, aspect croûteux empiétant sur la peau voisine ; marche sub-aiguë de l'affection. Du reste, l'examen bactériologique direct de filaments de pus recueillis vers l'angle externe montre une grande quantité de diplobacilles associés à des pneumocoques. La cornée est intacte, sans ulcération. L'examen de l'œil gauche ne révèle rien de particulier.

Rhinoscopie antérieure : rhinite hypertrophique, forte crête de la cloison à droite, dans sa partie inférieure ; crête légère à gauche, dans sa partie inférieure aussi.

Prélèvement de muco-pus nasal pour l'examen bactériologique direct : nombreux pneumocoques — gros diplocoques non encapsulés (diplococcus catarrhalis) et diplobacilles Morax-Axenfeld en abondance.

Traitement. — Chaque matin, injection dans le sac droit d'une solution de sulfate de zinc à $\frac{1}{2}$ pour cent, badigeonnages, avec la même solution, des conjonctives, dans leur partie tarsienne, et aussi de la région cutanée des angles. D'autre part, badigeonnages des méats inférieurs au moyen d'un tampon d'ouate adapté à un porte-ouate et imprégné de la même solution.

A domicile, instillations dans l'œil, quatre fois par jour, d'un collyre contenant 2 cgr. de sulfate de zinc pour 10 gr. d'eau distillée.

Les jours suivants : même traitement. Amélioration rapide.

Le 10 mai. — Il ne s'écoule plus de pus par pression au niveau du sac lacrymal droit. La conjonctivite diplobacillaire est guérie.

Néanmoins, le traitement est continué pendant une huitaine de jours pour éviter une récidive.

Le 26 mai. — Ablation de la crête de la cloison du côté droit, au rabot, sous cocaïne-adrénaline. Pansements à la gaze en bandelettes à la ferropyrine.

Ultérieurement. — Pommade boro-mentholée.

Le 16 juin. — La malade est totalement guérie. La respiration nasale, quoique encore imparfaite, se fait beaucoup mieux qu'auparavant. Pulvérisations d'adrénaline inhalante. Bons résultats par la suite.

Cas 3, 4, 5 et 6. — En faisant l'examen bactériologique systématique du muco-pus provenant de sacs lacrymaux malades, nous avons découvert quatre fois encore les diplobacilles Morax-Axenfeld, dans deux cas à l'état de pureté, dans les autres en association avec des pneumocoques mais prédominants.

La perméabilité du canal nasal subsistait intégralement, car les injections médicamenteuses faites dans le sac passaient facilement dans les fosses nasales.

L'affection revêtait un caractère subaigu et s'accompagnait dans deux cas de conjonctivite angulaire à diplobacilles (vers les angles aspect croûteux empiétant sur la peau voisine), l'un de ces deux cas étant au surplus compliqué d'un ulcère cornéen à diplobacilles.

La rhinoscopie antérieure nous a fourni les renseignements suivants :

Cas 3. — Rhinite hypertrophique avec cornets inférieurs volumineux (dacryocystite droite).

Cas 4. — Rhinite hypertrophique. Déviation de la cloison en S avec anosmie (dacryocystite gauche).

Cas 5. — Rhinite hypertrophique (dacryocystite gauche).

Cas 6. — Rhinite hypertrophique. Déviation du septum à gauche (dacryocystite gauche).

Examen bactériologique du muco-pus nasal dans tous ces cas : diplobacilles en masse.

Traitement institué basé sur le sulfate de zinc qui a la valeur d'un véritable spécifique vis-à-vis des diplobacilles. Injections dans le sac lacrymal à l'aide de la seringue d'Anel d'une solution de $ZnSO_4$ à $\frac{1}{2}$ pour cent ou 1 pour cent, aidées dans deux cas par l'incision du canalicule lacrymal inférieur (méthode de Bowman). Contre la conjonctivite diplobacillaire : instillations d'un collyre au $ZnSO_4$ (2 cgr. pour 10 grm. d'eau distillée). Badigeonnages de la région cutanée des angles, attouchements de l'ulcère cornéen au moyen d'une solution à $\frac{1}{2}$ pour cent.

Dans les fosses nasales, pommade boro-mentholée, pulvérisations d'adrénaline inhalante. Opérations indiquées.

Quelles réflexions nous suggèrent, au point de vue de leur pathogénie, ces six cas de dacryocystite subaiguë à diplobacilles Morax-Axenfeld ?

Ces dacryocystites proviennent du nez, par voie ascendante (le canal nasal étant encore perméable), ce qui est facile si nous nous souvenons, d'abord que les diplobacilles sont hôtes habituels du nez ensuite que la muqueuse du canal nasal et du sac lacrymal possède vis-à-vis d'eux la même réceptivité que la pituitaire parce qu'elle en présente les caractères histologiques, soit normaux, soit pathologiques.

Quelle a été maintenant le cause occasionnelle de ces dacryocystites ? C'est l'état de rhinite hypertrophique aggravé par quelque déformation

intra-nasale (cornets inférieurs hypertrophiés, déviations de la cloison, crêtes du septum).

Ces dacryocystites peuvent se compliquer de conjonctivite diplobacillaire, mais n'en procèdent pas. Les conjonctivites diplobacillaires primitives, elles sont provoquées par le transport direct sur la conjonctive, par l'intermédiaire des doigts, mouchoirs, etc., des diplobacilles qui sont, nous l'avons dit, hôtes habituels du nez.

Catégorie VI

Quatre cas de dacryocystite ozéneuse à diplocoques de Loewenberg, d'origine nasale

Nous avons observé quatre cas de dacryocystite ozéneuse à diplocoques de Loewenberg, purs dans deux cas et dans les autres associés à des bacilles pseudo-diphtériques.

Dans l'un de ces cas il y avait conjonctivite concomitante à diplocoques encapsulés de Loewenberg. Notons en passant que dans onze cas d'ozène Terson et Gabriélidès ont trouvé six fois ces microorganismes au niveau des conjonctives.

Ils peuvent être amenés là par les linges, par les doigts, etc., transport tout mécanique, ou bien y arriver pathologiquement, par voie ascendante, en remontant le canal nasal, puis le sac lacrymal, provoquant au niveau de celui-ci une dacryocystite plus ou moins marquée cliniquement.

Dans les quatre cas que nous avons observés, il s'agissait de dacryocystites à symptômes habituels (larmoiement, avec écoulement de muco-pus par les points lacrymaux), et c'est l'examen bactériologique seul qui nous a permis de différencier ces cas.

Le canal nasal était resté perméable, fait important qui donne la clef de la pathogénie des dacryocystites ozéneuses.

L'examen rhinoscopique nous a renseigné :

Cas 1er. — Rhinite atrophique, cavité sèche, pharynx vernissé (dacryocystite gauche).

Cas 2. — Rhinite fortement atrophique, croûtes verdâtres, cavité sèche (dacryocystite droite).

Cas 3. — Rhinite atrophique considérable, nez en 'selle' (dacryocystite bilatérale).

Cas 4. — Rhinite atrophiante, cornets réduits, insensibilité de la muqueuse (dacryocystite droite).

Examen bactériologique direct de mucus nasal étalé sur des lames et coloré à la solution de Ziehl (fuchsine phéniquée) : diplocoques encapsulés de Loewenberg, souvent disposés en chaînes. Prélèvement de filaments par arrachement des croûtes ou râclage de la muqueuse.

Et cultures (*a*) sur gélatine, colonies blanchâtres piriformes à la surface ; colonies plutôt jaunâtres et arrondies dans l'épaisseur.

(*b*) Sur gélose, surface uniformément recouverte d'une légère couche grisâtre.

(*c*) Sur bouillon peptonisé à ½ pour cent au fond, dépôt grumeleux, au-dessus duquel le bouillon est resté à peu près limpide.

A l'examen des cultures, microbe de Loewenberg reconnu.

Comme traitement, dans deux cas (1 et 4), des injections dans le sac, à l'aide de la seringue d'Anel, et des solutions de protargol, à doses progressives, ont amené une guérison assez rapide ; mais dans les deux autres cas (2 et 3), l'extirpation des sacs lacrymaux a dû être faite, d'après la méthode indiquée par le Dr de Ridder à la réunion du 27 avril 1901 de la Société belge d'Ophtalmologie.

Catégorie VII

La dacryocystite chronique à pneumocoques ; étude historique et critique de la valeur des diverses méthodes thérapeutiques utilisées dans cette affection

Nous ne nous attarderons pas à décrire en détail les symptômes cliniques de la dacryocystite chronique à pneumocoques ; c'est, de toutes les affections des voies lacrymales, celle qui est de beaucoup la plus fréquente ; elle se caractérise essentiellement par du larmoiement et de l'écoulement de muco-pus par les points lacrymaux soit spontanément, soit sous l'influence d'une légère pression au niveau du sac.

Dans un certain nombre de cas, nous avons observé de *l'ectasie plus ou moins importante du sac lacrymal*. Or, il est intéressant de rechercher comment fonctionne dans ces cas l'appareil lacrymal, d'en étudier en quelque sorte le mécanisme pathologique.

Normalement, l'élimination des larmes se fait, si nous nous basons sur les dernières recherches de Schirmer (' Nachtrag zu meiner Theorie der Thränenabfuhr ', von Professeur Otto Schirmer in *Greifswald* ; Graefe's *Archiv für Ophthalmologie*, Bd. lxiii), à ce sujet, de la façon suivante :

Dans un premier temps se produit l'aspiration des larmes vers le sac, par les canalicules lacrymaux, à cause de la tension du ligament palpébral interne, qui attire en avant la paroi du sac y adhérente.

Dans un second temps se produit la chasse des larmes vers le nez, grâce à la compression élastique du sac. Mais pourquoi, dans le premier temps, n'y a-t-il pas aussi bien aspiration des liquides se trouvant dans le canal nasal, et pourquoi, lors du second temps, n'y a-t-il pas aussi bien chasse des liquides du sac vers les canalicules lacrymaux ? C'est, dit Schirmer, avec expériences à l'appui, parce que le revêtement de mucus qui garnit l'orifice du canal nasal dans le méat inférieur joue le rôle d'une véritable soupape qui existerait là, vers la partie inférieure du canal nasal donc, et s'ouvrirait seulement du côté du nez. Lors du premier temps, le sac ne peut aspirer que du côté des canalicules lacrymaux, puisque le bouchon de mucus de l'extrémité inférieure du canal nasal tient celui-ci fermé. Lors du second temps, le sac ne peut se vider

que du côté du nez, les canalicules lacrymaux ayant leurs parois accolées grâce à leur élasticité.

Or, *dans les cas d'ectasie du sac,* au cours d'une dacryocystite chronique à pneumocoques, si même on parvient à rétablir, intégralement, la perméabilité des voies lacrymales, le défaut de compression élastique lors du second temps empêchera la chasse vers le nez des liquides accumulés dans le sac, d'où une tendance de celui-ci à s'ectasier toujours davantage.

L'une de nos observations est à cet égard significative. Il s'agit d'un vrai cas d'ectasie simple, primitive, du sac lacrymal gauche, lequel soulève le ligament palpébral interne. Le mécanisme normal de l'écoulement des larmes est rompu, puisqu'il y a de l'épiphora depuis six mois déjà. Cependant les injections, poussées par les canalicules lacrymaux à l'aide d'une seringue d'Anel, passent très bien dans le nez ; à plusieurs reprises, nous avons cherché en vain à faire sortir par pression du mucopus du sac ; du reste, les paupières ne sont jamais collées le matin. Il ne s'agit donc pas dans ce cas d'une dacryocystite chronique avec ectasie du sac consécutive. Celle-ci paraît autonome. Et, dans ce cas, l'ectasie du sac, seule, correspondant à une raréfaction des fibres élastiques (nous avons pratiqué l'examen histologique de ce sac extirpé), enraie le second temps du mécanisme normal de l'écoulement des larmes.

Ces considérations nous amènent à déjà dire, en passant, que dans les cas de dacryocystite chronique à pneumocoques avec ectasie du sac, les diverses méthodes thérapeutiques ayant comme but final le rétablissement de la perméabilité des voies lacrymales sont inutiles, puisque, même en supposant ce résultat atteint, le second temps du mécanisme normal de l'élimination des larmes ne peut plus se faire. Dans ces cas donc, l'extirpation du sac lacrymal s'impose.

Quant au *traitement des dacryocystites chroniques à pneumocoques sans ectasie du sac concomitante,* il peut être orienté dans deux sens bien distincts. Il peut avoir comme but final, soit la conservation des voies lacrymales avec rétablissement de leur perméabilité, soit la destruction des voies lacrymales.

Examinons d'abord les *méthodes qui se proposent comme but final, la conservation des voies lacrymales.*

Il en est une que nous ne voulons citer que pour mémoire, à titre documentaire, parce qu'elle intéresse particulièrement notre spécialité : c'est le *cathérérisme inférieur,* pratiqué par l'orifice inférieur du canal nasal, par le *procédé de Laforest.*

Rochon-Duvigneaud s'est cependant encore érigé en protagoniste de cette méthode du cathétérisme inférieur, mais pas par le procédé de Laforest, en mai 1900 (*Archives d'Ophtalmologie,* p. 265). ' Pourquoi,' dit-il, ' aborder les voies lacrymales par leur extrémité la plus anfractueuse et la plus étroite ? Qu'on les aborde donc par leur orifice nasal.' Voici, en effet, ce qu'il fait, après avoir au préalable réglé sur le cadavre son manuel opératoire : Ayant son miroir frontal, il maintient la fosse nasale dilatée, béante, par un écarteur bivalve. Il incise le plus près

possible de son insertion au maxillaire le tiers antérieur du cornet, puis sectionne le lambeau ainsi détaché. La région où s'ouvre le canal nasal est alors découverte, et en introduisant un stylet par un des points lacrymaux, on aperçoit son extrémité inférieure. A l'aide d'un galvanocautère, on détruit sur le stylet la languette muqueuse qui le recouvre, et on obtient ainsi un orifice situé immédiatement sous le bord réséqué du cornet et qui correspond à l'orifice du canal osseux. Par cet orifice, on peut ultérieurement faire du cathétérisme des voies lacrymales, soit à l'aide du cathéter de Laforest, soit à l'aide d'un stylet de trousse en argent, fin et de courbure voulue, soit surtout à l'aide de la sonde de Gensoul, dont la courbure est meilleure ; et on peut aussi par cette voie pratiquer des curetages méthodiques, cautériser à l'intérieur du canal nasal avec un galvanocautère spécial, mettre des crayons médicamenteux, des mèches de gaze, etc.

Nous croyons bien que cette méthode de Rochon-Duvigneaud, tout originale et ingénieuse qu'elle soit, n'est plus utilisée par aucun spécialiste.

Mais il n'en est pas de même du *cathétérisme supérieur progressif des voies lacrymales à l'aide de sondes numérotées de Bowman, associé ou non aux injections antiseptiques dans le sac.* Ce cathétérisme progressif est encore, on ne sait trop pourquoi, couramment usité. Or, nous considérons qu'il doit être catégoriquement rejeté, parce qu'il est *douloureux* et *inutile, sinon nuisible.*

Douloureux : En effet, l'arrivée du cathéter dans le canal nasal provoque une compression vive de la muqueuse, donc de tous les filets nerveux qui s'y distribuent, contre la paroi osseuse rigide de ce canal, d'autant plus que la muqueuse est hypertrophiée et de sensibilité accrue par le processus inflammatoire.

D'autre part, s'il existe une obstruction plus ou moins complète du canal nasal, ce qui est généralement le cas, le cathéter ne passe qu'en arrachant mécaniquement les brides qui rattachent les parois de la muqueuse, d'où une douleur intolérable pour le malade, qui tombe fréquemment en syncope.

Il est compréhensible que ce n'est pas l'instillation de cocaïne, de stovaïne ou d'alypine, ou de novocaïne où de quelque autre anesthésique local, dans l'angle interne de l'œil, ni même leur injection dans le sac, qui diminuera grandement ces douleurs, puisque celles-ci proviennent surtout du passage du cathéter dans le canal nasal, où l'anesthésique local arrive à peine, ou même n'arrive pas du tout, à cause des brides d'obstruction.

Inutile : En effet, quel est le but du cathétérisme progressif des voies lacrymales ?

1° Ce peut être simplement de se rendre compte de leur degré de perméabilité. Dans ce cas, l'injection d'un liquide quelconque, d'eau stérile, de sérum physiologique, d'eau boriquée, etc., est la méthode la plus concluante, puisqu'on n'utilise comme effort de poussée que

la faible pression avec laquelle le liquide est injecté, tandis que dans le cathétérisme l'impulsion donnée à la sonde par la main est toujours considérable, au point qu'il est impossible d'affirmer qu'un liquide pourrait passer comme le cathéter.

2° C'est le plus souvent de provoquer dans un but thérapeutique la dilatation progressive des voies lacrymales. Et, dans ce cas, les résultats que l'on obtient sont nuls, comme valeur définitive. Car le fait qu'on arrive à faire passer des cathéters de plus en plus gros ne signifie rien, relativement au rétablissement normal du cours des larmes. En effet, la pratique de la dilatation progressive pèche surtout par le fait qu'elle n'est qu'un traitement palliatif n'attaquant pas le mal dans son essence. Qu'importe que les voies lacrymales soient plus ou moins dilatées si leur infection persiste, cause permanente de recrudescences suppuratives et de réobstructions cicatricielles.

On objectera qu'on peut fort bien, en même temps qu'on pratique la dilatation progressive à l'aide des sondes numérotées de Bowman, s'attaquer à la lésion au moyen d'injections antiseptiques. Mais qu'est-ce que ces injections, même nombreuses, peuvent faire à une muqueuse tout hypertrophiée, avec de l'infiltration lymphoïde considérable et des formations villeuses qui organisent par leurs replis de vraies cryptes dans lesquelles les liquides injectés n'arrivent même pas ? Ni le cathétérisme progressif, ni les injections antiseptiques ne remettront définitivement en état une muqueuse altérée comme elle l'est dans les dacryocystites chroniques à pneumocoques, et ne viendront à bout des obstructions du canal nasal. Il se produira peut-être une amélioration passagère, et après combien de mois d'un traitement pénible ! mais tôt ou tard la dacryocystite reprendra son cours et tout sera à recommencer.

Sinon nuisible : En effet, que de poussées de dacryocystite aiguë, que de péricystites phlegmoneuses ont été provoquées par un cathétérisme forcé ou maladroit, avec déchirures de brides cicatricielles, fausses routes, etc., et en même temps avec infection survenue, sinon en descendant, du moins en remontant, l'extrémité inférieure du cathéter étant allée se charger de microorganismes dans le méat inférieur ! Et que de phlegmons orbitaires aussi ont été provoqués dans ces conditions ! La littérature en contient plusieurs cas retentissants. En 1863, Graefe signale un cas de phlegmon de la paupière inférieure suivi d'infiltration du tissu cellulaire de l'orbite, qui aboutit à un phlegmon orbitaire grave avec gonflement inflammatoire papillaire et péripapillaire, d'où la perte totale définitive de la vision. En 1863 encore, Leplat (*Bulletin de la Société d'Ophtalmologie de Paris*, 7 novembre) cite un cas de mort par méningite survenue à la suite d'un phlegmon orbitaire dû à un sondage maladroit des voies lacrymales suivi d'injection antiseptique. En 1895, Valude (*Bulletin de la Société française d'Ophtalmologie*, t. viii, p. 60) signale, suivant de quelques jours un cathétérisme forcé avec sensation manifeste de déchirure, de l'exophtalmie avec œdème des paupières, suivie de névrite, puis d'atrophie optique.

Ce sont là trois cas retentissants de la littérature. Mais il y en a quantité d'autres. Nous connaissons le cas d'un malade chez lequel un phlegmon orbitaire s'est produit consécutivement à un cathétérisme forcé.

Il faut le dire, beaucoup d'oculistes ont renoncé au cathétérisme progressif et ne font que très rarement un cathétérisme isolé, quand la dacryocystite est récente et qu'il y a lieu d'espérer qu'une simple désobstruction mécanique sera avantageuse.

Quant à nous, en conclusion de tout ce qui précède, nous répétons notre proposition du début, à savoir que le cathétérisme progressif des voies lacrymales au moyen des sondes numérotées de Bowman, ou des sondes de Galezowski (que certains oculistes encore partisans convaincus du cathétérisme préfèrent, d'abord parce qu'elles sont simples, et non doubles, comme celles de Bowman, d'où un maniement plus facile ; ensuite parce qu'elles existent en des calibres beaucoup plus forts que celles de Bowman, puisque, outre les numéros 1 à 6, il y a encore les sondes 7 à 12) doit être catégoriquement rejeté, parce qu'il est douloureux et inutile, sinon nuisible.

Voyons maintenant les *autres méthodes qui ont comme but final la conservation des voies lacrymales.*

En 1899, lors de la séance du 2 mai de la Société française d'Ophtalmologie, Kalt a proposé le mode de traitement suivant : après cathétérisme progressif au moyen des sondes de Bowman, jusqu'au No. 5 ou No. 6, introduire dans les voies lacrymales, par le canalicule lacrymal supérieur, une sonde creuse de Wecker No. 4 avec son mandrin ; puis, celui-ci ayant été retiré, faire passer dans la sonde creuse un long crin de Florence plié en deux, convexité en bas ; saisir l'anse dans le méat inférieur au moyen d'un crochet (anesthésie nasale à la cocaïne), l'attirer au dehors par le nez, puis y adapter une ' chenille ' de soie imbibée de glycérine phéniquée à $\frac{1}{20}$ et nettoyer ainsi les voies lacrymales comme on nettoierait une cheminée.

En 1899 également, Antonelli a préconisé l'emploi des solutions de protargol en injections dans le sac lacrymal atteint de dacryocystite chronique. Et il a proposé, après cathétérisme progressif des voies lacrymales au moyen des sondes de Bowman jusqu'au No. 5 ou No. 6, l'introduction de sondes de gélatine contenant 50 pour cent de protargol, de 3, 4 centimètres de longueur et d'un calibre correspondant au No. 3 ou au No. 4 des sondes de Bowman. Il voulait ainsi accroître la durée du contact avec la muqueuse, partant, augmenter le pouvoir de diffusibilité, déjà grand, du protargol, sel organique d'argent.

En 1902, Tartuferi, après de très remarquables ' études sur l'anatomie pathologique macroscopique et microscopique des dacryocystites catarrhales et purulentes chroniques ', études au cours desquelles il s'attache à prouver que dans la plupart des cas l'oblitération du canal nasal et l'hypertrophie de la muqueuse du sac ne permettent pas d'espérer un résultat stable des injections antiseptiques ni de la dilatation graduelle

par les sondes de Bowman, ni de la 'stricturotomie de Stilling' (qui consiste à faire trois, quatre incisions longitudinales dans le canal nasal, méthode qui, suivant Panas, amène réellement parfois une amélioration, mais transitoire, à cause du dégorgement par saignée locale du tissu sous-muqueux), Tartuferi, disons-nous, cherche néanmoins à établir un mode de traitement conservateur pour les cas les plus favorables de dacryocystite chronique. 'Le segment des voies lacrymales,' dit-il, 'qui présente les plus remarquables altérations et les plus graves conséquences étant le canal nasal, c'est à cette partie que nous nous attaquerons surtout.' Et Tartuferi, sur cette idée, fait construire des curettes tranchantes spéciales, fenêtrées ou non, terminées par une petite olive-guide, avec tige flexible constituée par une bande en acier roulée en une spirale à l'intérieur de laquelle est un fil d'acier qui constitue le prolongement de la curette vers le haut. Les curettes ont différents calibres (1, 2, $2\frac{1}{2}$, 3 millimètres) et leur longueur est de 6 à 10 millimètres.

La technique est la suivante :

1° Incision du canalicule lacrymal supérieur et section sous-cutanée du ligament palpébral interne avec le petit couteau de Weber, comme dans la méthode de cet auteur.

2° Cathétérisme avec des sondes de Bowman No. 4, 5, 6, pour préparer le chemin.

3° Curetage du canal nasal avec des curettes spéciales.

4° Irrigations antiseptiques avec la seringue d'Anel ($HgCl_2$, formol 1 ‰).

En mars 1910, Elschi recommande encore, dans les cas anciens de dacryocystite chronique, l'élargissement du canal nasal, suivi de son curetage, la suite du traitement devant consister en lavages au nitrate d'argent.

En mars 1910 aussi, Goebel, reprenant la méthode préconisée par Kalt en 1899, recommande le drainage du canal nasal au moyen d'un fil de soie qu'il introduit de haut en bas.

Arrivons maintenant aux *méthodes qui ont comme but final la destruction des voies lacrymales.*

Les moyens de destruction des voies lacrymales comprennent :

1° Des *agents chimiques,* par exemple les crayons au chlorure de zinc employés avant 1899 par Maisonneuve comme caustique profond destiné à détruire la muqueuse altérée, par exemple les perles de nitrate d'argent fondu employées dans le même but, etc.

Ces moyens chimiques sont mauvais. Ils provoquent des eschares qui entretiennent de longues suppurations ou amènent des péricystites phlegmoneuses. Et puis le traitement est d'une longueur désespérante.

2° *Le curetage suivi de thermocautérisation.* C'est le traitement de choix des péricystites phlegmoneuses, mais non des dacryocystites chroniques, à cause de la lenteur de la cicatrisation et des résultats plastiques parfois peu favorables que celle-ci entraîne. Néanmoins, le résultat fonctionnel est habituellement excellent. Et, à ce propos, nous

avons remarqué que le larmoiement est infiniment moindre, après un curettage suivi de thermocautérisation dans les cas de péricystite phlegmoneuse qu'après l'extirpation aseptique du sac, dans les cas de dacryocystite chronique (nous avons de même maintes fois constaté que lorsqu'on a de la suppuration après une extirpation de sac lacrymal, le résultat fonctionnel est meilleur). C'est probablement dû au fait de la destruction plus complète des filets nerveux qui se rendent aux voies lacrymales, ce qui supprime l'hypersécrétion réflexe des larmes, d'où l'atrophie progressive rapide de la glande lacrymale.

3° *L'extirpation du sac lacrymal avec réunion par première intention.* C'est, d'après nous, le traitement de choix des dacryocystites chroniques, avec ou sans ectasie du sac lacrymal.

Dans le service d'ophtalmologie de l'hôpital Saint-Jean, à Bruxelles, M. le Dr Coppez père, ancien chef de service, et M. le Dr Gallemaerts, son successeur, sont partisans convaincus de l'extirpation du sac lacrymal.

La méthode employée à l'hôpital Saint-Jean est celle que M. le Dr de Ridder, adjoint du service, a indiquée lors de la réunion du 27 avril 1901 de la Société belge d'Ophtalmologie.

On a cependant essayé dans le service une petite modification à cette méthode, sous forme d'injections de paraffine dans le sac avant son extirpation, mais cela a été sans succès appréciable.

Valude, lui, en juillet 1902, a recommandé, préalablement à l'extirpation du sac, des injections de blanc de baleine stérilisé, qui fond à 49° (seringue de Pravaz maintenue dans de l'eau chaude) et est parfaitement dur à la température du corps. Mais ces injections ne sont pas indispensables.

On emploie dans le service l'anesthésie générale au chloroforme, mais en Allemagne beaucoup d'oculistes préfèrent l'anesthésie locale sous forme d'injections sous-cutanées de cocaïne à 1 pour cent ou de stovaïne à 2 pour cent. H. G. Stutzer, en juin 1907, a même fixé un nouveau mode d'anesthésie locale de la région du sac lacrymal. Il emploie une solution de cocaïne à 0,5 ou 1 pour cent dans du sérum physiologique et ajoute à 1 centimètre cube de cette solution une goutte d'adrénaline-takamine. Il se sert également d'eusémine, produit spécialisé en ampoules de 1 à 2 centimètres cubes, chaque centimètre cube contenant 0 gr. 0075 de cocaïne et 0 gr. 00005 d'adrénaline. Il fait d'abord une injection sous-cutanée dans la région du sac. Dix minutes après, il insensibilise le nerf frontal interne (infra-trochléaire) en faisant une injection sous la poulie du muscle grand oblique, devant le périoste. Puis il insensibilise les rameaux internes du nerf sous-orbitaire en faisant l'injection au devant du périoste à la crête lacrymale. Quinze minutes après il commence l'opération.

La méthode de M. le Dr de Ridder, sous anesthésie générale, comprend les diverses phases suivantes :

Expression du contenu du sac.

Lavage du sac à l'aide d'eau boriquée.

Désinfection de la région opératoire.

Incision cutanée, de 1½ cm. de longueur, commençant à 2, 3 millimètres au-dessus du ligament palpébral interne et se dirigeant en bas et un peu en dehors, en décrivant une légère courbure à concavité externe ; cette incision est faite de préférence un peu en dehors de l'apophyse cristalloïde, parce que, si on la fait en dedans elle s'accompagne d'une hémorragie abondante, parfois incoercible.

Mise à découvert du ligament palpébral interne. Sa section.

Dégagement du sac de ses diverses adhérences. D'abord, dégagement de sa surface antéro-externe (au contraire, Valude, Kuhnt et d'autres auteurs conseillent de procéder au dégagement du sac de dedans en dehors, en commençant par la fossette lacrymale). Puis dégagement de sa surface postéro-interne, limite osseuse, au moyen d'une petite rugine courbe spéciale.

Enlèvement du sac.

Curetage, le cas échéant, du canal nasal (stylet d'abord enfoncé dans le canal nasal pour en fixer l'état de perméabilité).

Arrêt des hémorragies.

Drainage à l'aide d'une petite mèche de gaze iodoformée (24 h.).

Suture endermique au fil de soie terminé par deux plombs, un à chaque bout.

Pansement sec bien compressif.

Le lendemain, enlèvement de la mèche et renouvellement du pansement.

Quatre, cinq jours plus tard, enlèvement du fil de soie.

Le résultat opératoire est toujours excellent.

Le résultat fonctionnel est généralement avantageux, en ce sens qu'il y a diminution progressive de larmoiement. Si celui-ci continue, on fait l'extirpation de la glande lacrymale palpébrale.

A ce propos, Salva, de Grenoble, conseille d'enlever toujours la glande lacrymale palpébrale en même temps qu'on pratique l'extirpation du sac.

Et Camille Fromaget, de Bordeaux, conseille, lui, ce qu'il appelle le ' complet lacrymal ', c'est-à-dire l'extirpation du sac, le curetage du canal nasal et l'ablation de la glande lacrymale palpébrale en même temps.

Pour ce qui nous concerne, nous croyons qu'il vaut mieux attendre une diminution naturelle du larmoiement par atrophie progressive de la glande lacrymale, quitte à recourir ultérieurement à l'ablation de celle-ci si l'épiphora ne s'amende pas.

Il ne nous reste plus qu'à signaler la technique récemment communiquée par Jocqs à la Société d'Ophtalmologie de Paris (réunion de janvier 1906). Cet auteur, après l'incision cutanée, met à découvert la paroi antérieure du sac et l'ouvre verticalement. Il bourre le sac au moyen d'une boulette de coton hydrophile mouillée et exprimée. Puis, une pince hémostatique saisissant les deux lèvres du sac incisé,

celui-ci est facilement disséqué, puis enlevé. Jocqs passe ensuite une sonde dans le canal nasal, sans le curetter. Et il ne tamponne pas la cavité du sac après l'opération ; mais pour éviter toute hémorragie, il met un tampon en forme de coin, afin de pouvoir exercer une forte pression.

Cette technique de Jocqs est absolument logique et a certes pu lui donner d'excellents résultats.

Nous avons ainsi terminé notre revue des diverses méthodes thérapeutiques utilisées dans le traitement de la dacryocystite chronique.

Catégorie VIII

Un cas d'ectasie enkystée du sac lacrymal

Cr..., 29 ans, employé de commerce.

Le 24 avril. — Depuis 5 ans, larmoiement à gauche, avec écoulement de pus par les points lacrymaux lors d'une pression, même légère, sur la région du sac ; mais depuis près d'un an plus rien ne sort ; par contre, gonflement progressif de la région du sac, surtout depuis 4 mois. Actuellement, au niveau de l'angle interne de l'œil gauche, tumeur volumineuse, de la grosseur d'une noisette. Peau mince mobile sur cette tumeur. Celle-ci occupe nettement la loge du sac lacrymal. Elle est fluctuante. Elle est assez mobile sur le plan périosté. Le point lacrymal inférieur est à sa place, et agrandi (Bowman ancien).

Le point lacrymal supérieur, lui, est dévié. Tous deux sont impraticables. La pression exercée sur la tumeur ne fait sortir son contenu ni par les points lacrymaux, ni par le nez. Épiphora intense, baie étendue centrale de la cornée. Conjonctives très rouges, irrégulières, abimées, (interventions diverses).

Diagnostic : ectasie enkystée du sac lacrymal gauche.

Ponction aseptique qui permet de retirer un mucus épais, visqueux, opalescent. *Cultures sur agar* : quelques colonies de staphylocoques dorés.

Traitement : Extirpation en masse, sous chloroforme.

Cicatrice opératoire un peu prééminente, mais solide.

Diminution progressive de l'épiphora.

Ce cas ne nous arrêtera pas longtemps. Il constitue ce qu'on appelle l'ectasie enkystée du sac lacrymal. Cet état survient lorsqu'au cours d'une dacryocystite chronique un sac d'abord simplement ectasié perd à un moment donné toute communication naturelle avec l'extérieur par oblitération des canalicules lacrymaux et du canal nasal. Dans ces conditions une pression exercée sur la ' tumeur ' ne parvient plus à l vider. L'ectasie enkystée peut atteindre un volume considérable variant du volume d'un pois à celui d'une noisette ou même d'un œuf de pigeon (*Bulletin et mémoires de la Société française d'Ophtalmologie*, 1904, p. 212. Ectasie géante du sac lacrymal, par Étienne Rollet, de Lyon).

La bactériologie de l'ectasie enkystée du sac lacrymal est variable ; mais généralement la virulence des microorganismes en cause est peu intense. Mazet a trouvé, dans un cas, du staphylocoque doré pur, dans un autre cas du staphylocoque doré associé au bacille massué, dans un troisième cas du staphylocoque doré associé à la sarcina lutea.

Dans notre cas nous avons trouvé du staphylocoque doré peu abondant.

Comme traitement la seule chose à faire est l'extirpation du sac ectasié-enkysté, avec réunion par première intention.

ÉTUDE DE L'HISTOLOGIE PATHOLOGIQUE DU SAC LACRYMAL DANS SES RAPPORTS AVEC LE CANAL NASAL ET LA PITUITAIRE

Normalement, la muqueuse du sac lacrymal et du canal nasal présente les caractères histologiques de la pituitaire : une couche profonde ou chorion fortement infiltrée de corpuscules lymphoïdes ; une couche superficielle, épithéliale, formée de cellules cylindriques à cils vibratiles, qui s'amincissent à leur extrémité basale et interceptent ainsi entre elles des espaces libres dans lesquels s'amassent des cellules plus petites et de forme sphérique, les cellules basales, qui sont de même nature que les cellules basales de la pituitaire.

A la partie inférieure du canal nasal se trouvent des glandes muqueuses analogues à celles de la pituitaire.

Sur le sac lacrymal il n'y a que des veinules sans importance. Mais au niveau du canal nasal il y a, entre la couche épithéliale et le périoste, un lascis veineux abondant, amas de canaux toujours béants et gorgés de sang très rapprochés les uns des autres et reliés entre eux par de nombreuses anastomoses, rappelant par sa disposition le tissu érectile qui existe sur le cornet inférieur et avec lequel du reste il se continue et se confond au niveau de l'orifice inférieur du canal nasal. Ainsi le réseau veineux du conduit lacrymo-nasal communique en bas avec celui de la pituitaire.

Bref, nous voyons qu'il existe normalement une similitude histologique parfaite entre la muqueuse du sac lacrymal, du sac nasal, et de la pituitaire.

Et bien, *pathologiquement*, cette similitude histologique persiste.

Nous nous en sommes convaincu de la façon suivante : chaque fois qu'une extirpation du sac lacrymal a pu être faite dans de bonnes conditions opératoires, nous avons recueilli le sac en vue de l'examen histologique ; et de plus, dans chacun de ces cas, nous avons prélevé un petit fragment de la muqueuse au niveau du méat inférieur. Et nous avons inclu le sac et ce fragment de muqueuse dans la paraffine. Voici du reste exactement la *technique* que nous avons *suivie pour nos examens histologiques* :

A. — Fixation. *Passage dans les alcools successifs jusqu'à l'alcool absolu. Pénétration par une essence. Inclusion dans la paraffine.*
Diverses méthodes utilisées :

(1) *Formol* à 10 pour cent, 24 h.
Alcool à 70, alc. à 90, alc. absolu.
Xylol ou toluol.
Xylol ou toluol-paraffine.
Paraffine pure fusible à 58.

(2) *Liquide de Boin* (Formol-picrique).

24 h. ds.	Ac. picrique sol. aqueuse	70
	Formol	20
	Ac. acétique glacial	5

Alcool à 70 — carbonate de lithine.
Alcool à 70 pur, alcool à 90, etc. (comme dans 1).

(3) *Liquide de Zenker.*

24 h. ds.	Sublimé	5
	Bichromate de K.	2.50
	Sulfate de Na.	1
	Eau distillée	100

N.B. — Au moment de l'usage ajouter 5 pour cent d'acide acétique glacial.

C'est donc la liqueur de Müller additionnée de 5 pour cent de sublimé corrosif et de 5 pour cent d'acide acétique glacial au moment de l'emploi.

Lavage à l'eau courante, quelques heures.
Alcool à 70 iodé.
Alcool à 70 pur, alc. à 90, etc. (comme dans 1 et 2).

B. — Confection des Coupes (10) *et leur collage en série sur porte-objets au moyen d'eau albumineuse* (1 pr. 40).

C. — Coloration. Diverses Méthodes utilisées.
(1) *Hématoxyline-éosine.*
Dissolution de la paraffine des coupes par l'essence de térébenthine, alc. absolu, alc. à 90, alc. à 70, H_2O, hématoxyline (temps variable) eau de source, éosine, H_2O, alc. à 70, alc. à 90, alc. absolu-térébenthine-baume de Canada.
(2) *Orcéine,* suivant deux procédés :
Premier procédé, 2 solutions :

Sol. 1 :	Orcéine	1
	Alc. à 95	200
	Eau distillée	50
Sol. 2 :	Ac. chlorhydr. conc.	1
	Alc. à 95	200
	Eau distillée	50

Dans verres de montre, à 10 gouttes de la Sol. 1, addition de 5, 6, 7, 14 gouttes de la Sol. 2.

Coupes collées sur couvre-objets placées dans ces différents mélanges pendant 24 h., ce qui permet de voir quel mélange est le meilleur pour la coloration. Coupes montées à la glycérine.

Deuxième procédé (Unna-Taenzer).

Avec matériel fixé dans le formol à 10 pour cent ; coloration des coupes, 24 h., dans :

Orcéine	1
Ac. chlorhydr.	,,
Alc. absolu.	100

Lavages dans alc. à 70. — Décoloration partielle de ce qui n'est pas fibres élastiques dans :

Ac. chlorhydr.	0·5
Alc. à 95	100
Eau distillée	25

Lavage à l'eau. Déshydratation progressive dans la série des alcools jusqu'à l'alcool absolu-xylol-baume de Canada.

(3) *Mélange tétrachrome* (méthode de Gabriel Delamare).

Matériel fixé par le liquide de Boin. Noyaux teints en violet, protoplasme et fibres musculaires en jaune, fibres conjonctives en rose, fibres élastiques en brun plus ou moins noir.

Pour obtenir le mélange tétrachrome, verser dans un tube de Borre parties égales des deux solutions suivantes :

Sol. 1 :	Orcéine de Grubler . . .	1 gr.
	Ac. chlorhydr.	1 cm^3
	Alc. absolu	150 cm^3
Sol. 2 :	Hématoxyline de Bohmer . .	4 cm^3
	Fuchsine acide de Grubler .	1 cm^3 sol. aq. sat.
	Ac. picrique (sol. aq. sat. à chaud)	200 cm^3.

Coupes dans le mélange pendant vingt minutes à 45°. Puis lavages dans de l'eau légèrement acidulée (5 gouttes d'HCl pr. 100 cm^3 d'H_2O). Rapide passage dans l'eau de source, puis dans la série ascendante des alcools jusqu'à l'alcool absolu.

Térébenthine. Baume de Canada.

Notre Technique étant exposée, nous allons passer en Revue les Résultats que nous avons obtenus

Nous pouvons classer nos examens histologiques en cinq catégories :

1° Dacryocystite hypertrophique correspondant à une rhinite hypertrophique.

2° Dacryocystite atrophiante (en voie d'atrophie) correspondant à une rhinite atrophiante.

3° Dacryocystite atrophique (ozéneuse) correspondant à une rhinite atrophique.

4° Dacryocystite chronique avec ectasie prononcée du sac lacrymal.

5° Ectasie enkystée du sac lacrymal.

1° *Stade hypertrophique*

Muqueuse. — La surface interne, au lieu d'être lisse et régulière comme à l'état normal, est caractérisée par la formation de plis constituant de vraies villosités.

L'épithélium, considérablement épaissi, comprend par endroits 15, 20 assises cellulaires dont les superficielles sont formées de cellules cylindriques allongées à noyaux ovalaires, et les profondes, de cellules plutôt arrondies à noyaux également arrondis. Toutes ces cellules épithéliales ont subi une tuméfaction trouble plus ou moins marquée : leurs noyaux sont presque incolores, leur protoplasme est chargé de granulations fines nombreuses.

On remarque parmi les cellules de la première assise de nombreuses cellules caliciformes ; de plus, il y a des anfractuosités, des invaginations véritables de la muqueuse du sac tapissées de cellules caliciformes ; ce sont là des formes pathologiques singulières qu'on pourrait prendre pour de vraies glandes muqueuses.

La limite de séparation entre l'épithélium et le chorion est restée assez nette. Parfois néanmoins des cellules lymphoïdes du chorion pénètrent, à travers les cellules épithéliales, vers la cavité du sac se continuant en elle avec les corpuscules du pus.

Le chorion, hyperplasié, présente une infiltration lymphoïde considérable, surtout marquée au niveau des villosités, où elle constitue des amas nodulaires plus ou moins distincts.

Ce sont ces amas leucocytaires que, le premier, Joerss a décrits. Divers auteurs les ont ensuite considérés comme des granulations trachomateuses.

Hertel signale qu'ils font penser de suite aux follicules lymphatiques.

Rochon-Duvigneaud, lui, admet nettement que ce sont des follicules lymphatiques ; et que ceux-ci seraient même des éléments constitutifs normaux du sac lacrymal chez l'adulte.

Mais quant à ce dernier point, Tartuferi, dans ses intéressantes ' Recherches anatomo-pathologiques relatives aux dacryocystites catarrhales et purulentes chroniques ' pretend que ces follicules lymphatiques n'existent qu'exceptionnellement dans les voies lacrymales normales de l'adulte.

Sous-Muqueuse. — Réseau veineux beaucoup plus développé que normalement. Dans les gaines péri-vasculaires infiltration considérable de tissu lymphoïde. Nombreuses cellules rondes également entre les fibres de tissu conjonctif de la couche périphérique de la paroi du sac.

2° *Stade en Voie d'Atrophie*

Muqueuse. — La surface interne est irrégulière et présente de nombreux replis.

Épithélium : Offre 3, 4 assises cellulaires seulement. Cellules superficielles cylindriques à noyaux ovalaires. Cellules profondes polymorphes à noyaux plutôt arrondis. Nombreuses cellules caliciformes.

Membrane basale : Généralement assez nette.

Chorion : Abondante infiltration lymphoïde diffuse.

Sous-Muqueuse : Abondant réseau veineux. Guère de fibres élastiques.

3° *Stade atrophique*

Muqueuse : Surface interne lisse.

Épithélium : Transformation de la couche épithéliale cylindrique en un épithélium plat pavimenteux.

Membrane basale : Généralement disparue.

Chorion : Infiltré par des cellules rondes.

Sous-Muqueuse : D'importance réduite. Plus de fibres élastiques.

4° *Dacryocystite chronique avec ectasie prononcée du sac lacrymal*

Muqueuse : Nombreux replis.

Épithélium : 3, 4 assises cellulaires dont la couche superficielle est formée de cellules cylindriques allongées à noyau ovalaire, et les autres couches de cellules arrondies à noyaux arrondis.

Replis de la muqueuse tapissés de cellules caliciformes.

Membrane basale : Généralement disparue.

Chorion : Infiltration lymphoïde diffuse, et par paquets de cellules rondes au niveau des replis.

Sous-Muqueuse : Réseau veineux développé. Cellules rondes nombreuses. Disparition plus ou moins parfaite des fibres élastiques.

5° *Ectasie enkystée du sac lacrymal*

Muqueuse : Peu de replis.

Épithélium : Réduit à 2, 3 couches de cellules dont les superficielles seules sont cylindriques, à noyau ovalaire tout à fait à leur base, et en voie de dégénérescence graisseuse plus ou moins importante (granulations graisseuses). Nombreuses cellules caliciformes interposées, véritables glandes unicellulaires mucipares, avec leur bouchon de mucus plus ou moins détaché.

Membrane basale : Habituellement disparue.

Chorion : Épaisseur réduite. Infiltration lymphoïde diffuse.

Sous-Muqueuse : Réseau muqueux peu développé. Disparition complète des fibres élastiques.

Voilà, schématisées autant que faire se peut, les cinq catégories histologiques que nous avons pu établir dans nos cas de dacryocystites.

Ces notions nous seront du reste bientôt d'une grande utilité, lorsque nous devrons déterminer la classification des affections du sac lacrymal, qui nous paraît la plus logique et la plus scientifique, en tenant compte, et de nos recherches bactériologiques, et de la fixation exacte de l'état histologique de ces affections.

Conclusions d'Ensemble

Nos recherches relatives à l'étude des rapports bactériologiques entre les affections nasales et les affections des voies lacrymales, soutenues par notre étude de l'histologie pathologique du sac lacrymal dans ses rapports avec le canal nasal et la pituitaire, nous permettent de déclarer, mal établie, la classification habituelle des affections inflammatoires des voies lacrymales (nous laissons de côté leurs traumatismes et leurs néoplasmes) généralement admises par les auteurs, et d'en proposer une nouvelle basée sur la connaissance exacte des microorganismes infectants et de l'état histopathologique du sac.

Les auteurs divisent les affections inflammatoires du sac lacrymal en deux groupes :

(*a*) *Dacryocystite simple.* — Synonymie : dacryocystite catarrhale ou catarrho-purulente, ou chronique, ou blennorrhée du sac.

(*b*) *Dacryocystite aiguë.* — Synonymie : dacryocystite phlegmoneuse, tumeur lacrymale enflammée, phlegmon du sac lacrymal.

Cette division en deux groupes est basée uniquement sur l'aspect extérieur de l'affection, ou sur les caractères macroscopiques de l'écoulement, mais ne tient compte, ni de l'état histologique, ni de la nature bactériologique de l'affection. En somme, à donner notre avis franc, cette classification est la première qui se présente à l'esprit et la plus facile, certes, mais aussi la moins scientifique.

C'est pourquoi *nous proposons de fixer comme suit la classification des affections inflammatoires du sac lacrymal :*

1° *La péricystite phlegmoneuse.*

(*a*) A streptocoques, d'origine nasale, surtout fréquente chez les adultes.

(*b*) A bacilles de Koch, sous la dépendance d'une ostéo-périostite tuberculeuse sous-jacente, surtout fréquente chez les enfants.

2° *La dacryocystite tuberculeuse primitive* ou consécutive à une tuberculose conjonctivale, ou nasale (à bacilles de Koch).

3° *La dacryocystite aiguë à streptocoques* d'origine nasale au cours d'une dacryocystite chronique à pneumocoques.

4° *La dacryocystite subaiguë à diplobacilles Morax-Axenfeld* d'origine nasale, purs ou associés à des pneumocoques mais prédominants.

5° *La dacryocystite chronique à pneumocoques de Talamon-Fraenkel* généralement purs, mais parfois associés aux staphylocoques (aureus, albus, citreus), ou au Bacterium Coli (surtout chez des enfants, des vieillards, ou des malades mal soignés) plus rarement avec bacille pyocyanique,

gonocoque, bacille du xérosis, bacillus subtilis, pyogenes fœtidus, proteus vulgaris, bacille de la morve, bacille de Weeks, etc.

(*a*) Sans ectasie marquée du sac lacrymal (stade hypertrophique).

(*b*) Avec ectasie prononcée du sac lacrymal (stade de disparition progressive des fibres élastiques).

6° *La dacryocystite ozéneuse à diplocoques de Lœwenberg*, d'origine nasale (stade en voie d'atrophie et surtout stade atrophique).

7° *La dacryocystite syphilitique à spirochètes de Schaudinn*, parfois primitive (très rarement) presque toujours consécutive à de la syphilis nasale.

8° *La dacryocystite diphtérique, à bacilles de Lœffler*, complication fréquente de la diphtérie nasale.

9° *L'ectasie enkystée du sac lacrymal* (à staphylocoques dorés purs ou associés). Disparition complète des fibres élastiques.

Nous ne croyons pas nécessaire d'exposer encore en détail les caractères cliniques, histopathologiques, bactériologiques de chacune de ces divisions de notre classification, attendu que nous l'avons déjà fait au cours de l'examen des diverses catégories de notre travail.

Notre classification n'est que la conséquence, la conclusion logique de nos recherches et de nos études. Nous émettons le vœu qu'elle soit adoptée, parce qu'elle est la seule reposant sur des bases scientifiques stables, non seulement cliniques, mais aussi et surtout histopathologiques et bactériologiques.

SECTION XV

RHINOLOGY AND LARYNGOLOGY

INDEPENDENT PAPER

LA PITUITRINE DANS LES HÉMORRAGIES OPÉRATOIRES ET SPONTANÉES DES VOIES RESPIRATOIRES

Par M. le Professeur CITELLI, de Catane

L'extrait aqueux de la glande pituitaire tout récemment a été démontré très utile, spécialement dans les accouchements prolongés (pour atonie de la matrice) et dans les hémorragies de l'utérus. Cela parce que l'extrait hypophysaire a le pouvoir de provoquer une contraction progressive et de longue durée (de plusieurs heures et aussi d'un jour) des fibres musculaires lisses. Par la même action cet extrait a été trouvé utile comme galactagogue, comme diurétique et péristaltique intestinal et, par son action tonique sur les fibres musculaires cardiaques, a été trouvé utile pour combattre le shock opératoire et spontané ou consécutif à maladies infectieuses. D'autre côté quelque auteur assure d'avoir aussi obtenu des bons résultats avec cet extrait dans l'ostéomalacie, l'obésité, la maladie d'Addison, la maladie de Basedow, et moi-même j'ai eu des résultats satisfaisants dans un syndrome psychique que j'ai trouvé chez quelque adénoïdien et que j'assigne à l'altération de la glande pituitaire.

Pour combattre les hémorragies, cependant, l'extrait hypophysaire avait été employé seulement contre les hémorragies utérines, peut-être parce qu'on pensait qu'il agit là plutôt indirectement (par la contraction prolongée qu'il provoquait de l'utérus) que directement sur les vaisseaux utérins. Personne n'avait jamais employé ce nouveau produit pour combattre des autres hémorragies.

Étant donnée cependant son action stimulante sur les fibres-cellules musculaires, j'ai pensé que peut-être l'extrait hypophysaire aurait pu donner de bons résultats dans les hémorragiess pontanées et opératoires des voies respiratoires; spécialement dans les résections des cornets inférieurs lesquelles peuvent, pas rarement, donner des hémorragies ennuyeuses.

J'ai expérimenté la *pituitrine* (extrait aqueux du lobe postérieur de l'hypophyse préparé en fiales stérilisées par la maison Parke et Davis) dans de très nombreuses maladies et les effets ont été plus ou moins surprenants: beaucoup supérieurs, c'est-à-dire, à ceux que je pouvais attendre.

J'ai employé la pituitrine, parmi les hémorragies spontanées, dans les épistaxis, dans les hémorragies de la bouche et du nez pour diathèses hémorragiques spécialement dus à la malaria, dans les bronchorragies. Parmi les hémorragies opératoires, dans les résections des cornets (ici avec des résultats vraiment surprenants), dans la résection sous-muqueuse de la cloison, dans les polypes nasales qui donnent beaucoup d'hémorragies, dans les opérations sur les sinus nasals, dans les tonsillectomies, etc. Dans presque tous ces cas les résultats ont été magnifiques, parce qu'en injectant la pituitrine un quart d'heure ou une demi-heure avant l'opération, pendant et spécialement après l'opération, les hémorragies habituelles dans ces cas deviennent très réduites, et on épargne beaucoup de tamponnements dans le nez.

L'action de la pituitrine dans ces hémorragies, et vraisemblablement dans les hémorragies d'autres organes (des voies urinaires, du canal intestinal, etc.) est précieuse, et sans doute supérieure aux autres produits employés jusqu'ici au même but. C'est pour cela que j'ai cru utile de vous en recommander l'usage.

Je pratique une injection sous-cutanée ou musculaire de pituitrine avant l'opération et, si le besoin l'exige, je fais une autre injection 2 ou 4 heures après, et aussi une troisième dans les 24 heures : les jours suivants on peut faire une autre injection. Pour les hémorragies opératoires une ou deux injections suffisent presque toujours.

Je n'ai jamais eu d'inconvénients par l'usage de ce précieux produit.

SECTION XV

RHINOLOGY AND LARYNGOLOGY

INDEPENDENT PAPER

TREATMENT OF DISEASES OF THE NOSE AND THROAT BY THERAPEUTIC INOCULATION, EXCLUSIVE OF TUBERCULIN AND DIPHTHERIA ANTITOXIN

By W. DOUGLAS HARMER, M.C. Cantab., F.R.C.S. England, Surgeon, Nose and Throat Department, St. Bartholomew's Hospital, London, E.C.

Mr. President,

When I received an invitation to present a paper on the treatment of diseases of the nose and throat by therapeutic inoculation, I realized that I should have great difficulty in the preparation of my subject. Nevertheless, deeply conscious of the honour bestowed upon me, I accepted the proposal, because the subject is one upon which I have been engaged for the last four years.

The greater part of the bacteriological investigation has been carried out by Dr. M. H. Gordon, of St. Bartholomew's Hospital. When I tell you that he alone has made upwards of 200 autogenous vaccines for my patients, and repeated examinations of secretions during the treatment of each case, it will give you some idea of the work which has been done.

Apart from this, however, I have given up most of my spare time to the question and set aside one day in each week during these years when nothing but vaccine cases has been seen. Great care has been observed in the selection of patients, and no treatment has been undertaken without repeated examinations.

Examination of Secretions

(*a*) *By films.* These have been stained by ordinary methods, especially Gram-fuchsin, to determine what organisms are present and to gain a rough idea of their nature and relative number. Such examinations are most important to show whether proper care has been taken in collecting the secretion, whether the organisms persist in the same number at different stages of treatment, and whether there is evidence of phagocytosis.

(*b*) *By cultures.* The collected secretion has been smeared on blood-agar tubes or plates and incubated at once. The cultures have been examined at the end of twenty-four and forty-eight hours to determine

the principal organisms. The fact that some of these grow more readily than others is important. The prevalent infection must be determined by the number and not by the size of the colonies which have grown, and by comparing the cultures with films made from the secretion. It has been found that pure cultures are very rare, but in many instances one type of organism is prevalent, especially influenza or streptococcus. These two are the commonest persistent infections of the nose. The culture more generally found is a mixed one, and in most cases only two organisms are prevalent. In many instances the same combination persists for long periods to the exclusion of everything else. This is found even with relapsing infections such as colds. To make certain of the exact nature of an infection the examinations must be repeated, and, in order that mistakes may be avoided, vaccine treatment should never be commenced until this has been done.

Mixed cultures with more than two organisms prevalent are also common in infections of the lower air-passages.

Sterile cultures are almost unknown, with the exception of chronic influenza infections, and these are extremely difficult to cultivate, and likely, therefore, to be overlooked.

Contaminations from the mouth, vestibule, skin, and air are difficult to avoid, and it is essential that the clinician should consult with his bacteriologist regarding the symptoms of every case. Our observations on these lines have convinced us that it is often possible to say beforehand what the infection is likely to be.

Blood cultures and serum diagnosis may be of great value, especially in acute infections. The opsonic index has not been determined.

Vaccines (non-sensitized). In most of my cases autogenous vaccines have been used in preference to stock preparations. The doses have varied according to the vaccine employed and the general condition of the patient. It has been found that some people were very susceptible, and that reactions, both local and general, were much more frequent than is generally supposed. When treating diseases of the upper air-passages, therefore, one must be careful of the so-called negative phase. The following is a rough guide to doses :

With Influenza the primary dose of vaccine should be						1	to	5	millions.
,,	Streptococcus	,,	,,	,,	,,	1	,,	5	,,
,,	Pneumococcus	,,	,,	,,	,,	1	,,	5	,,
,,	Coliform	,,	,,	,,	,,	1	,,	5	,,
,,	Diphtheria group	,,	,,	,,	,,	1	,,	5	,,
,,	Coryza	,,	,,	,,	,,	1	,,	5	,,
,,	Friedländer	,,	,,	,,	,,	5	,,	10	,,
,,	Catarrhalis	,,	,,	,,	,,	5	,,	10	,,
,,	Staphylococcus	,,	,,	,,	,,	10	,,	25	,,

The doses should be increased gradually if the first reaction is not too marked. At least six to eight injections should be given at regular

intervals of about a week. To get a patient rapidly under the influence of vaccine, increasing doses are given on three succeeding days (Fornet and Müller). This is a safe method when starting treatment, as it is possible to inject a small first dose and then to increase it if no reaction is obtained. With smaller doses, shorter intervals than a week are required. In many instances it is necessary to continue treatment for long periods with gradually increasing intervals, because the duration of immunity varies greatly with different patients. For instance, one person may be cured of colds with a few doses and remain immune for years; another may need treatment to be repeated every winter; rarely, a good result is obtained after a long course lasting one to two years.

I have made it a general rule to continue a vaccine treatment for so long as I was convinced that the patient was better, but a word of caution is necessary. After a course of vaccines, whether the patient is better or not, the secretion should be re-examined, because there is sometimes a definite change in the infection, and in this case it is necessary to make the vaccine correspond. There seems little doubt that some organisms are more difficult to dislodge than others, e.g. influenza, which is more persistent than catarrhalis.

Sensitized vaccines of Besredka. A sensitized vaccine is prepared in the following manner: A pure culture of the patient's organisms is suspended in saline and counted. The emulsion is then mixed with a serum taken from an animal which has been immunized against the same type of organism, and is left for twelve hours at room temperature. At the end of this period the organisms have attracted the antibodies present in the serum and are agglutinated, so that they form a thick paste at the bottom of the tube. The serum is then drawn off, and the organisms are washed with saline, so that all traces of the serum are removed. Finally the organisms are suspended in saline and put up in doses which are known. This is the so-called sensitized vaccine of Besredka, and consists of live organisms plus antibodies extracted from the serum.

Such vaccines have now been used extensively during the last ten years in animals:

(*a*) To produce immunity against infections with streptococcus, pneumococcus, plague, dysentery, tubercle, and poliomyelitis. It has been claimed that they are not so toxic as ordinary vaccines; that animals can be immunized more rapidly than with ordinary killed vaccines; that when so immunized the animals would withstand many times the fatal dose with no ill effect. As an instance, Levy and Aoki showed that after increasing doses of sensitized pneumococcus vaccine on successive days, a rabbit would withstand, ten to seventeen days later, 10,000 times the dose of living pneumococcus fatal to a control animal.

(*b*) To test their curative value, and there is evidence to show that if large doses are given early very good results can be obtained; thus the same workers injected 100,000 virulent pneumococci into a rabbit and at

the same time a sensitized pneumococcus vaccine into another part of the same animal. They found that large doses were necessary to save the animal's life : thus with 1–2 c.c. the animal died, with 4 c.c. of vaccine two out of three survived, and with 6–8 c.c. all their rabbits lived. With non-sensitized vaccines the results were not so good. These workers concluded that sensitized vaccines act better and more quickly than non-sensitized.

Up to the present time we have had only three sensitized vaccines in use, namely, streptococcus, pneumococcus, and staphylococcus, and in all of them the organisms have been killed by phenol before injecting the patient. The first dose of these vaccines may be 50–100 millions, the second 250–500, and the third 500–1000. Even with such large doses a general reaction is rare, and after a course of three or more injections the patient may tolerate very large doses of non-sensitized strains.

General Considerations

Reaction. *At site of injection.* Slight swelling and redness and, later, tenderness and stiffness of the parts injected are the normal conditions after vaccination. These signs usually disappear after forty-eight hours. It is by no means uncommon, however, for local reactions to be more severe, and acute inflammation may be caused and last for several days. Rarely, an abscess forms, but only after sensitized vaccines.

Local condition. Patients often complain that their symptoms are worse. Thus in cases of sinusitis the discharge may be definitely increased for a short time. It has been said that a sharp reaction of this kind is a favourable sign, but there is little evidence to prove it.

General condition. Lassitude, slackness, and a slight degree of malaise often follow within twelve hours of injection. The next day there may be a feeling of exhilaration, which tends to make the patient confident of success. In many people vaccines serve as a general tonic. I have found that a small dose of staphylococcus makes one energetic for several days. The same result is not obtained by injecting saline.

Dangers. What is the danger of vaccines ? The question is asked repeatedly by doctors as well as patients. Most of us have a natural dislike for germs ; a few people obstinately refuse to have them injected.

Vaccines have been accused of causing disastrous results, but the cases are difficult to find. Doubtless there is real danger, especially in acute diseases. I feel convinced that we do not yet know to what extent and to what lengths it is safe to give this treatment. I am always nervous about sending vaccines to a general practitioner who has no experience. I must confess that I have seen no case which was permanently worse after inoculation, but slight difficulties and complications are often met with. Even then it is hard to decide how much is *propter hoc* rather than *post hoc*.

It has been found that lassitude, faintness, nausea, and colds in the head are fairly common results. Epistaxis is occasionally caused, but

anything else is rare. Instances have occurred of high temperature (4 cases), mental depression (common in slight forms), inflammation of glands (2 cases), of ears (2 cases of middle-ear suppuration forty-eight hours after vaccine), of joints (1 case), of skin (especially a return of some old skin eruption such as urticaria), of nerves (2 cases of severe neuritis), of eyes (1 case of recurrent iritis), of abdominal inflammation (e.g. return of gastritis or colitis), of jaundice (1 case), which were probably due to the vaccines. I have found very few instances of later complications, but know of two cases which had a return of their phthisis, one which suffered from erysipelas, one from carbuncle, and one from double pneumonia, within three months of treatment.

Failures. Doubtless a fair proportion of failures is due to our ignorance about the whole question. We have still much to learn about the causes of air-passage infections, and it is to be hoped that vaccines will help in the solution of the problems. Until we know more, we cannot avoid giving wrong vaccines and wrong doses. We are still very ignorant about the duration of treatment which is necessary, and many people have neither the confidence nor the patience to persevere. We now know that similar vaccines may have different curative values; and, apart from such considerations, it must be remembered that in diseases of the upper air-passages the organisms concerned are chiefly situated in the secretions on the surface of the membranes, and are therefore to some extent removed from the immediate action of the blood and lymph.

Results that can be obtained with vaccines. To determine what results can be obtained with vaccines it is essential to approach the question from at least two standpoints. We want to know first whether better results can be obtained with some diseases than others, and secondly, whether vaccines of different types give variable results. If these differences can be proved it will help to solve our difficulties. Unfortunately our bacteriologists are too enthusiastic, and there are some people who seem totally unable to distinguish whether their patients are better or worse. The patients often expect too much, and say they are better, although the local condition is unchanged. It is natural to think of faith-healing, and doubtless a vaccine is as good in this respect as a bottle of medicine. Personally, I believe that vaccines have definite curative value in some of our diseases; that as a general rule they are more successful in cases with intermittent periods of good health than with persistent infections such as chronic sinusitis or bronchitis where gross changes have taken place in the mucous membranes; and that with them it is easier to improve the general health than the local condition of the patient.

Acute Septic Infections. To obtain a successful result in an acute septic infection large doses should be given early and repeated as quickly as possible. I believe that these requirements can be met better since the introduction of sensitized vaccines, because they are less likely than non-sensitized preparations to make the patient worse. In this connexior

it should be noted that Broughton Alcock, working at the Pasteur Institute under Metchnikoff, was the first to inject human beings with live sensitized typhoid organisms, and was able to demonstrate that they were harmless. They are now being tried both for protective inoculation and cure of typhoid in many parts of the world.

More recently, 12 out of 19 cases of acute streptococcus infection have been treated with killed sensitized vaccines in the wards of St. Bartholomew's Hospital by Gordon with great success. Several of his patients made rapid recoveries. Of the three that occurred in our department one was that of a man who had submitted to an operation on his septum and tonsils. Seven days later he had acute middle-ear suppuration and his drums were punctured. On the ninth day my colleague Scott performed a Schwartze operation on his mastoid, which was found to contain a pure streptococcus infection. On the eleventh day he appeared generally septic with signs of early meningitis. We were both afraid that he would die. The next day he seemed worse and 50 millions of sensitized streptococci were injected. Within a few hours he was definitely better, and sat up and ate his food. One hundred and 500 millions were given on the next two days. On the fourteenth day he was practically well. His temperature chart showed a drop of one degree after each injection, a result also observed by Gordon, who has lent me similar charts. I have very little doubt that the vaccine had saved the patient's life. (Chart 1.)

Shortly afterwards one of our house-surgeons was ill with acute inflammation of tonsils and pharynx. The infection was very severe and was followed by cellulitis of neck and laryngeal obstruction. On the third day his neck was incised and tracheotomy and further incisions were necessary four days after. Sensitized streptococcus vaccines (50, 100, 500 millions) were given on the fifth, sixth, and seventh days. The temperature gradually fell and was normal seven days later. The tracheotomy tube was removed on the twelfth day. Although this case is not so convincing as the former, the recovery was very good and probably the vaccine helped. (Chart 2.)

A third case, under my colleague West, had a radical mastoid operation with drainage of extra-dural abscess. Four days later the lateral sinus was explored, but was not thrombosed. The next day the cerebellum was explored, but no abscess found. Three days later streptococci were present in the blood, and he was given sensitized vaccines in doses of 50, 100, and 500 millions. The next day temperature was normal, and rapid recovery ensued. (Chart 3.)

In this connexion West informs me that he has treated two other ear cases, both suffering from lateral sinus thrombosis and desperately ill, with anti-streptococcic serum and sensitized vaccine, with no effect. Both patients died. It is yet too early, therefore, to say how much these inoculations will help us in the treatment of acute infections of the upper respiratory tract.

Serum. Anti-streptococcic serum is constantly used in our wards and

Chart i

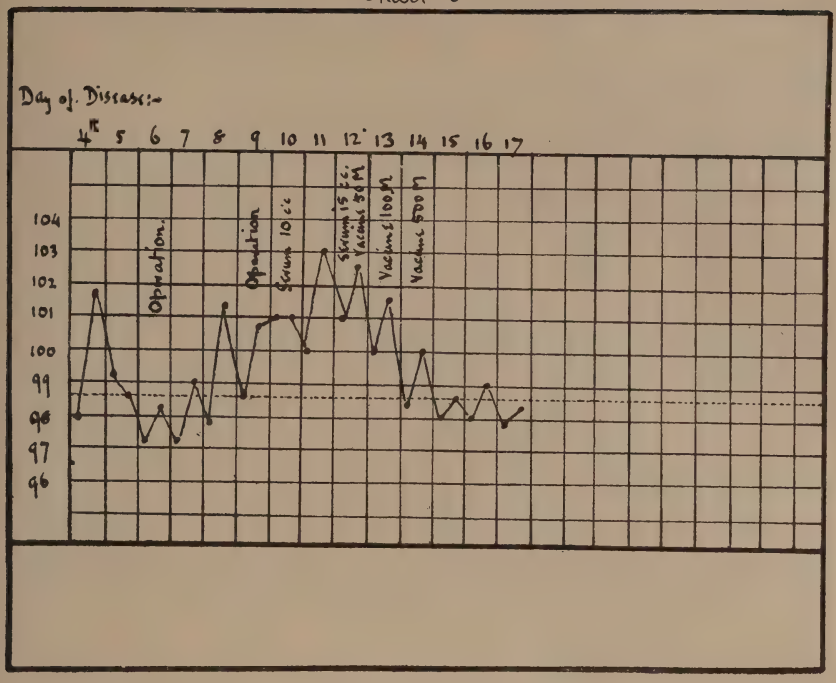


Chart ii

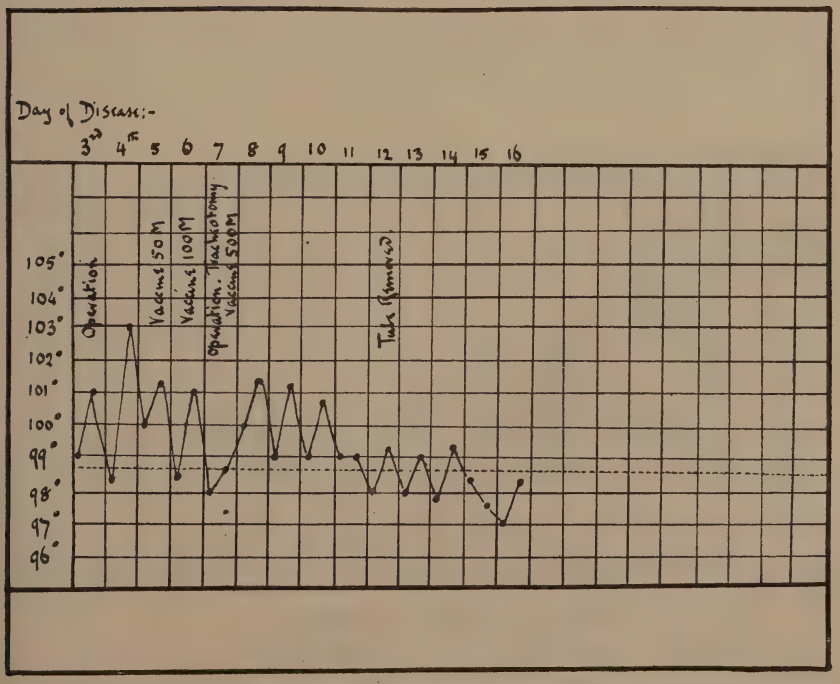


Chart III

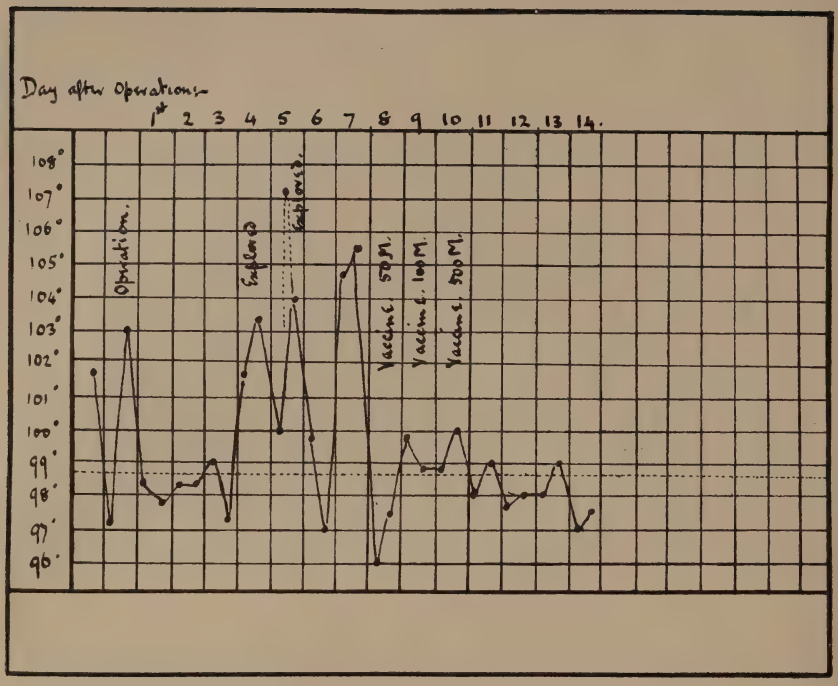
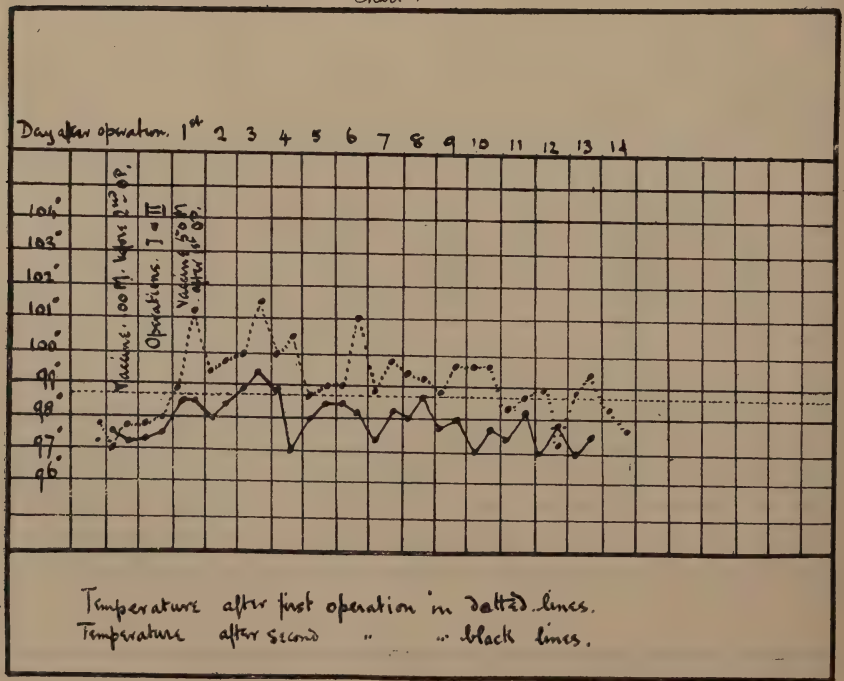


Chart IV



Temperature after first operation in dotted lines.
Temperature after second " " black lines.

occasionally with good results. Recently six of West's ear cases, who were desperately ill and septic, were given intravenous injections of doses from 25–50 c.c. over a period of one to three days. The largest amount in any one patient was 150 c.c. Five of these cases died, the serum having no effect. The sixth improved at once after exploration of the lateral sinus and ligature of the jugular vein. This patient had two 25 c.c. doses, the first at the time of operation, and possibly the serum helped.

'Colds.' Rhinitis, Pharyngitis, Laryngitis, Tracheitis, Bronchitis

1. **Acute Infection**. I have treated a large number of these infections in the last four years, the majority with catarrhalis, pneumococcus, or influenza vaccines, or combinations of these. Coryza cases have not been so common, and other organisms have been rare.

The vaccine should be given early, if possible within the first twenty-four hours. By injecting myself with a single dose (5 millions each) of catarrhalis, pneumococcus, and influenza vaccines, as soon as the severe symptoms commenced, I have succeeded in curing my last three bad colds, and the attacks have been completely relieved in twenty-four hours. From the second day onwards vaccines must be used with great caution; after the sixth day they are less likely to upset the patient, and cases of copious discharge of ten days' duration can sometimes be cured rapidly by small doses on two or three successive days. It should be a rule never to give a large dose to a patient with a severe cold.

During the past six months there has been an epidemic of severe colds followed by irritating cough and expectoration lasting for several months. Such colds have affected adults as well as children, and have caused many a sleepless night. Sickness has been a common symptom in children, and asthmatic attacks in men. Pneumococcus and catarrhalis have been the prevalent organisms. Such cases do well with vaccines, but everything else has failed.

There seems to be abundant evidence that vaccines will often cure a cold, and the question naturally arises whether they should be used for preventive inoculation. When it is known that a severe epidemic, say of influenza, is prevalent in a town, and the facts have been proved by bacteriological examinations, the general practitioner should have his stock ready, and, as soon as one case is affected, he should not hesitate to inoculate the rest of the household. Some of his patients will be protected, others may have the disease slightly,—none of them will be any worse.

2. **Chronic Infections**. The list of 31 cases which is appended comprises patients suffering from severe intermittent infections of their upper air-passages. Such cases are common and present a remarkable similarity in their histories. The patients complain of having had colds for many years or all their life; that their attacks last for weeks and recur at frequent intervals; that they get their colds regardless of exposure,

more frequently in the winter; that they know when a fresh attack is coming because of unpleasant sensations; many of them are slightly feverish in the early stages of each attack, and some of them are suspected of tuberculosis. The fact that sinusitis never occurs in this group is of great importance and difficult to explain.

It has been found that in 29 of these cases the prevalent infection was influenza, pneumococcus, or catarrhalis; that coryza is very rare (2 cases); that Friedländer, diphtheroids, streptococci, and staphylococci are of little practical importance; that it is the rule for the same organisms to reappear with each succeeding cold; in other words, that they are very persistent. That the majority of cases fall into two main groups: (*a*) influenza, commonly combined with pneumococcus; (*b*) pneumococcus and catarrhalis, constantly found together. That vaccines have a definite curative value and give markedly better results than are obtained with cases of sinusitis, presumably because of the intermittent periods of normal health which occur in the former class.

Thus, of 14 cases with influenza infections, definite improvement was noted in 8; and still better results were obtained in the pneumococcus and catarrhalis group, 11 out of 15 receiving benefit. That the sense of smell and taste may return even in cases which have had none for many years. That 6 of these cases, a fair proportion of the whole number, were highly susceptible to vaccines and were unable to tolerate the ordinary doses. Case 5, double pneumonia two months after apparent cure, also requires careful consideration.

Post-Nasal Catarrh and Deafness. My experience of vaccines in post-nasal catarrh and deafness has been limited, but my impression is that it is easier to improve the catarrh than the deafness. Very rarely a patient will say that he has heard better for a time. West states that: 'Friedländer infections seem to do well, and catarrhalis quickly disappears . . . staphylococcus aureus seems to be a favourable case for vaccination, and streptococci certainly diminish or disappear in some cases. On the other hand the pneumococcus has so far defied my efforts . . .'

Asthma and Hay Fever. It is not within the scope of this paper to enter into a discussion on the results that can be obtained by vaccination in cases of asthma and hay fever. My experience is limited to those cases which were associated with infections of the upper air-passages, and I have found that when the rhinitis and bronchitis improved with vaccines the asthma and hay fever were less severe. There seems to be conclusive evidence that inoculations will in some instances protect a patient from asthmatic attacks, and the same is true to a less extent with the secondary infections of hay fever.

The researches of Dunbar, Noon, Freeman and Ellern on hay fever are worthy of passing notice.

Furunculosis. Four cases of furunculosis affecting the vestibule of the nose, and skin of nose and face, were treated with staphylococcus vaccines and all improved.

Glands in neck. In 1911 a patient was sent to me with numerous glands in the neck which on section appeared to be lymphadenoma. He was found to have a muco-purulent discharge in his nose and the prevalent organism was staphylococcus aureus. A vaccine was prepared and given in doses of 10 to 200 millions by Dr. C. R. Stewart, who reported in June 1913 that 'the vaccines undoubtedly relieved to a great extent the rhinitis, which was thought possibly to account for the enlarged glands. The patient also had a course of X-rays and the glands have gone down markedly but return at times.' 'He is,' I understand, 'well, and has just returned from six months work in Burma.'

When I was in America I was informed by Dr. Goodale that he had seen similar cases of lymphadenoma improved by vaccines of staphylococcus, but I have not been able to find any reports.

Sinusitis, Acute. A few cases of acute sinusitis have been treated with vaccines of streptococcus, influenza, or pneumococcus. Other infections appear to be rare, with the exception of stinking abscesses of the antrum to which reference will be made later.

Since most of these cases recover quickly, if treated early by a specialist, it is difficult to determine the exact value of inoculations. I believe, however, that streptococcus and pneumococcus infections can often be cured more quickly by vaccination than any other method, and I have no doubt that sensitized vaccines are of great value because it is possible to give a succession of large doses in a short period without affecting the general health. Influenza vaccines, on the other hand, are very depressing. They should be used cautiously and in small doses on three successive days. With this method rapid improvement has been obtained with them.

Sinusitis, Chronic. We have treated a large number of cases of chronic sinusitis with vaccines for varying periods, and of these 41 have been selected for description because they were constantly under observation before, during, and after their inoculations. In most instances there was a long history of suppuration, and other treatment such as operations had failed to cure.

It is necessary to diverge for a moment to note the following points:

Spontaneous recovery of old-standing sinusitis is rare. There has only been one instance, in a patient sent to me by Sir StClair Thomson. She had been under treatment, including vaccines, for three years. The prevalent organism in her discharge was staphylococcus, and I advised a further course of inoculations. Before treatment was begun, however, her suppuration suddenly ceased. Possibly such a result occurs more frequently than we think.

These persistent discharges were more often due to infections with influenza (11), streptococcus (9), and Friedländer (6), than other organisms.

The same organism was often prevalent in each case for long periods.

Mixed infections were more common than pure. In many of these only two organisms were prevalent at one time. In some instances the

combination changed. Influenza and pneumococcus were more persistent than any other combination.

Mixed infections with more than two organisms present in quantity were not often selected for treatment (5 cases) because the bacteriology varied with different examinations, and it was difficult to determine the exact nature of the infection.

Cases with stinking discharge in the antrum were never inoculated because they invariably did well with operations.

Catarrhalis and coryza were considered of little clinical importance because they rarely persisted long enough to require vaccines.

We have had little experience of other organisms such as diphtheroids apart from atrophic rhinitis.

We have made it a rule to use vaccines containing as few strains as possible with the idea of ascertaining whether better results can be obtained with some vaccines than others. My conclusions are as follows :

Streptococcus vaccines gave fair results. Six of nine cases were definitely improved. The same is true of Friedländer vaccines, as 3 out of 6 did well.

Of pneumococcus vaccines it is rather difficult to speak. Two patients out of six recovered, but both had recent infections. The cases with long histories were not improved at any time. It seems possible that better results may be expected by treatment with sensitized strains.

Influenza vaccines were useless. Eleven cases were treated and no good result was obtained. Such infections are most difficult to cure with any treatment ; operations are rarely successful. I regard the influenza bacillus as the worst and most persistent infection of the nasal sinuses.

Staphylococcus and coliform vaccines (2 cases each) were valueless, but all four patients had poor general health and very advanced disease in their sinuses. Lastly, of the 5 cases treated with mixed vaccines 3 seemed less susceptible to colds.

It appears, therefore, that chronic sinusitis can rarely be cured by vaccines. I am unable to explain why better results were obtained with streptococcus and Friedländer than with other organisms. Possibly these infections are less persistent.

A remarkable effect is sometimes obtained with sensitized streptococcus vaccines. A patient (Case 45) came to our hospital with purulent discharge from her antra and fronto-ethmoidal cells of many years' duration. Her antra were drained, but the discharge did not improve. She was then given vaccine, and forty-eight hours after the second dose (25 millions) the discharge completely ceased in the left nasal fossa. A third dose of 100 millions was injected in due course, and seven days later it was found that the suppuration in the right side had almost disappeared. She remained well for three months and then went to Canada.

A second case (Case 46) had had repeated operations on the septum and nasal sinuses. During the past year she had used 30 to 40 handkerchiefs regularly every week. She had headaches and very poor health.

She was suffering from a streptococcus infection of all the sinuses. After the second dose of sensitized streptococcus her discharge was suddenly reduced so that she only required one handkerchief a day. Her headaches were slightly better and the improvement lasted for nine weeks. She did not return, however, for two months, when four more doses were given with no effect. Six months later the same infection was present and the vaccine was tried again. After the second dose the discharge immediately lessened and once more her health improved. This patient is still under treatment.

There have been no other cases in which a similar result was obtained, but one cannot fail to be impressed by the effects produced in these two instances. Moreover, we have had several cases of acute streptococcic tonsillitis which were cured rapidly by sensitized vaccines. These patients stated that they felt better within a few hours of injection and their pain and temperature improved almost at once.

I must again draw attention to the great susceptibility of these patients, especially to influenza vaccines which are often depressing. It is easy to exacerbate the discharge with large doses, and there is no evidence to show that marked reactions are followed by a corresponding improvement in the sinusitis. On the other hand I know of no case which was made permanently worse by inoculations.

Atrophic Rhinitis. (Only 1 male, ages chiefly 25–35.) In the series of 14 cases treated by vaccines, nothing has been noted to suggest the true cause of this disease. The most prevalent organism was a Friedländer bacillus which was found in every case, and which grew with such ease and rapidity that other forms were overshadowed. Influenza was present in 5 cases, pseudo-diphtheria in 4, streptococci in 2, and staphylococcus aureus in only one. Almost without exception the infections appeared to be mixed and two or more organisms grew freely with each examination. In only 5 cases was a course of vaccination repeated, because the strain appeared to have changed.

The results of inoculation with the different autogenous vaccines have been completely unsatisfactory in spite of the fact that doses from 10–1000 millions were employed and treatment was continued in some cases for many months. In only two was there any real improvement in local conditions, and this was probably due to other treatment given at the same time. In most cases treatment was not continued for long periods, because there was no sign of any improvement, and eight of the patients were distinctly upset and complained of various symptoms including lassitude, colds in the head, exaggeration of headaches, faintness, sickness, and abdominal pains. It is interesting to note that gastritis or colitis was complained of by several patients, and one of the series refused to continue the treatment because it made her colitis worse. When Friedländer vaccine has been used for other conditions it has not caused similar toxic troubles. Although the series is not a large one, there has been nothing to make one believe that vaccine treatment has any curative

value in atrophic rhinitis. It seems possible, however, that cases with recent history may be more amenable to improvement than those of many years' duration; and, if the treatment had been continued for longer periods, the results might have been more satisfactory. None of the patients was really worse.

Vaccines before Operations

When an operation on the upper air-passages is followed by general septic infection of the blood or by cerebral complications the result is due in most instances to streptococcus or pneumococcus invasion. It occurred to me, therefore, that attempts ought to be made to immunize my patients before undertaking serious operations upon them.

In this connexion it is now generally recognized that many animals can be protected against infections of various kinds by protective vaccination, and I decided to make experiments to see whether similar results could be obtained in men. One of my first patients (Case 49) was a man who had eight operations in India for a persistent suppuration of the frontal sinuses. I performed a Killian operation on the right frontal sinus, and gave 50 millions streptococcus vaccine twenty-four hours later. His wound healed slowly, and the temperature remained above normal for ten days. Six weeks later a similar operation was performed on the left side, but a dose of 100 millions had been given twenty-four hours before. This wound healed rapidly and the temperature was hardly raised (Chart 4). The after-history is interesting; on the left side the result was excellent, but the right did not do well. The discharge persisted, and the patient complained of headaches from time to time. The case impressed me so much that I determined to make further investigations, and shortly afterwards another case came to me suffering from streptococcus infection of the frontal and antral cells. This patient (Case 47) was carefully prepared by vaccines previous to his Killian operation. The result was most successful, and there was rapid cessation of discharge. Since then I have made it a rule to give vaccine or serum to any patient before undertaking a major operation. Of five patients with malignant growths in the jaw or larynx, four have done uncommonly well; the fifth, a case of carcinoma of the larynx, had a septic wound and secondary hæmorrhage eight days after operation, but ultimately made a recovery.

It remains to be seen, therefore, whether it is possible to immunize a patient temporarily, and so avoid the high temperature, septic wounds, periostitis, septic discharge, and cerebral complications which are so dangerous in our department.

In conclusion, my impression is that we have already reached a point at which it is safe to say that vaccines have a definite curative value in some of our complaints. The subject is intensely complicated, and I am well aware that some of my conclusions may be wrong. There is no doubt that these problems must receive a great deal more attention before vaccines will be of great practical value to the laryngologist. It behoves

us, however, to look around, and we must not forget that striking results are being obtained in other departments of medicine and surgery. We must remember that antityphoid vaccination is to be made compulsory in the United States army. As Lt.-Colonel J. R. Kean observed, when reporting the results of treatment of 16,000 troops in Texas, by typhoid prophylaxis, 'although this force had been for more than three months exposed to heat, dust, mud, flies, and all other vicissitudes of camp life, only one case of fever had developed up to June last. The medical authorities of the army,' he said, 'are much elated at this great feat of preventive medicine. . . .'

Mr. President, I look forward in the near future to similar feats in our department.

COLDS.—INFLUENZA. 14 CASES. 8 IMPROVED.

No.	*Sex.*	*Age.*	*History.*	*Treatment.*		*No. of doses.*	*Range of doses.*	*Reaction.*	*Results and Remarks.*
				General.	*Vaccine.*				
1	♀	60	Many winters colds and bronchitis which confined her to the house most of the winter.	Medical	1911 Influenza Pneumococcus	6	5–50	0	'Slight improvement.'
					1912, Jan. Pneumococcus Catarrhalis	8	10–250	0	Three months in Egypt. Much better.
					Nov. Catarrhalis Streptococcus	8	5–100	0	'Has been much better this winter' (Dr. F. M. Rowland).
2	♀	18	6 years before two operations for adenoids. Since then constant colds and bronchitis.	Medical	1912 Influenza Pneumococcus	8	5–75	0	'Distinct improvement' (Dr. T. D. Pryce).
3	♂	25	All his life colds and bronchitis with feverish attacks lasting one week.	Medical	1911 Influenza Pneumococcus Catarrhalis	6	5–100	0	1913. 'Attacks have been less frequent and afebrile and I consider that the vaccine had a good effect in controlling subsequent attacks' (Dr. N. Raw).
4	♂	12	Since childhood constant colds and bronchitis. Operations for adenoids at 6 and 9.	Medical	1911, Sept. Influenza Pneumococcus	6	5–100	0	'Temporary improvement.'
					Nov. Pneumococcus	4	50–200	0	'Seemed better till Feb. 1912, when he had acute bronchitis for 3 weeks. Next winter in Switzerland, slightly improved on return.'
5	♀	14	5 years constant colds and cough.	Medical	1912 Influenza Pneumococcus	7	5–75	0	'Condition and general health greatly improved. Cough almost disappeared. Two months after treatment had influenza which attacked others in family. Then double pneumonia with delayed resolution. Lungs not well two months later' (Dr. H. W. P. Young).

6	♂	50	Many years iritis, 2 months colds with loss of smell and taste.	Medical	1912 Influenza Pneumococcus	4	5–25	++	'No improvement. Vaccine discontinued owing to return of severe iritis' (Dr. Croft Hill).
7	♀	35	10 years hay-fever. 3 years repeated colds and deafness.	Medical	1912 Influenza Pneumococcus	8	5–100	0	No cold for 4 months. Still has nasal discharge.
8	♀	25	Many years repeated colds and loss of smell.	Medical	1912 Influenza Pneumococcus	8	10–100	0	No improvement in next 12 months.
9	♀	18	Many years nasal obstruction and discharge. Frontal headaches. Neurotic.	Medical	1911 Influenza Pneumococcus	3	10–100	++	Extremely depressed and hysterical for a week. No improvement.
10	♀	40	2 years constant colds, with purulent nasal discharge.	Medical	1911 Influenza Staphylococcus	5	2½–100	0	'The result was very good, but seemed slow in coming. It was 2 months before her nasal catarrh began to go. She has since kept free from it (18 months)' (Dr. J. L. Martin).
11	♀	25	5 years constant colds and bronchitis with feverish attacks. Has lived in a good climate in Australia.	None	1911 Influenza Staphylococcus	7	2½–50	0	Much improved. Only one cold in 3 months although she was sight-seeing in London and went everywhere. Afterwards returned to Australia.
12	♀	20	4 years repeated colds, laryngitis and bronchitis with feverish attacks and ill-health.	Open-air Medical	1911, July Influenza Streptococcus	2 series of inoculations		++	'First series upset her and made her very ill. After an interval of 1 month, treatment continued with small doses. Definite improvement 3 months after second series. Relapsed again 12 months later for a short time' (Dr. V. G. Ward).
13	♂	50	Last three springs persistent laryngitis and poor health.	Medical	1912 Influenza Streptococcus	3	10–20	++	Extreme depression during treatment lasting 6 days after last dose. Improved quickly and had no recurrence for 14 months. Improvement probably due to medical treatment.
14	♂	70	1 year irritating cough and bronchitis. Extreme pharyngitis. Occasional asthma. Insomnia due to cough. Heart disease.	Medical	1911 Influenza	3	5–15	0	Slight improvement, but would not continue treatment. Died suddenly of syncope some months later. (Dr. R. G. Cross).

PNEUMOCOCCUS AND CATARRHALIS. 15 CASES. 11 IMPROVED.

No.	Sex.	Age.	History.	Treatment.		No. of doses.	Range of doses.	Reaction.	Results and Remarks.
				General.	Vaccine.				
15	♀	25	3 years repeated colds in spite of every kind of remedy.	None	1909 Pneumococcus } Catarrhalis }	4	10–30	0	No colds in next 3½ years.
16	♀	20	Many years troublesome colds in winter. 9 months sore throat.	Medical	1911 Pneumococcus } Catarrhalis }	10	10–100	0	'No colds during next winter, which is remarkable. Still well a year later' (Dr. Clarke Morris).
17	♀	65	All her life colds in head.	Medical	1912, Oct. Pneumococcus } Catarrhalis }	10	5–550	0	'Improved. No colds during the winter. Catarrhalis still prevalent' (Dr. E. H. White, Mr. E. B. Waggett).
					Dec. Catarrhalis	5	?	0	
18	♀	19	4 years constant colds and cough troublesome day and night.	Medical	1911 Pneumococcus	8	5–100	0	'Practically cured' (Dr. C. E. Tanner).
19	♀	48	Some months violent persistent colds in head.	Medical	1911 Pneumococcus } Catarrhalis }	4	5–25	0	Cured. No return in next 12 months. 3 months after treatment large carbuncle on back.
20	♂	50	10 years irritable cough and pharyngitis. Many years furunculosis. Has lived in South America.	None	1912 Pneumococcus } Catarrhalis }	2	10–25	0	Cough at once ceased and pharyngitis improved. Returned to South America with his vaccine.
					Staph. aureus sensitized }	4	80–1500	+	
21	♂	25	Many years severe colds involving lower air-passages.	None	1912 Pneumococcus } Catarrhalis }	5	10–100	0	Improved for a time. Afterwards colds returned, but not so badly.
22	♀	25	2 months constant irritating cough after a cold.	Medical	1913 Pneumococcus } Catarrhalis }	4	4–25	0	Cough improved. Treatment stopped on account of urticaria reappearing after many years interval (? vaccine).
23	♀	30	Many years frequent severe colds.	Medical	1910 Catarrhalis } Friedländer }	7	50–250	0	Better while under treatment. Afterwards colds less frequent and not so severe.

24	♀	18	Many years repeated colds and deafness.	Medical	1912 Pneumococcus Streptococcus Diphtheroids	?	5–520	0	'Had full course of vaccines. Caught cold after one dose. Streptococcus and diphtheroids disappeared leaving pure pneumococcus. Hearing not improved' (Mr. C. E. West).
25	♂	30	Many years nasal obstruction and colds.	May 1911 Operation on septum.	1911, June Catarrhalis 1911, Nov. Staphylococcus	8 5	25–100 25–100	0 0	Much improved. Previous to treatment had frequent attacks of malaise which made it impossible for him to continue work. These disappeared at once.
26	♀	35	Many years vaso-motor rhinitis and colds. Recently violent rhinitis.	Medical	1913 Pneumococcus sensitized	10	50–500	+	Giddiness after first four doses. Sense of smell recovered after fifth dose. 'No obvious improvement from vaccine' (Dr. H. E. Powell).
27	♂	60	Many years colds and laryngitis in winter. General ill health.	None	1912 Pneumococcus Catarrhalis	10	10–100	0	No improvement noted. General health steadily became worse. No severe cold next winter.
28	♀	30	All her life troublesome colds. 1901, 'influenza' and middle-ear discharge. Never well since.	1907 Operation on septum. 1912 Staphylococcus vaccine given elsewhere.	1912, Sept. Catarrhalis Streptococcus Dec. Catarrhalis Pneumococcus 1913, Jan. Catarrhalis Pneumococcus	10 3 9	10–100 10–25 1–5	++ ++ 0	'Slight temporary improvement.' 'After second dose (10 millions) immediately started a tremendous influenza cold which lasted for a week. Then had 25 millions. 24 hours later high temperature, pains in back and legs, swelling and tenderness in neck, chiefly in salivary glands, but also in lymphatic glands. Ill for one week.' 'Slight improvement, but still has colds frequently. Discharge not so purulent. Advised open-air treatment' (Dr. H. B. Butler).
29	♀	37	Many years frequent colds and deafness.	Medical	Pneumococcus Streptococcus	7	6	++	After last dose (6 millions) had a very severe cold, sore throat, and swollen glands in neck and axillæ. In bed for 5 days. Ill for 14 days. No improvement after.

CORYZA. 2 CASES. 1 IMPROVED.

No.	Sex.	Age.	History.	Treatment.		No. of doses.	Range of doses.	Reaction.	Results and Remarks.
				General.	Vaccine.				
30	♂	12	4 years repeated colds and sore throats.	Medical	1911 Coryza Staphylococcus	6	10–100	0	Colds completely cured. Throat still troublesome. Tonsillectomy in 1912. Since then well.
31	♀	45	4 years constant colds affecting lower air-passages.	Medical	1911 Coryza	6	10–100	0	'Did not notice improvement, but thinks that colds and throats have been less troublesome' (Dr. T. B. Lloyd). 1913, reported no better.

CHRONIC SINUSITIS. INFLUENZA. 11 CASES. NONE IMPROVED.

No.	Sinuses.	Sex.	Age.	History.	Treatment.		No. of doses.	Range of doses.	Reaction.	Results and Remarks.
					Operation.	Vaccine.				
32	Multiple	♂	50	Many years copious discharge, colds, and bronchitis.	1910 Polypi and nasal drainage of antra.	1911 Influenza	9	10–100	+	Severe colds after doses 2 and 4. Better after treatment for 18 months when colds started again. 1913, influenza still present.
33	Multiple	♀	45	5 months copious discharge. Exophthalmos.	Medical	1912 Influenza Pneumococcus	3	5–20	++	'Seemed worse, so vaccine was stopped' (Dr. K. Pretty).
34	Multiple	♂	65	Many years constant discharge and polypi, violent colds, bronchitis, and vaso-motor rhinitis.	1910 Polypi	1911 Influenza Pneumococcus Catarrhalis	7	10–50	0	No improvement.
						1912 Ditto with Coryza	4	8–16	0	
35	Antrum Fr. Ethmoid	♂	55	Many years constant discharge.	None	1909 Influenza Pneumococcus	5	8–50	0	No change. Frequent examinations to 1913. Same organisms always present.

36	Antra Fr. Ethmoids	♂	28	7 years constant discharge, violent headaches, and dizziness following influenza.	1908 Nasal drainage of antra. 1909 Curetting ethmoids. 1910 Killian. Frequent polypi.	1912 Influenza 8 months later Streptococcus sensitized	9 2	10–100 50–500	+ 0	Always upset. 5 days after second dose (50 millions) severe epistaxis. Never improved.
37	Antrum Fr. Ethmoid	♂	20	All his life nasal catarrh and sore throats.	1911 Tonsillectomy, septum. 1913 Nasal drainage of antrum. Polypi.	1911 Influenza 1913 Streptococcus sensitized	4 6	5–25 50–1500	0 +	No improvement. Went to Australia, but returned with copious nasal discharge. Arm very inflamed after third and sixth doses. No improvement.
38	Fr. Ethmoid	♀	55	Many years constant discharge.	None	1912 Influenza	5	2½–10	+	No improvement. Severe cold after last dose. Patient frequently under observation for 3 years. Always pure influenza infection.
39	Antrum	♂	40	Many years constant discharge and unfit.	1912 Nasal drainage.	1912 Influenza	6	7–25	++	Very upset by treatment. No improvement. Vaccine given because operation failed.
40	Antrum	♂	22	Many years constant discharge.	Medical	1911 Influenza	6	5–100	0	No change.
41	Antrum	♀	19	Many years nasal obstruction, discharge, headaches, and dizziness.	1911 Nasal drainage.	1911 Influenza Pneumococcus	11	10–100	0	Vaccine given because operation failed. No improvement.
42	Antrum	♀	30	1 year constant discharge and pain after septic tooth. Poor general health.	1912 Alveolar drainage.	1912 Influenza	8	5–100	++	Much upset by first three doses. Feverish after third dose. General health improved. Discharge not changed. Later pneumococci and staphylococci. Antrum drained and cured.

STREPTOCOCCUS. 9 CASES. 6 IMPROVED.

No.	Sinuses.	Sex.	Age.	History.	Treatment. Operation.	Treatment. Vaccine.	No. of doses.	Range of doses.	Reaction.	Results and Remarks.
43	Antrum	♀	40	8 years constant thick yellow discharge since operation on antrum.	1902 Alveolar drainage.	1910 Streptococcus Pneumococcus	7	15–100	0	General health improved.
						Streptococcus	6	50	0	
						Streptococcus Staphylococcus	3	75–100	+	
						Streptococcus	4	20–70	0	
						1912 Streptococcus Catarrhalis	9	25–250	0	
						Streptococcus	10	100–500	0	Discharge varied until 1912 and then ceased. No return for 12 months, when she had a severe cold and discharge returned temporarily. Cured.
44	Antrum Fr. Ethmoid	♀	39	1 year constant yellow discharge and headaches.	Aveolar drainage.	1910 Streptococcus	8	5–100	0	Cured. No discharge or headaches for 18 months after.
45	Antra Fr. Ethmoids	♀	29	Many years constant purulent discharge.	Nasal drainage of antra.	1912 Streptococcus sensitized	4	10–200	0	Cured. 24 hours after second dose discharge completely ceased on left side. 24 hours after fourth dose discharge ceased on right side. Remained well for 3 months and then went to Canada.
46	Multiple	♀	47	Many years obstruction. 1 year constant purulent discharge.	6 years before polypi and middle turbinates removed. 1 year before septum.	1912, June Streptococcus sensitized	3	50–200	0	Improved. For 9 weeks discharge practically ceased; later gradual return.
						Dec. Ditto	3	100–500	+	Sterile abscess in forearm. No improvement. Staphylococci and influenza discovered during this winter.
						1913, May Ditto	4	50–500	0	Much improved.

47	Antrum Fr. Ethmoid	♂	30	9 months constant discharge and violent headaches.	Dec. 1912 Nasal drainage. Jan. 1913 Killian.	1913 Streptococcus sensitized	4	5–50	0	Vaccines given before Killian operation. Wound healed quickly. Temperature was never higher than 99. Discharge disappeared early.
						Ditto	1	100	+	
48	Antra	♀	25	2 years constant yellow discharge.	Nasal drainage.	1912 Streptococcus sensitized	2	100–250	+	Sterile abscess in forearm. Improved. Later operation on antra and rapid cessation of discharge.
49	Antra Fr. Ethmoids	♂	35	3 years constant discharge and severe headache.	7 external operations for fistula of frontals in India. Aug. 1912 Antrum L. Nov. 1912 Killian R. Jan. 1913 Killian L. May, 1913 Antrum R.	1912, Aug. Streptococcus sensitized	5	50–1000	0	No improvement.
						Nov. Ditto	3	50–100	0	Better result with second operation than with first, see temperature chart iv. June 1913. Still has discharge on right side.
50	Antrum Fr. Ethmoid	♀	19	5 years constant discharge and severe headache.	1907 Killian. 1912 Nasal drainage of antrum followed by erysipelas.	1912 Streptococcus sensitized	3	50–250	0	No improvement. 1913, no change.
51	Multiple	♂	41	5 years constant discharge and polypi. 1 year severe bronchitis.	1909 Polypi.	1911 Streptococcus	7	10–50	+	Much upset. Discharge less for a time.
						1912 Streptococcus Influenza	4	5–25	++	Much upset. No benefit. Steadily getting worse.
						Catarrhalis	3	10–25	+	Seemed worse. Vaccines stopped. Now has chronic bronchitis and is very ill (Dr. W. R. Grove).

PNEUMOCOCCUS. 6 CASES. 2 IMPROVED.

No.	Sinuses.	Sex.	Age.	History.	Treatment.		No. of doses.	Range of doses.	Reaction.	Results and Remarks.
					Operation.	Vaccine.				
52	Antrum	♂	23	Some months thick yellow discharge and poor health.	1911 Puncture of antrum 3 times without improvement.	1911 Pneumococcus	10	25–100	+	Cured. Depressed during treatment. Slight jaundice after fourth dose (100 millions). Very neurotic.
53	Antrum	♂	59	6 months before acute suppuration in antrum followed by intermittent thick yellow discharge.	1909 Alveolar drainage.	1910 Pneumococcus Later Pneumococcus Staphylococcus Friedländer	8 3	5–25 35–150	0 0	Completely cured. Later liable to severe colds associated with antral discharge, but never for long periods.
54	Antrum Fr. Ethmoid	♂	35	9 years thick yellow discharge.	1910 Antrum	1910 Pneumococcus 1911 Pneumococcus Staphylococcus	10 Full course	10–250	0 0	No change. Vaccinated by Dr. Balfour in Egypt. No change on return.
55	Antrum Fr. Ethmoid	♀	35	When a child operations for polypi. 10 years before for antra; 5 years before for frontal R. Many years copious thick yellow discharge.	None	1910, Dec. Pneumococcus	2	100	0	Refused further treatment as no improvement. 1913, unchanged.
56	Ethmoid Sphenoid	♀	35	Many years vaso-motor rhinitis. 7 years before operations on ethmoid and sphenoid. 3 months copious sticky muco-pus.	Ethmoid Sphenoid Polypi	1913 Pneumococcus sensitized	5	5–100	0	No improvement. Blood-stained discharge 48 hours after each dose, for 2 days.
57	Multiple	♀	30	3 years thick stringy muco-pus. Severe headaches. Polypi. Frequent operations.	1912 Sphenoid	1912 Streptococcus sensitized Streptococcus sensitized 1913 Pneumococcus sensitized	3 6 2	50–200 100–1000 10–500	0 + +	1911. Influenza prevalent in secretion. Sick and ill for one week after last dose. Arm acutely inflamed. Later pneumococcus, catarrhalis, streptococcus found. Never improved. No polypi since vaccination. Case probably due to pneumococcus.

MIXED INFECTIONS. 5 CASES. 3 IMPROVED.

XV	58	Antrum	♂	27	6 years frequent colds with copious discharge.	Punctured six times.	1910, Oct. Pneumococcus Streptococcus	3	8–20	0	Nature of infection never clearly determined as cultures varied each month.
							Nov. Influenza Friedländer	2	5–15	0	Treated by stock vaccines.
							Dec. Streptococcus Staphylococcus	2	5–25	0	1913. Wrote saying, 'colds have almost entirely left me; only three slight colds in 2½ years, lasting 2 days. Formerly colds lasted 6–8 weeks.'
							Staphylococcus	1	40	0	
X 2	59	Antra Fr. Ethmoids	♀	30	Many years copious discharge and polypi. Severe headaches.	1908 Ethmoids 1909 Septum. Antra. Ethmoids. Polypi frequently.	1910 Pneumococcus	6	10–100	+	Colds after 2 and 3.
							1911 Streptococcus	9	12½–100	0	Never cured, but not so liable to colds or headache. No return of polypi after treatment. 1913, streptococci and influenza prevalent.
							1912 Influenza	9	12–100	0	
							Catarrhalis Pneumococcus	1	25	0	
	60	Ethmoid	♂	55	4 years rhinorrhœa and colds with yellow discharge. Frequent operations for polypi.		1911 Staphylococcus	5	12½–100	0	
							Streptococcus	4	10–50	++	2 hours after last dose very drowsy. Next day severe cold in head. For 3 days blood-stained discharge. For 1 week languid. Slight improvement after treatment.
							Staphylococcus Coliform	4	10–100	0	
	61	Antra Ethmoids	♂	60	In 1907 influenza followed by antral suppuration and three operations.	1910 Antra	1910 Pneumococcus	2	5–10	0	
							Pneumococcus Streptococcus	4	20–30	0	
							Pneumococcus	1	50	0	
							Staphylococcus	1	50	++	Greatly upset. Discharge much increased. Bed for 1 week.
							1911 Staphylococcus sensitized	1	7	++	24 hours later pain in axilla. Severe neuritis for 1 month.

MIXED INFECTIONS (*continued*).

No.	Sinuses.	Sex.	Age.	History.	Treatment. Operation.	Treatment. Vaccine.	No. of doses.	Range of doses.	Reaction.	Results and Remarks.
62	Multiple	♀	17	1908, tonsillotomy. Since copious nasal discharge.	1910 Turbinectomy, antrum. 1911 Polypi, antra, exenteration of ethmoid.	1910 Influenza Pneumococcus Staphylococcus 1911 Influenza Streptococcus 1912 Streptococcus sensitized Catarrhalis	2 7 2 3	10–20 25–300 100–500 10–30	0 0 0 0	Never improved. 1913. Friedländer pneumococcus prevalent.
FRIEDLÄNDER CASES. 6 CASES. 3 IMPROVED.										
63	Antrum Ethmoid	♀	21	4 years constant discharge. Rhinitis caseosa.	1907 Antrum, polypi. 1908 Antrum Ethmoid 1909 Ethmoid	1910 Friedländer	5	40–80	0	Improved as soon as vaccines were started. Completely cured.
64	Ethmoid	♂	35	5 years discharge, violent colds, and headache. Frequent attacks of 'influenza'.	1910 Ethmoid	1910 Friedländer Pseudo-Diphtheria 1911 Ditto	8 8	10–100 10–100	0 0	Improved. No severe colds or fever for three years. Discharge never cured. 1913. Friedländer and pseudo-diphtheria still prevalent.
65	Fr. Ethmoids Sphenoids	♀	38	Many years discharge and headaches.	Sphenoids	1912 Friedländer	4	100–400	0	Sinusitis completely disappeared. 5 months later still well.
66	Multiple	♀	40	Many years ozæna.		1912 Friedländer	6	25–200	+	Arm bruises after injections 2 and 5. Complained of being tired, faint, sick, and short of breath. Increase of discharge during treatment. April 1913, no change.

67	Antrum Ethmoid	♀	34	Three years nasal discharge and crusting. Troublesome headaches.	Operations: Antrum. Ethmoid. Previous vaccine treatment elsewhere.	1912 Friedländer	5	20–100	+	No improvement.
68	Antrum Ethmoid	♀	30	Two years constant discharge and crusts.	1908 Nasal polypi 1909 Antrum Ethmoid	1911 Friedländer 1912 Friedländer Pneumococcus	7 3	10–100 10–50	o o	March 1912. No improvement.

STAPHYLOCOCCUS. 2 CASES. NONE IMPROVED.

69	Multiple	♀	19	Nasal discharge. Sore throats. Wassermann negative. No T.B.'s.	1912, March Ethmoid Sphenoid April Watson-Williams	1910 Staphylococcus Streptococcus	10	50–200	o	Slight improvement.
						Staph. Aureus	4	50–100	o	Discharge distinctly worse.
						1911 Staphylococcus Streptococcus	9	10–600	+	Very sleepy after dose of 100 millions. Condition worse. Regained sense of smell and taste which had been absent for years. May 1912, death from intracranial complications.
70	Antrum	♀	55	One month copious discharge after septic tooth and alveolar drainage. Many years lupus pernio.	None	1909 Streptococcus	4	10–45	o	No change in antrum. Lupus disappeared, but patient also had X-rays.
						1910 Staphylococcus	4	25–100	o	
						1911 Staphylococcus Streptococcus	3	10–250	o	

COLIFORM. 2 CASES. NONE IMPROVED.

71	Antrum Ethmoid	♂	45	3 months thin white discharge and polypi.	Polypi	1912 Coliform	6	5–50	o	No change. 1913, still had pure coliform infection.
72	Antra Fr. Ethmoids	♂	52	Many years thin white discharge and headaches.	None	1912 Coliform	6	10–100	+	Headaches better. Discharge unchanged. 1913, no improvement.

ATROPHIC RHINITIS. 14 CASES. 2 IMPROVED.

No.	Disease.	Sex.	Age.	History.	Treatment.		No. of doses.	Range of doses.	Reaction.	Results and Remarks.
					Operation.	Vaccine.				
73	Atrophic Rhinitis	♀	33	Many years ozæna. Headaches. Sphenoid affected.	Lotions, sprays.	1911 Friedländer Pneumococcus	8	50–400	0	April 1913. No change. Says headaches were better.
74	Atrophic Rhinitis	♀	21	6 years ozæna and headaches.	Lotions, sprays.	1910 Friedländer	5	10–30	++	Headaches and malaise. Feb. 1913, no improvement.
						1911, Jan. Friedländer Influenza	3	10–500	++	
						June Ditto.	7	25–500	++	
75	Atrophic Rhinitis	♀	30	Many years ozæna and headaches. Sphenoid affected.	1907, 1909, 1910. Sphenoid Ethmoid	1910, March Friedländer Staphylococcus	6	50–400	+	Improved for a short time.
						May Friedländer	3	300–800	0	Much improved for two months.
						Nov. Friedländer Pneumococcus	5	25–200	0	March 1913. Discharge and headaches unchanged. General health good.
76	Atrophic Rhinitis	♀	32	Many years ozæna and crusting.	Lotions, sprays.	1911 Friedländer Pseudo-Diphtheria Influenza	8	10–100	+	Feb. 1912. Condition unaltered.
77	Atrophic Rhinitis	♀	30	Many years crusts and offensive discharge.	1909 Septum Sphenoid	1910, Sept. Friedländer	1	25	0	March 1912. Condition unaltered.
						Oct. Friedländer Gram-Bacillus	8	30–500	+	Depressed. Very unwell.
78	Atrophic Rhinitis	♀	26	Seven years crusts and nasal discharge. Painful throat.	Lotions, sprays.	1911 Friedländer	1	800	++	
						1911 Friedländer	6	50–150	+	Pain under eye after two injections.
						1912 Friedländer	Course repeated			April 1913. Much better.

79	Atrophic Rhinitis	♂	24	Many years ozæna and husky voice.	Lotions, sprays.	1911, May Friedländer Streptococcus	7	10–100	o	Nov. 1912. Patient says voice has been better.
						June Friedländer Pseudo-Diphtheria	Full course		o	
80	Atrophic Rhinitis	♀	16	Many years discharge and crusts.	Lotions, sprays.	1911 Friedländer Influenza	5	10–100	o	Feb. 1912. No improvement.
81	Atrophic Rhinitis	♀	23	Many years ozæna.	1909 Operation on a sinus.	1910, April Friedländer	8	100–1000	+	Improved temporarily.
						Dec. Friedländer	6	50–200	o	April 1911. Increase in discharge. Very unfit.
						1911, April Friedländer Streptococcus	4	100–200	++	March 1912. No improvement.
82	Atrophic Rhinitis	♀	26	13 years nasal discharge.	Lotions, sprays.	1911 Friedländer Influenza	9	25–150	o	Said to have had erysipelas two months after treatment. After this definite improvement for a time. January 1913, no improvement.
83	Atrophic Rhinitis	♀	25	Many years offensive discharge and crusts.	Lotions, sprays.	1911 Friedländer Pseudo-Diphtheria	8	?	o	'Began to improve six months later.'
						1912 Ditto	8	?	o	'Improved, but was very painstaking with her local treatment' (Dr. V. G. Ward).
84	Atrophic Rhinitis	♀	39	Many years ozæna and ill health.	Lotions, sprays.	1911 Friedländer	6	10–200	+	Very depressed by first three doses. General health improved. Local condition unchanged.
85	Atrophic Rhinitis	♀	35	All her life offensive discharge and crusts. Ill health and colitis.	Lotions, sprays.	1910 Friedländer Pseudo-Diphtheria	6	15–50	+	Colitis worse during treatment. No improvement.
86	Atrophic Rhinitis	♀	30	4 years offensive discharge after colds. Ill health.	Lotions, sprays.	1911 Friedländer	4	25–100	+	Felt ill during treatment. Better for one month.
						1912 Ditto	6	25–100	o	Slight improvement after second course. Note short history of ozæna.

SECTION XV

RHINOLOGY AND LARYNGOLOGY

INDEPENDENT PAPER

NOTES ON FIFTY CASES OF NASAL DISCHARGE TREATED WITH VACCINES

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The following fifty cases underwent one or more courses of treatment by vaccines. None of them have been specially selected with a view to publication here, but they form a continuous series. In some instances the condition had failed to respond favourably to other methods of treatment, while in others again, vaccines were used at the outset. In every case an autogenous vaccine was prepared. The opsonic index was not ascertained in any instance. As a rule the injection was given once each week, and the treatment was carried out over a period of six to eight weeks, sometimes longer. If employed for a longer period than this, a second swab was taken from the nose and a fresh vaccine made from the organisms then found. The first dose was as a rule a small one, the second was double the first, and the third double the second, and so on.

The fifty cases treated in this way were grouped as follows :

1. *Cases of nasal catarrh,* eighteen in number : these included (*a*) eleven cases of chronic muco-purulent catarrh with or without some degree of nasal obstruction, also (*b*) seven cases of frequently recurring colds in the head.

2. *Cases of chronic purulent rhinitis,* nine in number : in a few of these slight crusting and commencing atrophy were noticeable

3. *Cases of fœtid atrophic rhinitis* (*ozæna*), eighteen in number.

4. *Cases of accessory sinus suppuration,* five in number. In this group, with one exception, the vaccine was used only after operation as an adjunct in the after-treatment.

1. *Nasal catarrh.* In the sub-group (*a*) eleven cases, we had to deal with the class of case in which there was a more or less persistent muco-purulent nasal secretion lasting in some cases for months but in others

for a number of years. Inferior turbinal engorgement caused a varying degree of nasal obstruction. In four of the cases there was an associated sycosis of the upper lip. Accessory sinus suppuration was excluded.

In the eleven cases comprising this sub-group, previous treatment had been carried out in nine, while in two, vaccines were employed at the outset. The treatment referred to consisted mainly in nasal douching: in three cases the galvano-cautery had been used and in one a partial turbinectomy had been performed. Because this treatment, however, failed to give the patients the desired relief vaccines were commenced. The organisms found in the nasal cavities in these cases were for the most part varieties of the staphylococcus pyogenes, also the pneumococcus, the streptococcus and the Bac. tetragenus. The results in this class of case were uniformly good. After an average of eight injections, the patients without exception expressed themselves as feeling better. Fewer handkerchiefs were used. The discharge disappeared or was greatly diminished in amount. In the four cases in which there was a secondary eruption upon the upper lip, this disappeared. In three of the cases a second vaccine was found necessary, some months after the first had been discontinued, because of recurrence of the symptoms. It is possible that some of the other cases may require further vaccine treatment, but at the present time they have not applied for it.

As an example of this class of case, we may quote the following: Female, chronic nasal catarrh with exacerbations and considerable nasal obstruction for some years. A mixed vaccine of the pneumococcus and streptococcus pyogenes was commenced. After four injections, she was breathing more freely, and after eight she felt very well and had no trouble with her breathing. An intercurrent cold was not so severe as had been the case before treatment was commenced. Six months after the twelfth and last injection there had been no recurrence of symptoms.

In the sub-group (*b*) there were seven patients who complained of frequently recurring acute colds in the head. In two of the cases there was always very considerable elevation of temperature associated with the onset of the rhinitis. Sprays, douches, and even cautery treatment had been previously employed in some of them. The staphylococcus aureus and albus were the organisms most frequently met with, but the pneumococcus was also found. The vaccine was made from the nasal discharge at the time of an acute attack. There is no doubt that in these cases the acute attacks occurred with much less frequency and their severity was diminished. In four of the patients a second course of vaccines was carried out on account of recurrence of the acute catarrhal symptoms. As regards the three other patients in this sub-group, we have no notes regarding their further history. It is probable that in this class of case occasional courses of vaccine may be necessary.

2. *Nine cases of purulent rhinitis* with a tendency in some to a slight degree of crusting and atrophy. In this group of cases we had to deal with a condition of the nasal cavities which is generally recognized as

the early stage or predisposing cause of atrophic rhinitis. The cases treated may be divided into two classes, (i) three in which no improvement appeared to follow the injections. The organisms were the staphylococcus aureus and albus and a Gram-bacillus in two of them. Nasal douching for a considerable period had failed to give relief. A course of vaccines for six weeks did not seem to improve the condition. (ii) In the six remaining cases, however, distinct improvement was soon observed and in five a cure may be said to have resulted, in one case indeed, a year after the treatment, no nasal discharge could be detected. The staphylococcus aureus was present in the discharge in four cases, being in pure culture in two and combined with a short streptococcus in one and a gram-bacillus in another. The pneumococcus was present in pure culture in another case. Where more than one organism was present a mixed vaccine was given. Nasal douching had been previously tried in these cases without success.

3. *Eighteen cases of fœtid atrophic rhinitis* (*ozæna*). In the light of the collective investigation into the nature of ozæna, the use of vaccines in this group of cases was of special interest. All the cases were characterized by crusting and some degree of atrophy of the mucous membrane : the fœtid character of the discharge was not recognizable in every instance, possibly on account of the syringing treatment that was being adopted, but there was no doubt as to the exact nature of these cases. In all of them nasal douching with or without preliminary plugging of the anterior nares had been carried out for varying periods of time from a few months to several years. A certain degree of benefit naturally accompanied the douching, but relapse took place on cessation of treatment.

The cases may be divided into two groups according to the results obtained by the use of vaccines : (i) in eight cases, no benefit appeared to be derived from the injections ; (ii) while in ten others, more or less distinct improvement followed their use. It is necessary to analyse the two groups more carefully.

(i) Of the eight cases not benefited, the Bac. mucosus ozænæ (Abel) was cultivated and used in the vaccine in six, in pure culture in three, and associated with a staphylococcus in two, the Bac. proteus in one, and the Bac. pyocyaneus in one. In the remaining two cases mixed staphylococcal forms alone were procured. In these cases the maximum number of injections was six, the results not being sufficiently encouraging to warrant their further employment.

(ii) Of the ten cases in which vaccine treatment was undoubtedly followed by improvement, the Bac. mucosus ozænæ (Abel) was cultivated and employed in nine. In six a pure culture was obtained : in the remaining three it was associated with the streptococcus pyogenes and a diphtheroid bacillus in two, in one with the pneumococcus and staphylococcus aureus. In the tenth case a mixed culture of staphylococcus aureus and a Gram-bacillus was obtained.

In nine cases, therefore, the vaccine contained Abel's bacillus, in six being used in pure culture. The number of injections varied from six to fourteen. The dose of the Abel's bacillus varied from 50,000,000 to 500,000,000 bacilli. The improvement that was noticeable alike to the patient and the observer varied in degree but was obvious in all. In five of the cases there was less crusting, less discharge, and an almost complete disappearance of the odour and the patient felt better. In four patients the crusting disappeared and in two of them the headaches ceased. As regards the duration of this improvement, the following points were noted: two patients seen two months after the commencement of treatment showed no crusts, and no syringing was being carried out. One patient seen four months later, had no crusts or odour, but a very slight discharge. One patient seen six months later had no discharge or crusting, and one had no discharge, odour, or headaches, eight months after the last injection.

4. In this group there are *five cases of accessory sinus suppuration*. In one case, left-sided frontal and maxillary sinus suppuration, no operation was performed, but the treatment consisted in nasal douching and the administration of a staphylococcal vaccine along with a Gram-bacillus. After five injections had been given, there seemed to be some diminution in the amount of the discharge, but as the improvement was doubtful, the treatment was not continued.

In the remaining four cases, vaccines were employed after operative interference with the view of accelerating the healing. In one case of bilateral ethmoidal suppuration, after intranasal operation, a mixed vaccine of streptococcus pyogenes and staphylococcus aureus was administered. Six injections were given without improvement. In one case after the radical antral operation a vaccine was used, but it was, of course, difficult to estimate the part played by the vaccine. In a third case, in which an intranasal operation had been performed on the antrum, very little improvement followed lavage carried out for nearly three months. A staphylococcal vaccine was then used, improvement commenced and cure resulted. The fourth case was one of radical operation upon the frontal and ethmoidal cells, in which a fistulous opening persisted in the lower part of the incision for many weeks, apparently due to the persistence of some discharge from the ethmoidal region. A vaccine of the pneumobacillus was prepared and after three injections there was a marked change both in the character and amount of the discharge which gradually ceased and the wound permanently closed. Vaccines may prove a useful adjunct in the treatment of sinus suppuration after operation.

In conclusion we desire to express our sincere thanks to Dr. J. S. Fraser for permission to incorporate some of his cases in this paper and also to Dr. W. G. Porter and Dr. Ian Struthers Stewart for the use of three cases of ozæna under their care.

SECTION XV

RHINOLOGY AND LARYNGOLOGY

TREATMENT OF DISEASES OF THE NOSE AND THROAT BY THERAPEUTIC INOCULATION, EXCLUSIVE OF TUBERCULIN AND DIPHTHERIA ANTITOXIN

DISCUSSION

Mr. E. B. Waggett (London) congratulated the introducers upon their highly important communications and wished to express the special hope that they would not relax their efforts in the department of chronic septic conditions of the nasal sinuses.

The speaker had done a considerable amount of work with the assistance of expert bacteriologists some four or five years ago, upon these chronic cases. Among fifty cases carefully treated by vaccines and simple lavage, no single cure resulted, although coincident with the positive phase, as shown by the opsonic method, a sense of well-being was observed and also diminution of discharge. It was suggested that better results might be obtained by combining Bier's method of artificial engorgement with the vaccine treatment.

Sir StClair Thomson (London) congratulated the authors on their good work, although at present the positive results were few. As in other scientific researches they had to look first for light before they could expect fruit. Vaccine-therapy was alluring as it was founded on a hypothesis which appealed to every one in having an undoubted basis of truth. He had to confess that the clinical results in his experience were disappointing, even although he had transferred his patients to the hands of several skilled pathologists. In fact, his successful cases could be counted on the fingers of one hand, and were so few in proportion to the number of failures, that a wise scepticism prompted one to ask whether even these successful cases would not have done as well if left alone. The feelings of the patient were not worthy of any account: nearly all sanatorium patients expressed a great sense of *bien-être* under tuberculin treatment and yet, at the recent Congress of the National Association for the Prevention of Consumption, the majority of speakers held that, at present, tuberculin treatment was of trifling importance.

Dr. Dan McKenzie (London) referred to the caution and moderation with which Mr. Harmer had discussed the subject, for it was one which in certain quarters had been advocated with unnecessary and perhaps even unfortunate warmth.

Immunizing patients prior to operation was in the present state of their knowledge difficult to ensure, and indeed the attempt at immunization might be followed by a depreciation instead of an increase in the natural powers of defence.

The subject was only at the beginning and they were indebted to Mr. Harmer and Dr. Logan Turner for having given such a good lead in the method of presenting the case.

In contrast with Harmer's and Logan Turner's work that of others was apt to lead to a dissemination of ideas regarding vaccines which ended in the most foolish and dangerous behaviour utterly unworthy of men who had received a scientific training.

Le docteur Trétrôp (Anvers) pense que la vaccinothérapie a de l'avenir à condition que pour son emploi on fasse les distinctions qu'elle comporte. Il y a lieu de distinguer les stock-vaccins et les auto-vaccins ; ces derniers auront toujours plus d'efficacité, *a priori* puisqu'ils agiront contre les variétés microbiennes du malade lui-même. En outre, c'est dans les cas aigus que cette efficacité sera la plus grande. Une sinusite maxillaire purulente chronique sera justiciable du traitement chirurgical et non point de la vaccinothérapie. Un épanchement purulent dans une cavité osseuse se trouve en effet à distance des phagocytes de la muqueuse et échappe en grande partie à leur action. C'est le cas de tout exsudat dans une cavité et c'est ce qui a compliqué le problème de la vaccinothérapie pour le choléra et la fièvre typhoïde, par exemple. En résumé : auto-vaccins de préférence et emploi dans les cas aigus, le plus près possible de la période de début, tel semble être l'avenir efficace de la vaccinothérapie.

Dr. Lyon Smith (London) had observed, during the last three years in his treatment of rheumatic cases by inoculation, that a large proportion (about 30 per cent.) obtained a very marked immunity from the common colds to which they had formerly been subject.

The disappointing results to which the President and other speakers had referred were probably in some measure due to the failure to detect all the infecting agents. The work was very tedious and needed most persistent efforts to ensure success.

Reply

Mr. Douglas Harmer (London) agreed that at present it was necessary to be extremely careful in making statements regarding vaccines and their results. He believed that the subject needed much more research in order to be of practical value.

SECTION XV

RHINOLOGY AND LARYNGOLOGY

INDEPENDENT PAPER

CONSIDÉRATIONS SUR LES MYCOSES DES MUQUEUSES DES VOIES RESPIRATOIRES ET DIGESTIVES SUPÉRIEURES

PAR M. LE DOCTEUR E V. SEGURA, PROFESSEUR D'OTO-RHINO-LARYNGOLOGIE À LA FACULTÉ DE MÉDECINE DE BUENOS-AYRES, CHEF DU SERVICE OTO-RHINO-LARYNGOLOGIQUE À L'HÔPITAL SAINT-ROCH.

L'ÉTUDE des mycoses est toute d'actualité, aussi croyons-nous utile d'apporter à cette question notre modeste contribution.

Les observations de manifestations cutanées sont à l'heure actuelle, depuis les remarquables travaux de MM. de Beurmann et Gougerot, nombreuses et très documentées, il n'en est pas de même des altérations des muqueuses dont les cas publiés sont encore très rares.

Nos observations ont trait à des lésions des muqueuses des voies respiratoires et digestives supérieures ; elles sont au nombre de onze ; dix me sont personnelles, une m'a été très obligeamment prêtée par le docteur Gougerot.

Au point de vue clinique, les lésions ont l'aspect typique bourgeonnant de la sporotrichose ; les ulcérations sont à bords déchiquetés ; leur fond bourgeonnant sécrète du séro-pus et au niveau des fosses nasales se recouvre de croûtes épaisses. En certains endroits, on constate la présence de petites saillies folliculaires visibles sous la forme de petites taches jaunes qui ont déjà été observées au cours des conjonctivites sporotrichosiques.[1] Enfin dans un cas comparable à celui de Letulle[2], nous avons constaté une particularité pas encore signalée, c'est la destruction de la luette et d'une partie de ses piliers (obs. n° 7). Notre observation (n° 8)

[1] Burnier et Weill, 'Un cas de sporotrichose gommeuse hypodermique ulcéreuse disséminée avec localisations conjonctivales,' *Ann. d'oculistique*, mai 1909, p. 344 et *Gaz. des Hôpitaux*, 21 septembre 1909, n° 107, p. 1339.

[2] Letulle et Détré, 'Sporotrichose de la peau, de la bouche, du pharynx et du larynx,' *Bull. et Mém. de la Soc. méd. des Hôpitaux de Paris*, 28 février 1908, n° 10, p. 379 ; *Presse méd.*, 18 mars 1908, n° 23, p. 182.

de sporotrichose de l'épiglotte est tout à fait comparable au cas déjà signalé par MM. Gastou et Brodier qui avaient constaté un larynx recouvert de végétations papillomateuses.

Enfin l'observation n° 11 ayant trait à un cas de sporotrichose dégénérée en néoplasme se rapproche beaucoup d'un cas récemment publié par M. Brocq à la Société de dermatologie.

Au point de vue anatomo-pathologique et bactériologique, si la réaction de Wassermann et la cuti-réaction ont toujours été négatives, il faut bien reconnaître que rarement concluants ont été les examens histologiques et les cultures.

Jusqu'ici, dans tous les cas connus, comme dans celui de Gougerot et dans le cas de sporotrichose du pharynx et du larynx de Letulle, le sporotrichum s'était trouvé en abondance sur les lames sous la forme oblongue décrite par de Beurmann et Gougerot ; cette abondance de germes dans les lésions muqueuses, d'après Gougerot, serait même remarquable par rapport à leur rareté au niveau des lésions cutanées et dans les dermites végétantes. Il se peut d'ailleurs que, dans les cas où l'examen biopsique a été négatif, on n'ait pas fait attention aux formes incolores du parasite. Aucune autre raison ne peut être invoquée, car même en présence de germes secondaires, le sporotrichum pousse et finit par être très apparent.

Dans toutes nos observations, l'étiologie est obscure ; seules, les observations 1 et 2 sont tout à fait intéressantes, car la contagion de mari à femme est certaine ; or les cas de contagion sont exceptionnels : il n'existe dans la littérature médicale qu'un seul cas signalé ; c'est celui de Widal et Joltrain[1]. Dans notre cas, la période intercalaire ne fut pas longue ; néanmoins il ne faut pas perdre de vue ' qu'un sporotrichosique peut porter en lui le parasite, même après la guérison apparente. MM. de Beurmann et Gougerot, MM. Sicard, Bith et Gougerot ont montré que le sporotrichum pouvait végéter quelquefois dans la bouche des sporotrichosiques. On sait d'autre part qu'une expulsion nouvelle de gommes sous-cutanées peut se faire dans quelques cas plusieurs mois après que les dernières ulcérations sont déjà cicatrisées '.

Enfin, au point de vue thérapeutique, nos observations sont toutes des plus concluantes. Dans tous les cas, la guérison fut obtenue dans un délai très rapide, allant de 20 à 30 jours ; là où le traitement mercuriel avait échoué, la médication iodo-iodurée employée seule donna un résultat.

Dans deux cas (obs. n° 4 et n° 9), après quelques cuillerées d'iodure de potassium, les malades se croyant guéris suspendirent le traitement ; mais les lésions ne tardèrent pas à réapparaître et à reprendre leur activité, d'ailleurs vite enrayée par une reprise de traitement ioduré. Gougerot attache une grande importance à ces récidives après cessation trop brusque du traitement. Le traitement iodo-ioduré a la valeur d'une

[1] Widal et Joltrain, *Bull. Soc. méd. des Hôp.*, 27 novembre 1908, n° 36, p. 647.

épreuve décisive : sa merveilleuse efficacité, la presque constante rechute *in situ* après l'interruption prématurée de la cure iodique sont très caractéristiques d'une sporotrichose.

OBSERVATIONS

Observation n° 1. — Mme de A..., âgée de 45 ans, demeure à Las Flores, province de Buenos-Ayres (République Argentine).

Les premières manifestations de sa maladie actuelle ont consisté dans une obstruction des fosses nasales due à la formation de croûtes et à l'existence de sécrétions muco-purulentes bilatérales que la malade attribuait à un ancien coryza mal soigné. Cette obstruction des fosses nasales ne fit que s'accentuer de jour en jour, les croûtes devenant de plus en plus abondantes ; en outre survint une gêne à la déglutition accompagnée de sécheresse du pharynx. Ces troubles n'avaient nullement tendance à rétrocéder, et au contraire s'accentuaient peu à peu, aussi la malade se décida-t-elle à aller consulter son médecin, le docteur Arostéguy, qui porta le diagnostic d'ulcérations tuberculeuses du voile du palais et l'adressa au docteur Woolmer (de Buenos-Ayres), afin que celui-ci fit des applications de radium. Ce dernier confrère examina donc la malade, confirma le diagnostic du docteur Arostéguy, mais, se basant sur ce que Mme de A... présentait des élévations vespérales de température (celle-ci atteignait souvent 39°), il se demanda si, outre les lésions du voile du palais, elle ne présentait pas quelque foyer viscéral ; il crut donc que le traitement par le radium n'était pas indiqué dans un pareil cas et il conseilla à la malade de venir me consulter afin que je puisse pratiquer sur elle un traitement local, et ensuite lui indiquer quel climat montagneux réussirait le mieux à son état.

Je pratiquai mon examen en avril 1912 : à gauche, la partie antéro-inférieure de la cloison était recouverte de croûtes grisâtres qui, après leur ablation, laissaient une surface ulcéreuse légèrement granuleuse peu saignante ; au niveau de la partie antérieure du cornet inférieur même aspect des lésions, mais à noter cependant que les granulations sont légèrement plus exubérantes et qu'entre elles existent des pertes de substance assez profondes, comme si se trouvaient à ce niveau de véritables petits abcès miliaires ; enfin, dans la profondeur de cette même fosse nasale, on constatait des lésions identiques à celles que nous venons de décrire au niveau du cornet ; elles étaient d'ailleurs masquées par une grande quantité de sécrétions purulentes très épaisses.

A droite, les lésions sont plus accentuées ; elles présentent les mêmes caractères d'ulcération superficielle et de granulations ; elles siègent sur la cloison et sur les deux tiers antérieurs du cornet inférieur, enfin sur la partie toute antérieure du cornet moyen.

La rhinoscopie postérieure montra le cavum rempli de croûtes et la partie supérieure du palais mou légèrement infiltré, la muqueuse n'ayant plus son brillant habituel.

Sur le voile du palais siégeait une ulcération superficielle étendue presque à la totalité du voile et qui gagnait les piliers postérieurs, principalement celui du côté gauche ; au niveau de la luette existait une perte de substance un peu plus profonde.

Enfin la paroi postérieure du pharynx présentait disséminées sans aucun ordre quelques petites ulcérations, qui siégeaient sur les follicules lymphatiques. A gauche, en arrière du pilier postérieur, existait une ulcération assez étendue.

Ces lésions avaient évolué très rapidement ; en effet les premiers

accidents dataient de deux mois ; il était donc impossible d'incriminer le bacille de Koch et de regarder ces lésions soit comme une manifestation muqueuse d'une tuberculose viscérale soit comme une forme de lupus. Les caractères macroscopiques de l'ulcération vélo-palatine, qui à la première impression ressemblaient beaucoup à ceux des ulcérations de nature tuberculeuse, en différaient cependant par de légers détails : les pertes de substances étaient beaucoup plus grandes ; on aurait dit qu'elles étaient dues à de véritables petits abcès. Les éléments qui constituaient l'ulcération étaient plus exubérants, de couleur rose pâle, et donnaient une impression semblable à celle qu'on aurait eu en regardant une ulcération miliaire à travers une lentille grossissante. Les lésions de la muqueuse nasale auraient pu par leur simple examen faire croire à un lupus, mais la rapidité de leur production et de leur extension d'une part, la vitalité des lésions d'autre part, devaient faire abandonner un pareil diagnostic. Je me trouvais donc fort perplexe, dans l'impossibilité absolue de poser sur le champ un diagnostic définitif. Désirant m'entourer de tous les éléments d'informations possibles, je commençai par faire pratiquer la réaction du Wassermann et la cuti-réaction : ces deux examens furent négatifs. Je pratiquai alors un prélèvement en pleine lésion et je demandai à mes distingués confrères les docteurs Bachmann et Marco del Pont de faire des coupes et des cultures sur différents milieux, car, à mon avis, les lésions devaient être sous la dépendance d'un parasite spécial. Le résultat de leur examen confirma d'ailleurs pleinement ma façon de voir, puisqu'ils conclurent à un cas classique de *sporotrichose* : ' Les coupes histologiques montrèrent un revêtement continu de tissu épithélial pavimenteux, pauvre en papilles et très infiltré de leucocytes polynucléaires qui en certains points forment de petits abcès miliaires. Cet épithélium est presque partout nettement séparé du tissu conjonctif, mais cependant par endroits existe un envahissement de ce tissu conjonctif par des cellules atypiques de tissu épithélial. Dans ce tissu conjonctif se trouvent des amas de petites cellules mononucléaires, du type lymphocytaire, qui avec les cellules ordinaires du tissu conjonctif forment un revêtement épais autour des glandes muqueuses ; ces dernières sont hyperplasiées et hypertrophiées et constituent de petits adénomes dans lesquels quelques tubes sont pourvus d'un revêtement à plusieurs assises de cellules épithéliales infiltrées de leucocytes polynucléaires, tout comme un épithélium de revêtement. Sur d'autres parties de la coupe se groupent trois ou quatre cellules géantes, qui ne forment pas de véritables follicules, mais sont disséminées au milieu d'un tissu très riche en cellules épithéliales ; à l'intérieur de ces cellules géantes, on constate la présence de formes mycéliennes courtes, très nettes, se colorant bien à l'hématoxyline et ne prenant pas le Gram. Ces mycéliums inclus dans des cellules géantes étaient deux ou trois fois plus volumineux et beaucoup plus faiblement colorés que ceux qui étaient libres.'

La culture ne fit que confirmer l'examen microscopique puisqu'elle permit d'étudier le parasite qui avait tous les caractères du sporotrichum Beurmani.

Cet examen anatomo-pathologique modifiait d'une façon complète le pronostic, puisque d'une lésion tuberculeuse très grave, on passait à une lésion mycosique au contraire très guérissable.

Le traitement devait consister à soumettre le malade à l'iodure de potassium à l'intérieur : je le lui fis donc prendre sous la forme de solution de Souffron, à raison de deux cuillerées par jour pour commencer, ce qui équivalait à deux grammes d'iodure par jour. Ce traitement fut commencé le 10 mai 1912 et il fut immédiatement suivi d'une amélioration ;

je n'hésitai donc pas à augmenter la dose et à la porter à trois cuillerées par jour. L'amélioration devint de plus en plus marquée, à tel point qu'un mois après, lorsque je revis la malade, la guérison était complète ; les lésions avaient disparu, laissant à leur place des cicatrices superficielles un peu plus pâles que la muqueuse environnante, mais néanmoins le voile du palais avait conservé toute sa souplesse.

Observation nº 2. — Le résultat de l'observation de Mme de A... (obs. nº 1) me fit penser immédiatement que M. de A..., son mari, que je soignais depuis environ dix mois pour une lésion nasale soi-disant lupique pouvait très bien être, lui aussi, atteint d'une lésion mycosique. L'observation suivante pourra être divisée en deux étapes : une première, pendant laquelle le malade fut considéré comme un lupique et une seconde, consécutive à la guérison de Mme de A..., pendant laquelle il a été traité comme un mycosique.

M. C... A..., âgé de 52 ans, résidant à Las Flores, province de Buenos-Aires (République Argentine), n'a aucun antécédent héréditaire ni personnel, ayant des relations avec l'affection qui nous occupe.

J'examinai ce malade pour la première fois en octobre 1911 : et son interrogatoire me fournit les renseignements suivants :

En janvier 1910, après une journée passée en plein soleil pendant laquelle il s'était tout particulièrement fatigué, le malade avait été pris d'une sorte de coup de chaleur et avait remarqué à droite, au niveau de la peau du nez, plus exactement au niveau de l'apophyse montante du maxillaire supérieur, une légère tuméfaction accompagnée de rougeur ; cette irritation, loin de s'atténuer, continua au contraire à s'étendre insensiblement et atteignit le dos du nez et une partie de la région malaire droite. En avril 1910, c'est-à-dire exactement trois mois après le début des accidents, la zone d'irritation allait toujours en s'accentuant et le malade commençait à se plaindre d'une légère gêne au niveau des fosses nasales et de la production de croûtes et des sécrétions muco-purulentes qui rendaient la respiration particulièrement difficile.

C'est à la suite de tous ces ennuis qu'il résolut de consulter un médecin de Buenos-Ayres lequel fit le diagnostic de lupus érythémateux et conseilla des applications de radium. Ce traitement, par de nombreuses applications de radium faites par le docteur Woolmer, amena une guérison complète des lésions cutanées, mais malheureusement la gêne nasale alla toujours en augmentant ; les croûtes devinrent de plus en plus nombreuses, aussi, sur les conseils de son médecin, M. de A... se décida-t-il à venir me consulter.

Mon examen me permit de constater que les fosses nasales étaient obstruées par de nombreuses et volumineuses croûtes, de couleur brunâtre, qui, une fois enlevées, découvraient une surface ulcéreuse très légèrement mamelonnée ; ces lésions existaient aussi bien au niveau des cornets inférieurs qu'au niveau du plancher des fosses nasales et de la cloison ; il est cependant à noter qu'au niveau de cette dernière, sur le cartilage quadrangulaire, existait une perforation à bords mamelonnés dont les dimensions étaient à peu près celles d'une pièce de 50 centimes. La rhinoscopie postérieure me laissa voir des lésions semblables tant au niveau de la paroi supérieure du voile du palais qu'au niveau de l'orifice postérieur des fosses nasales ; les croûtes se reproduisaient avec une rapidité extraordinaire, ce qui expliquait l'obstruction nasale permanente.

En présence de ces lésions, me basant sur le diagnostic déjà posé de lupus érythémateux de la peau, je n'hésitai pas un seul instant à considérer les lésions nasales comme d'ordre lupique et en conséquence je

conseillai à M. de A... un traitement hygiénique local tel que lavage, pommades, etc. Cependant pour avoir plus de tranquillité je fis faire une cuti-réaction et une réaction de Wassermann qui d'ailleurs furent toutes deux négatives.

J'avais pour ainsi dire perdu le malade de vue, lorsque je fus amené à soigner sa femme, Mme de A...

Ayant obtenu la guérison de celle-ci, comme nous l'avons vu dans l'observation nº 1, je pensai que les lésions du nez pouvaient très bien être de même nature que celles de sa femme, par conséquent mycosiques. Je lui écrivis donc de bien vouloir venir se soumettre à un nouvel examen.

Ce second examen, qui fut d'autant plus attentif de ma part que j'étais plus averti, me permit de constater les mêmes lésions que j'avais déjà observées dans mon premier examen en 1911, mais elles étaient actuellement beaucoup plus marquées.

Les ulcérations avaient envahi le vestibule nasal. La perforation de la cloison avait pris des dimensions plus considérables ; quant aux croûtes elles étaient toujours très abondantes.

Je pratiquai une biopsie dont je confiai l'examen à MM. Bachmann et Marco del Pont, mais ni l'examen histologique, ni l'ensemencement ne me donnèrent de résultats positifs.

J'envoyai le malade à mon confrère le docteur Aberastury, le très distingué dermatologiste de Buenos-Ayres, pour lui demander son opinion sur les quelques vestiges de lésions cutanées que présentait M. de A... au niveau de la face. Mon confrère me répondit qu'il pouvait assurer qu'il ne s'agissait certainement pas de lupus ni de toute autre forme de tuberculose, mais qu'il croyait à un cas de gomme sporotrichosique ; cette opinion était basée sur les renseignements donnés par le malade au sujet de l'aspect primitif et de l'évolution des lésions, sur l'apparence furonculeuse des nodules, qui, donnant lieu à une suppuration, étaient entourés d'une zone d'empâtement presque ligneux, enfin sur la forme linéaire de la cicatrice et sur le manque absolu de pigmentation périphérique.

Les anatomo-pathologistes auraient pu me faire abandonner mon opinion puisque tous leurs examens furent négatifs ; mais à la suite de mes examens d'une part, à la suite d'autre part de l'opinion très autorisée du docteur Aberastury, je n'hésitai pas à considérer qu'il pouvait y avoir une relation entre les deux cas, ceux de M. de A... et de Mme de A..., et que ces deux observations pouvaient permettre de soupçonner qu'il y avait eu contagion de mari à femme ; aussi n'hésitais-je pas à ordonner un traitement par la solution d'iodure de potassium.

Le résultat fut vraiment extraordinaire ; tandis qu'avec les pulvérisations, les pommades, les attouchements, les croûtes se reproduisaient avec une ténacité désespérante, il a suffi de quelques jours de traitement ioduré pour les faire disparaître et permettre ainsi le retour de la respiration nasale. Les bords de la perforation de la cloison se cicatrisèrent et la guérison complète était obtenue au trentième jour ; il est à noter cependant que le malade conserva une anosmie absolue.

Observation nº 3. — Les deux résultats précédents m'ayant laissé très perplexe, je pris le parti de rechercher parmi les malades que j'avais eu l'occasion d'examiner, ceux qui auraient pu être l'objet d'une erreur de diagnostic. Parmi ceux-ci, je pensais surtout à un malade de l'hôpital des cliniques de Buenos-Ayres qui avait été considéré et traité longtemps comme un cas de lupus du nez. Je n'avais pas osé me dresser contre ce diagnostic, mais néanmoins j'avais gardé quelques doutes sur la vraie

nature de la lésion ; aussi le fis-je revenir et c'est l'histoire de ce malade que je vais rapporter.

José T..., âgé de 26 ans, employé de commerce, n'a aucun antécédent ni tuberculeux ni spécifique. Il fait remonter le début de ses accidents à l'année 1910, époque à laquelle il commença à éprouver quelques troubles au niveau de son nez : ceux-ci consistaient surtout dans la formation de croûtes qui, augmentant progressivement, finissaient par déterminer une obstruction presque complète des fosses nasales.

Ce malade consulta plusieurs médecins qui tous firent le diagnostic de lupus du nez, et conseillèrent tour à tour pommades, lavages, attouchements et même cautérisations électriques ; mais ces diverses thérapeutiques n'amenèrent qu'un soulagement très momentané, car les croûtes se reproduisaient avec une ténacité désespérante.

C'est à la fin de juin 1912 que j'eus l'occasion d'examiner ce malade : il présentait une perforation de la cloison cartilagineuse, et au niveau de la muqueuse de la cloison ainsi qu'au niveau de celle des cornets inférieurs et moyens se trouvaient des croûtes noirâtres plus ou moins épaisses qui, une fois enlevées, laissaient à nu une surface ulcérée légèrement mamelonnée ; l'examen du pharynx permettait de constater l'existence d'une zone légèrement infiltrée au niveau du pilier postérieur droit. Enfin la rhinoscopie postérieure nous montrait que la choane était, elle aussi, légèrement infiltrée et que la partie postérieure de la cloison et la voûte du cavum étaient recouvertes de croûtes et que celles-ci enlevées, la muqueuse apparaissait très altérée.

Je fis faire la réaction de Wassermann et la cuti-réaction ; toutes deux furent négatives.

Un fragment de lésion pris au niveau du cornet inférieur fut examiné par MM. Bachmann et Marco del Pont qui m'envoyèrent les indications suivantes : ' Sur la coupe histologique de la pièce que vous nous avez envoyée, nous avons constaté un revêtement continu de tissus épithéliaux pavimenteux avec cellules typiques non altérées et une membrane basale bien conservée ; à l'intérieur du tissu conjonctif existent quelques glandules muqueuses avec leur épithélium nullement altéré ; mais au niveau de ce tissu se trouvent disséminés quelques petits tubercules, les uns à limite très nette, les autres au contraire à limite vague, s'insinuant peu à peu dans le tissu voisin. Ces néoformations sont constituées par des cellules mononucléaires du type lymphocytaire et par des éléments fixes de tissu conjonctif. A leur niveau existent des vaisseaux néoformés comme il en existe dans les productions actinomycosiques ; enfin, dans l'intérieur de ces tissus néoformés, on constate la présence de grandes masses filamenteuses mycéliales qui s'entremêlent les unes dans les autres, formant des mailles assez serrées. Les ensemencements ont fait pousser une moisissure sur laquelle nous allons poursuivre nos recherches.

' Il s'agit certainement d'un cas de mycose, peut-être streptotricose.'

A la suite de cet examen qui ne faisait que confirmer mes idées, j'ai soumis le malade au traitement ioduré utilisant la solution de Souffron ; le résultat fut presque immédiat ; quelques jours après ce début du traitement, les croûtes commencèrent à disparaître, aussi le malade put-il avoir recours à la respiration nasale.

Je revis le malade au bout d'un mois et demi, il était complètement guéri, conservant seulement sa grande perforation de la cloison et des cicatrices là où siégeaient les ulcérations de la pituitaire.

Observation nº 4. — José P..., 25 ans, bien portant, ayant même un développement musculaire très complet, grand fumeur, se marie, quand,

deux mois après son mariage, il commence à éprouver au niveau de la gorge des picotements et surtout une gêne à la déglutition ; ces troubles allèrent d'ailleurs en s'accentuant, surtout la gêne à la déglutition, qui devint si marquée que le malade se décida à aller consulter.

Son médecin posa le diagnostic de tuberculose du voile du palais et l'adressa à mon confrère et assistant le docteur Zambrini, qui, très désorienté par l'aspect de ces lésions chez un sujet aussi vigoureux, conseilla au malade de venir me demander mon avis.

Ma première impression fut mauvaise, mais l'aspect de la lésion vélo-palatine ressemblant beaucoup à celui de l'observation nº 1, j'orientai mon diagnostic vers une lésion mycosique : le malade présentait au niveau du voile du palais et des deux piliers une ulcération superficielle qui était constituée par de petites pertes de substance, ressemblant à une ulcération tuberculeuse, mais en outre sur la luette siégeait une ulcération plus profonde ; je fis faire une réaction de Wassermann et une cuti-réaction qui toutes deux furent négatives ; je prélevai un fragment des lésions dont l'examen histologique, les ensemencements et les inoculations furent également négatifs tant au sujet de la tuberculose qu'au point de vue mycosique.

Malgré mes diverses recherches dont aucune ne me permettait de m'orienter dans une direction ou dans une autre, tenant compte seulement de l'aspect de la lésion et de sa ressemblance avec les cas que j'avais déjà observés, je soumis le malade au traitement ioduré.

Le malade partant à la campagne, ce fut mon confrère le docteur Corbella qui continua à le suivre. Le résultat fut, paraît-il, tout à fait satisfaisant ; au bout de quelques jours de traitement par l'iodure, le malade éprouvait un soulagement si marqué qu'il eut même le tort de ne pas terminer sa cure iodurée.

Lorsqu'il rentra à Buenos-Ayres, environ un mois après, J. P... vint me montrer sa gorge et je pus constater au niveau du voile une cicatrisation presque totale des anciennes ulcérations, mais la muqueuse pharyngée et velo-palatine était le siège d'une rougeur assez marquée. L'interrogatoire du malade m'apprit alors qu'il avait continué à fumer d'une façon exagérée ; je pris donc le parti de lui interdire le tabac d'une façon absolue et je le remis de nouveau au traitement ioduré.

Au bout de quinze jours, la guérison était complète.

Observation nº 5. — Manuel M..., âgé de 25 ans, marié, n'a aucun antécédent ni personnel ni héréditaire qui puisse nous intéresser.

C'est en juin 1912 qu'il vint me consulter ; il se plaignait d'une irritation continuelle au niveau du carrefour aérodigestif qui, loin de s'amender, ne faisait qu'augmenter et, d'une obstruction complète de la fosse nasale gauche par des croûtes épaisses et abondantes. Aux dires du malade, tous ces ennuis auraient débuté en février 1912.

Mon examen me permit de constater, à gauche, une fosse nasale obstruée par des croûtes noirâtres, qui, après leur ablation en masse, laissaient la muqueuse de la cloison et du cornet inférieur avec des ulcérations superficielles et granuleuses ; à droite, la fosse nasale ne renfermait pas de croûtes, mais elle était le siège de sécrétions muco-purulentes.

La rhinoscopie postérieure me montra qu'au niveau du cavum existaient aussi des croûtes, surtout au voisinage du pavillon tubaire gauche qui en était presque entièrement recouvert.

A l'examen du pharynx je constatai une ulcération qui occupait toute la moitié gauche du bord libre du voile du palais, englobait les piliers antérieurs et postérieurs de ce même côté, et se propageait même un

peu au niveau de la base de la langue. Sur la base de la luette cette perte de substance était tout particulièrement nette.

L'aspect de cette lésion était tout à fait semblable aux ulcérations tuberculeuses et syphilitiques superficielles, mais comme dans les cas précédemment décrits, un examen approfondi faisait apercevoir quelques petits caractères un peu spéciaux : l'aspect de cette ulcération était plus net, sa coloration était rose-pâle sauf en certains endroits où on remarquait la présence de petits points jaunes, enfin, la zone inflammatoire tout en dépassant les limites de l'ulcération n'empêchait pas le voile du palais de conserver toute sa mobilité. Sur la paroi postérieure du pharynx existaient quelques follicules lymphatiques infiltrés et ulcérés.

Tout d'abord, je pensai que ce cas pouvait être d'ordre mycosique, mais pour affirmer mon diagnostic je voulus avoir recours à la réaction de Wassermann à la cuti-réaction et enfin à la sporo-agglutination. Ces deux premières recherches furent négatives, quant à la sporo-agglutination, elle fut visiblement positive à 1 pour cent et totalement négative dans les autres proportions ; enfin les cultures n'ont donné aucun résultat.

Malgré le peu d'éclaircissements que me donna cet examen, je résolus de traiter ce malade comme un mycosique, et je le soumis au traitement ioduré à raison de deux grammes par jour.

Le résultat fut aussi surprenant que dans les cas précédemment observés. A la fin de la première semaine les lésions de la muqueuse vélo-palatine s'étaient déjà modifiées sensiblement ; les croûtes avaient diminué, et la respiration nasale commençait à devenir libre ; cette amélioration alla d'ailleurs en s'accentuant et au 25e jour la guérison complète était obtenue.

Malgré les résultats des examens histologiques et des cultures, je pense que ce cas peut être rapproché des cas précédents et être envisagé comme un cas de mycose, car d'une part le traitement ioduré fut efficace, et d'autre part la syphilis, qui pouvait être la seule à prêter confusion, ne pouvait pas être mise en cause, la réaction de Wassermann ayant été négative, et l'amélioration ayant été presque immédiate par le simple traitement ioduré.

Observation nº 6. — Antonio P..., âgé de 45 ans, mécanicien, est bien constitué et n'a aucun antécédent héréditaire ou personnel pouvant se rattacher à l'affection qui nous occupe.

Lorsqu'il se présente à la consultation de l'hôpital Saint-Roch, en juillet 1912, je constate les lésions suivantes : au niveau de la paroi postérieure du pharynx siège une ulcération très étendue qui occupe en largeur toute sa moitié gauche et une partie de sa moitié droite, et qui, en bas, vient se terminer au niveau des aryténoïdes et atteint les replis pharyngo-laryngés. Les antécédents spécifiques manquant totalement d'une part, et d'autre part l'aspect de l'ulcération et le fait que le malade faisait remonter son existence à une vingtaine de jours, je pensai que ce cas pouvait être une mycose et je résolus de faire venir ce malade à la Société oto-rhino-laryngologique de Buenos-Ayres qui justement se réunissait ce même jour. A l'examen des lésions, la plupart de mes distingués confrères penchèrent à considérer cette lésion comme de nature syphilitique, mais quelques-uns participèrent cependant à mes doutes : aussi dès le lendemain je fis pratiquer chez ce malade la réaction de Wassermann et la cuti-réaction ; ces deux recherches furent négatives. Il en fut de même d'un examen histologique et des cultures qui n'amenèrent aucun éclaircissement au problème.

Je n'en considérai pas moins ce malade comme un mycosique et en

conséquence je le soumis au traitement par l'iodure de potassium, à raison de 2 gr. par jour. Le résultat prouva que ma manière de voir était excellente, car l'amélioration ne se fit pas attendre et fut même d'une rapidité telle que seule une ulcération de nature mycosique était capable de se cicatriser aussi vite sous l'action de l'iodure.

Au bout de 20 jours, la guérison complète était obtenue.

Observation nº 7. — Amalia T..., âgée de 28 ans, habite Rosario (République Argentine) ; mariée depuis huit ans, elle a eu quatre enfants et a fait une fausse-couche.

Les antécédents de son mari ne sont pas très clairs ; au point de vue de spécificité, il accuse avoir eu pendant sa jeunesse différentes maladies vénériennes, mais il est incapable d'en préciser la nature ; cependant il déclare qu'il n'a jamais été soumis au traitement antisyphilitique.

Ses enfants sont tout à fait bien développés et ne semblent présenter aucune tare héréditaire.

Lorsque je vis cette malade au mois d'août 1910, ma première impression fut que j'avais à faire à un cas de syphilis. Elle présentait au niveau du voile du palais une ulcération assez étendue et qui avait entraîné la perte d'une partie de la luette. Ces ulcérations étaient beaucoup plus profondes que celles que j'avais eu l'occasion d'observer dans toutes les observations précédentes, et, en certains points, sur la muqueuse ulcérée, on pouvait constater quelques petits reliefs blanchâtres d'apparence fibreuse ou cicatricielle ; le début de ces lésions, aux dires de la malade, remontait à mars 1912.

Je communiquai au mari que je pensais à une lésion spécifique mais qu'ayant quelques doutes je croyais nécessaire de faire une réaction de Wassermann, une cuti-réaction et enfin une sporo-agglutination. Malheureusement la malade avait besoin de retourner ce même jour à Rosario, aussi ces différents examens ne purent-ils être faits. Le mari me promit cependant qu'il chargerait son médecin habituel, le docteur Mangiante, de soumettre sa femme au traitement mercuriel.

Je restai un certain temps sans revoir cette malade et ce ne fut qu'en octobre 1912 qu'elle revint me consulter. Elle me déclara qu'elle avait subi deux séries de 20 injections de sel mercuriel, et tout dernièrement une injection de salvarsan. Je l'examinai et constatai que l'aspect de l'ulcération était identique à celui que j'avais observé lors de mon premier examen et, s'il y avait une modification, c'était plutôt en faveur d'une légère extension de la lésion et d'une mortification encore plus complète de la luette.

Le résultat négatif du traitement me poussa à soumettre ma malade aux différentes réactions que j'avais déjà demandées en août 1912, car je pensais que ce cas pouvait être rangé comme un cas de mycose. La réaction de Wassermann et la cuti-réaction qui furent pratiquées par mes confrères les docteurs Raffo et Justo furent négatives, ainsi d'ailleurs que les cultures. L'examen histologique ne fut guère plus concluant, il révéla seulement des lésions d'inflammation chronique qu'il fut impossible d'étiqueter.

Je soumis la malade au traitement ioduré. Le résultat fut excellent : alors qu'en deux mois avec le mercure et le salvarsan on n'avait obtenu aucune amélioration, avec l'iodure de potassium dès les premiers jours, alors que la malade n'avait encore pris que deux grammes par jour de la solution de Souffron, l'amélioration fut des plus nettes et ne tarda pas à être complète et définitive, confirmant ainsi le diagnostic de mycose.

Observation nº 8. — Mlle Amalia de V..., âgée de 20 ans, n'a aucun antécédent personnel ni héréditaire pouvant avoir une relation avec la maladie actuelle.

Il y a 15 jours la malade commença à éprouver, dans la partie gauche de la gorge, une gêne qui s'accentuait surtout pendant les mouvements de déglutition ; elle n'avait aucune élévation thermique ; son état général n'était nullement altéré. Mais elle constata bientôt que la gêne pharyngée devenait plus marquée, qu'elle éprouvait même une douleur assez nette au passage des aliments ; en outre, bien que sa respiration nasale fût parfaite, elle remarqua que sa voix se modifiait, ayant tendance à devenir rauque.

Elle vint consulter dans mon service de l'hôpital Saint-Roch où elle fut examinée par mon assistant, le docteur Zambrini, qui fit les constatations suivantes :

Le naso-pharynx est le siège d'une vaste ulcération, légèrement végétante, granuleuse, à bords irréguliers, qui s'étend depuis la partie la plus reculée du pharynx au niveau de l'implantation de la cloison, jusqu'au bord libre du voile ; cette ulcération a dans son ensemble la forme d'un trèfle et est entourée d'une zone inflammatoire.

Le bucco-pharynx ne présente aucune modification.

Au niveau du larynx, l'épiglotte n'est pas perceptible ; elle est remplacée par une tumeur végétante, légèrement arrondie, et ce n'est qu'entre certaines végétations qu'on peut apercevoir des débris du cartilage qui est en partie détruit. Cette masse granuleuse avance des deux côtés sur les replis aryténo-épiglottiques ; enfin au niveau des replis glosso-épiglottiques, l'extension est très marquée, surtout à droite, où au niveau de la base de la langue on peut apercevoir deux ou trois ulcérations à bords plus ou moins végétants. La cavité du larynx ne présente aucune altération.

Les antécédents de la malade ne me permettaient pas de penser soit à la tuberculose, soit à la syphilis, aussi ai-je dirigé mon diagnostic vers la mycose et ai-je soumis la malade à la réaction de Wassermann et à la cuti-réaction, qui furent d'ailleurs toutes deux négatives.

Je remis un fragment de la tumeur au laboratoire d'anatomie pathologique ; on y constata l'existence d'un tissu de sclérose très abondant et très vasculaire, revêtu d'une couche d'épithélium pavimenteux stratifié ; en certains points, l'épithélium était proliférant mais les lésions constatées ne permettaient pas de poser un diagnostic précis. On me promit de multiplier les coupes et de continuer les recherches, mais comme nous le verrons ultérieurement la malade ayant été très rapidement guérie, on a oublié de pousser plus loin les recherches.

Les cultures qui avaient été pratiquées sur différents milieux donnèrent des colonies de microorganismes banaux ; mais on ne constate ni champignons ni septothrix.

Le traitement fut néanmoins orienté vers la mycose et c'est à l'iodure de potassium qu'on eut recours ; la cure a commencé le 6 août et fut immédiatement suivie d'une amélioration ; la guérison complète était assurée le 24 août.

Observation nº 9. — Élisa M..., âgée de 45 ans, Espagnole, ayant constaté que depuis 5 mois sa voix avait tendance à devenir rauque et que, tout en conservant une santé générale excellente, elle éprouvait une certaine gêne à la déglutition, vint en juin 1912 à la consultation de l'hôpital des cliniques, dans le service de laryngologie dont j'avais alors la direction.

Son examen fut pratiqué par mon assistant, le docteur Basalvilbaso, qui fit les constatations suivantes : ' toute la partie visible des cornets inférieurs des deux côtés, et tout spécialement du gauche, était recouverte par une surface mamelonnée, muriforme, avec de petites nodules, du volume d'une tête d'épingle ; la coloration était rose et la superficie de la muqueuse était très sèche, aucune sécrétion ne se produisant.

' Le tiers moyen de la cloison était recouvert de petites croûtes qui, une fois enlevées, laissaient voir une exulcération dont la superficie était comme enduite d'une sécrétion purulente.

' Au niveau du naso-pharynx, mêmes lésions, mais cependant il n'y a pas à ce niveau la même régularité et à mesure qu'on descend à un niveau inférieur, les ulcérations sont de plus en plus accentuées et profondes. Cette paroi du pharynx présente une muqueuse irrégulièrement épaisse avec des saillies séparées par des fissures ulcérées, ulcérations qui d'ailleurs n'ont nullement l'aspect des ulcérations tuberculeuses ou syphilitiques, elles auraient plutôt fait penser à des lésions lupiques, si la luette et le voile du palais n'avaient pas été indemnes.

' Sur les deux faces de l'épiglotte ainsi qu'au niveau de la région aryténoïdienne, les lésions avaient le même aspect que sur le cornet ; elles consistaient en ulcérations légèrement mamelonnées. L'intérieur de la cavité laryngée était indemne.'

Cet examen objectif fut complété par la réaction de Wassermann et la cuti-réaction qui furent négatives : enfin on fit un prélèvement des tissus altérés dont l'examen fut confié aux docteurs Bachmann et Marco del Pont.

Voici leurs conclusions : ' L'examen de la coupe ne permet de constater au niveau de l'épithélium de revêtement qu'une importante migration leucocytaire.

' Le tissu conjonctif est le siège d'une inflammation chronique avec ses lésions habituelles d'altérations cellulaires et des néoformations conjonctives, genre tuberculeuses ; mais en aucun point on n'observe l'aspect typique d'un tubercule dû au bacille Koch.

' Enfin on trouve disséminées des cellules géantes et des formations épithélioïdes.'

Cet examen anatomo-pathologique n'amenait aucun éclaircissement.

On mit la malade à un traitement mixte par le sirop de Gibert et l'iodure de potassium. Après l'absorption d'un flacon de 300 grammes, il y eut une amélioration très sensible, mais il était impossible de parler d'une guérison.

La malade suspendit tout traitement, les lésions ne tardèrent pas à reprendre de l'activité, c'est alors que je vis la malade pour la deuxième fois, en fin juillet 1912. Je lui ordonnai la solution iodurée. Le résultat fut alors excellent ; de jour en jour, on pouvait suivre la rétrocession des lésions et bientôt la guérison totale et définitive était obtenue.

Observation n° 10. — Cette observation nous a été très obligeamment prêtée par M. Gougerot, auquel nous adressons nos remerciements.

Observation de MM. Gougerot et Quellien. Il s'agit d'une femme de 50 ans, qui présenta une ulcération du pharynx au sujet de laquelle les plus éminents spécialistes furent d'avis différents, les uns la considérant comme syphilitique, et la traitant par le mercure qui d'ailleurs ne donna rien, les autres au contraire la croyant tuberculeuse et essayant de divers traitements qui tous échouèrent.

Les lésions ne faisaient que progresser et s'aggraver ; la malade quitta alors Paris ; c'est alors que le docteur Quellien eut l'occasion de la voir.

Il pensa à la possibilité d'une mycose et soumit la malade au traitement ioduré.

La guérison fut obtenue rapidement, en quatre semaines.

Une fois guérie, la malade fut amenée au docteur Gougerot qui fit une culture du mucus bucco-pharyngé. Le parasite fut trouvé et le sérum agglutina et fixa le complément.

Observation nº 11. — José M..., âgé de 45 ans, Italien, habite depuis 24 ans la République Argentine. Il n'a aucun antécédent héréditaire, ni personnel ; marié, il n'a pas d'enfant ; sa femme n'a jamais fait de fausses-couches.

La première manifestation de la maladie actuelle remonterait, aux dires du malade, à huit ans, date à laquelle il aurait remarqué l'apparition d'une tache blanchâtre au niveau de la paroi interne de la joue gauche; cette altération de la muqueuse ne lui occasionnait d'ailleurs aucun malaise, puisque ce fut tout à fait par hasard, à l'occasion d'une douleur dentaire, qu'il s'en aperçut.

Au début le malade, qui était un grand fumeur et qui ne dédaignait pas de chiquer, n'attacha pas grande importance à ces lésions, car si la plaque s'était recouverte de nodules à superficie blanchâtre, ceux-ci n'étaient nullement douloureux ni saignants.

Mais bientôt, tout en laissant au malade une santé générale parfaite, les lésions prirent une grande extension et alors seulement l'amenèrent à venir consulter les docteurs Ceballos et French qui, sans donner de diagnostic, déclarèrent que cette tumeur était guérissable par des cautérisations.

Je vis José M... pour la première fois le 1er juin 1912 : dans la même séance, je l'examinai et je fis un prélèvement en plein tissu altéré afin d'avoir l'avis des anatomo-pathologistes, les docteurs Bachmann et Marco del Pont.

L'aspect des lésions était typique : au milieu de bourgeons exubérants, on constatait une prolifération énorme de villosités de couleur blanchâtre, mates, tout à fait comparables comme aspect aux fines aiguilles que l'on observe dans le cas de mycose des amygdales, mais beaucoup plus exubérantes.

La réaction de Wassermann et la cuti-réaction furent négatives ; d'ailleurs il est à noter que le malade avait été soumis au traitement antisyphilitique qui n'avait déterminé aucune amélioration.

L'examen histologique donna les indications suivantes : ' L'épithélium est le siège d'une prolifération très marquée, accusée par la formation d'un grand nombre de papilles qui par suite de multiples ramifications finissent par déterminer une innombrable quantité de prolongements filiformes, recouverts à leur superficie par d'épaisses couches d'épithélium corné.

' En outre de cette prolifération superficielle, en beaucoup de points l'épithélium n'a pas de limites très précises avec le tissu conjonctif : en de nombreux endroits il y a une confusion de tissus par suite de la disparition totale de la membrane basale : cette invasion du tissu conjonctif par des éléments atypiques de l'épithélium se caractérise par la formation, à l'intérieur du tissu conjonctif, d'amas et de cordons ramifiés, anastomosés entre eux. Il s'ensuit que par endroits on peut très nettement apprécier l'union de ces cellules proliférées avec les papilles de l'épithélium de revêtement. Certainement cette prolifération épithéliale est du type adulte ; le tissu conjonctif est le siège d'une infiltration mononucléaire intense et diffuse, qui en certains points se condense en formant de petits

follicules au sein desquels de petites cellules géantes se trouvent irrégulièrement distribuées.

' En plein tissu épithélial existent des micro-abcès d'évolution certainement lente, car ils déforment les cellules périphériques qui s'aplatissent et se disposent en cercles concentriques autour de la lésion, lui formant ainsi une sorte de membrane. Le contenu de ces abcès varie avec leurs dimensions : les plus petits sont constitués par des agglomérations de leucocytes polynucléaires et d'une à trois grandes cellules géantes très fortement nucléées. Les fausses membranes ne se rencontrent que dans les grands abcès, déjà anciens, qui sont constitués en grande partie de détritus cellulaires se colorant d'une façon intensive par l'hématoxyline. Entre ces deux variétés extrêmes, existe une grande quantité de formes de transition.

' Au milieu de toutes ces lésions, expliquant leur pathogénie, se trouvent des formations mycéliales qui constituent un réseau assez serré.

' Se trouve-t-on en présence d'une mycose primitive qui a été la cause de toutes ces lésions aussi bien conjonctives qu'épithéliales, ou a-t-on affaire à un cancer avec invasion secondaire du parasite. Il est très difficile de l'affirmer, mais je penche plutôt vers la première hypothèse.

' Les cultures ont donné naissance à d'abondantes colonies d'un parasite facultativement anaérobie, très comparable à l'endomyces albicans (muguet). Nous allons en poursuivre l'examen.'

La sporo-agglutination faite avec le sérum du sang de ce malade et des levures de champignons a donné les résultats suivants :

A $\frac{1}{100}$ réaction positive.
A $\frac{1}{200}$ réaction faiblement positive.
A $\frac{1}{300}$ réaction négative.
A $\frac{1}{400}$ réaction négative.

L'opinion des anatomo-pathologistes fut confirmée par la marche ultérieure de la maladie ; il s'agissait certainement d'un cancer primitif ou secondaire.

A la suite du traitement ioduré, il se produisit en certains points une amélioration très nette, mais là où existaient des ulcérations profondes, il n'y eut aucune modification.

Les lésions ne tardèrent pas à s'étendre et même au point où furent prélevés les tissus donnés à examiner, les bourgeons réapparurent très rapidement. Peu à peu la joue fut atteinte par le processus ; elle constitua d'abord un placard dur qui empêchait les mouvements de la mâchoire de s'exercer librement, puis apparut à ce niveau une tuméfaction qui fut bientôt accompagnée de rougeur, d'infiltration ; la peau était comme plaquée, adhérente aux plans profonds et ne tarda pas à s'ulcérer ; son aspect était alors comparable à celui d'une ulcération consécutive à la rupture d'un anthrax, mais assez rapidement sur les bords de la perforation apparurent des bourgeons d'une activité toute spéciale, saignant très facilement, mais ne déterminant aucune douleur.

Les hémorragies se firent de jour en jour plus fréquentes, plus abondantes et plus inquiétantes ; elles se produisaient non seulement au niveau des lésions de la cavité buccale, mais aussi au niveau des lésions cutanées ; elles amenèrent un amaigrissement très marqué et un état de dépérissement qui ne fit qu'augmenter journellement.

Ayant été obligé de m'absenter de Buenos-Ayres, je n'ai pu savoir ce qu'était devenu ce malade.

Dans ce dernier cas, le traitement par l'iodure de potassium n'a certes pas donné le résultat attendu, qui avait été si merveilleux dans tous les

autres cas dont nous avons relaté les observations ; nous n'en considérons pas moins ce cas comme très intéressant, car je suis persuadé que si dès le début on avait fait un traitement ioduré, on aurait pu éviter la transformation néoplasique.

Je suis en effet convaincu que ce malade avait au début des lésions mycosiques, la lenteur du début de l'évolution étant en faveur de ces lésions.

Dans toutes ces observations, à quelques rares exceptions, on n'est pas arrivé à déceler l'existence du parasite, soit par les examens histologiques soit par les cultures ; on pourrait donc objecter que, vu ces examens négatifs, les lésions n'étaient pas d'ordre mycosique, mais relevaient de la syphilis dont certaines lésions peuvent très bien être guéries par l'absorption d'iodure de potassium. Mais, en faveur de la mycose, se dressent plusieurs arguments : d'abord la réaction de Wassermann qui a toujours été négative et surtout l'infime dose d'iodure de potassium à laquelle on a dû recourir pour obtenir la guérison. En cas de syphilis, ces doses auraient été totalement insuffisantes et on aurait dû recourir à des doses très élevées et surtout au mercure ou au salvarsan.

Je crois qu'il est des plus utile de publier tous les cas de mycose que l'on est amené à observer. Jusqu'à nos jours les observations de mycose des muqueuses sont encore relativement peu fréquentes. A quoi attribuer cette rareté : la raison c'est que la plupart des spécialistes ne pensent guère à ce genre de lésions. Depuis que j'ai eu l'attention attirée sur ces lésions parasitaires, je les recherche systématiquement : en quelques mois, j'ai eu l'occasion d'en observer dix cas ; je suis donc frappé de cette fréquence des maladies à champignons et je suis persuadé que nombre de malades considérés pendant longtemps comme des syphilitiques ou des tuberculeux sont en réalité des sporotrichosiques. J'irai même plus loin : je suis convaincu que certains malades, dont j'ai lu les observations, qui, considérés comme guéris de cancer du voile du palais, des piliers ou du pharynx, après un traitement chirurgical, des applications électriques ou des cautérisations, n'étaient autres que des mycosiques et que quelques grammes d'iodure de potassium auraient eu rapidement raison des lésions.

Enfin en réfléchissant aux malades que j'ai eu l'occasion d'observer avant d'avoir eu l'attention attirée sur la mycose, j'en retrouve un certain nombre que j'ai cru atteints de lupus, de syphilis ou de tuberculose, qui au fond étaient probablement des mycosiques, si je me base sur les résultats que j'avais obtenus par le traitement (l'observation nº 3 est un exemple typique).

L'étude des sporotrichoses présente donc un intérêt pratique et thérapeutique considérable.

Très grande aussi est l'importance de ce diagnostic au point de vue social ; comme l'a si bien dit le professeur Landouzy : ' Pris pour un tuberculeux, on condamnait le sporotrichosique au repos prolongé et trop souvent à la suralimentation, on essayait, mais sans grande conviction, des topiques locaux, et voyant les plus énergiques échouer, on s'en

remettait à la natura medicatrix pour guérir le malade ; la maladie s'aggravait lentement, . . . si par hasard, le patient guérissait à la suite de médicaments dont les iodo-tanniques avaient fait les frais, on gardait toute espèce de réserves pour son avenir ; on avait toutes difficultés à ne pas le voir promis à d'autres manifestations bacillaires.

' Pris pour un syphilitique, traité par le mercure, le sporotrichosique voyait sa maladie se prolonger et s'aggraver ; heureux si, revenant à la vieille pratique d'associer l'iodure au mercure, le médecin voyait les gommes guérir ; mais le patient restait justiciable de toutes réserves pronostiques, puisque la syphilis, qui semblait l'avoir atteint dans le passé, le menaçait dans l'avenir.

' Or, savoir dépister et reconnaître, sous l'un quelconque de ses aspects, la sporotrichose, c'est la guérir puisque l'on possède un traitement quasi-spécifique, le traitement ioduré ; c'est rassurer le malade sur son avenir, puisque, sauf exception, la sporotrichose est une infection bénigne.'

La gloire de nos très distingués collègues les docteurs de Beurmann et Gougerot sera donc de plus en plus grande ; les premiers, ils ont individualisé cette mycose et ce sont eux qui nous ont appris à la reconnaître cliniquement et qui nous ont donné la méthode du diagnostic simple et rapide de la culture à froid qui porte si justement leurs noms.

SECTION XV

RHINOLOGY AND LARYNGOLOGY

INDEPENDENT PAPER

LES INFECTIONS PAR VOIE NASALE

PAR M. LE DR. TRÉTRÔP, D'ANVERS (BELGIQUE)

I. — RÉSUMÉ

M. TRÉTRÔP, d'Anvers, après avoir rappelé la sensibilité de la muqueuse nasale intacte aux infections démontrée expérimentalement à l'aide de bacilles pesteux ou diphtéritiques, examine les deux modes essentiels de protection physiologique :

1° La disposition anatomique.

2° La sécrétion cellulaire.

En cas d'insuffisance ou de viciation de leur fonction, les fosses nasales respiratoires doivent être aidées thérapeutiquement ; M. Trétrôp cite les principaux agents à employer.

Il passe en revue les principales infections restant locales ou devenant générales et il conclut à la nécessité d'appeler souvent l'attention des familles sur la nécessité de la respiration nasale exclusive par des fosses nasales en bonne santé pour protéger le nez et l'organisme tout entier contre les infections locales ou générales.

II. — COMMUNICATION

Le nez a une double fonction : une fonction *olfactive*, une fonction *respiratoire*.

Je n'envisagerai pas dans ce court aperçu les infections de la région supérieure ou olfactive, je parlerai seulement des infections spontanées, venues du dehors, s'attaquant surtout à la partie inférieure ou respiratoire des fosses nasales.

Si nous nous en rapportons aux études expérimentales faites dans les laboratoires, le nez constitue une excellente porte d'entrée pour les *infections* soit *locales*, soit *générales*.

Vous rappellerai-je les données expérimentales obtenues vers 1894 pour la diphtérie et en 1898 pour la peste par le docteur Roux, l'éminent directeur de l'Institut Pasteur de Paris. Je répétai alors avec succès ces expériences au laboratoire de bactériologie et d'anatomie pathologique des hôpitaux civils d'Anvers dont j'eus la direction pendant quinze ans. Il suffit de déposer une très faible quantité de bacilles pesteux ou

diphtéritiques virulents sur une muqueuse nasale non-excoriée, absolument intacte, pour reproduire l'infection pesteuse ou diphtéritique.

Dans un récent travail sur la lèpre,[1] j'appelais l'attention de la Société française d'oto-rhino-laryngologie sur ce mode possible et probable de propagation de la lèpre. Je citais à l'appui de cette manière de voir l'étude de Janselme et Laurens [2] sur ce sujet.

Quels sont les moyens de défense des fosses nasales respiratoires ?

D'un côté : *la disposition anatomique*, d'un autre côté : *la sécrétion cellulaire*. La disposition anatomique est telle par suite de la surface aussi étendue que sinueuse des cornets que l'air est obligé au passage de frotter sur ces surfaces et qu'il s'y dépouille en majeure partie des germes qu'il contient. C'est un fait bien connu que l'arrêt des poussières par les fosses nasales. Les poils jouent le même rôle. Quant à la sécrétion, il a été démontré, notamment, par notre collègue Lermoyez, que le mucus nasal est nettement bactéricide.

Sinuosité du parcours rappelant le col de cygne à fonction aseptisante du ballon Pasteur, humeur bactéricide : voilà deux moyens de défense physiologique efficace.

Et, de fait, le nez arrête au passage et détruit journellement un grand nombre de bactéries nocives. Mais il peut devenir insuffisant, soit par suite d'un apport trop considérable de germes qu'il n'arrive plus à arrêter ou à détruire, soit par affaiblissement ou viciation de sa fonction. Il réclame alors aide et assistance.

Au moyen âge, les médecins appelés à visiter les malades dangereux, les pestiférés notamment, faisaient d'instinct de la prophylaxie bactériologique, ils se couvraient la face d'un masque pourvu d'un long cornet pointu au niveau du nez, cornet muni seulement de deux petites ouvertures et que l'on remplissait de matières parfumées.

Actuellement, pour protéger les fosses nasales et aussi par réciprocité pour empêcher la projection au dehors de particules nasales dangereuses, on a imaginé différents masques filtrant l'air inspiré ou expiré, tout comme le cornet des médecins du moyen âge. Le plus simple est constitué par quelques doubles de gaze aseptique.

Les poudres, surtout les poudres solubles et les pommades peuvent constituer une bonne protection contre les infections nasales. C'est la base de nos traitements courants dans les rhinites de divers genres. Et il est certain que les poudres ou pommades à base de sels mercuriaux : calomel, précipité blanc ou rouge, etc., à base de peroxydes alcalins ou alcalino-terreux, le tout à faible dose pour ne pas altérer la muqueuse, ont une réelle efficacité. Les bains peuvent agir dans le même sens, mais ils exigent plus de prudence, car les solutions non isotoniques compromettent facilement l'intégrité des cellules épithéliales.

[1] Dr. Trétrôp, ' Un cas de lèpre naso-bucco-pharyngée,' *Congrès de la Soc. franç. d'oto-rhino-laryng.*, Paris, mai 1913.

[2] Drs. Janselme et Laurens, ' Des localisations de la lèpre sur le nez, la gorge, et le larynx,' *Soc. méd. des Hôpit. de Paris*, 1897.

Si le nez est très sensible aux infections, s'il peut aisément servir de porte d'entrée à des maladies microbiennes générales, il conviendrait de le soustraire toujours aux moindres excoriations, qui ouvrent tant de portes à l'infection.

En pratique, nous voyons que cette règle de prophylaxie est loin d'être suivie et que le revêtement corné des doigts s'introduit fréquemment avec le plus grand dommage ou les plus grandes chances d'infection dans la partie antérieure des fosses nasales respiratoires.

Les principales infections nasales sont : les catarrhes, coryza et rhinites de toutes espèces compliqués ou non de sinusite, dont la cause infectieuse n'est pas contestable. Comme toute inflammation microbienne, ces inflammations nécessitent un terrain propice et prédisposé. L'ozène, rhinite infectieuse et sans doute contagieuse, sur la pathogénie de laquelle, n'étant pas encore suffisamment fixé, on fait en ce moment une enquête mondiale. Les inflammations furonculeuses et phlegmoneuses dues à l'introduction dans la muqueuse de germes de la suppuration : streptocoques, pneumocoques, staphylocoques, bacilles pyocyaniques, etc., et les infections morveuses, syphilitiques, tuberculeuses, lépreuses, qui rentrent dans la catégorie des inflammations spécifiques à productions gommeuses.

Je n'examinerai pas ici le traitement suffisamment connu de ces diverses infections restant locales ou devenant générales ni l'emploi des vaccins au point de vue prophylactique. Je suis convaincu qu'il en existe encore bien d'autres et que si maintes infections générales ont un début pharyngé, il en est nombre parmi elles dont la porte d'entrée, avant la localisation pharyngée, est constituée par les fosses nasales.

Il résulte de ces données rapidement énoncées que le nez, organe respiratoire, a une action essentielle de préservation tant au point de vue de l'organe lui-même qu'au point de vue de l'organisme tout entier, que l'intégrité de cette fonction est indispensable, qu'elle comporte des règles de prophylaxie et que la pathologie des fosses nasales réclame des soins immédiats et suivis jusqu'à guérison.

Il importe donc d'appeler de temps à autre l'attention des familles, encore trop ignorantes souvent, sur la nécessité d'avoir non seulement une bonne respiration nasale, mais encore des fosses nasales en bon état de santé. Une respiration nasale par des fosses nasales normales protégera efficacement les voies respiratoires sous-jacentes et le corps entier contre les infections. D'autre part, si le simple furoncle du nez peut déterminer par infection à distance des complications endo-crâniennes, il est non moins certain que des infections graves, susceptibles de se généraliser, telles : le lupus, la syphilis, la diphtérie, la lèpre et bien d'autres affections peuvent avoir, et ont en réalité, parfois un début insidieux endo-nasal qui, faute d'examen à temps, passe inaperçu.

Les infections par voie nasale constituent un chapitre important de la pathologie tant spéciale que générale et elles sont un terrain d'union entre le spécialiste et le médecin général.

SECTION XV

RHINOLOGY AND LARYNGOLOGY

INDEPENDENT PAPER

ÜBER DEN DIAGNOSTISCHEN UND PROGNOSTISCHEN WERT DER LOKALEN TUBERKULINREAKTIONEN AUF GRUND NEUERER UNTERSUCHUNGEN

Von Dr. ELEMÉR VON TÓVÖLGYI, ordinierender Kehlkopf-Arzt der Budapester ' Dispensaire '

Die Ansichten über den Wert der Subkutan-, Intrakutan- und Kutanreaktionen waren bis zu dem heutigen Tage, trotz der auf mehrere hunderte zu schätzenden Anzahl der Beobachtern, sehr geteilt und der vor 5 Jahren gehegte Wunsch Zieglers: ' Das Bedürfnis, dass zahlreiche sorgfältige Beobachtungen eine endgültige Entscheidung herbeiführen möchten, ist grösser denn je ' — hat sich bis heute noch nicht erfüllt.

Uns Kehlkopfärzte, denke ich, wird diese Frage mit Rücksicht darauf, dass wir es gewöhnlich mit den schwierigeren Fällen der Tuberkulose zu tun haben, noch in erhöhterem Masse interessieren als die Spezialisten für innerliche Krankheiten, und darin liegt der eine Hauptgrund, dass ich über meine bei der Tuberkulose mit den lokalen Reaktionen sowohl vom diagnostischen, als auch prognostischen Gesichtspunkte erreichten Resultate auf Grund meiner von den bisherigen Beobachtern gänzlich abweichenden Versuchen an dieser Stelle zu berichten das Vergnügen habe.

Sämtliche bisherige Beobachter der Ophthalmoreaktionen haben immer eines aus ihrer Rechnung vergessen, namentlich den Zustand der Nase und das enge Verhältnis dieses Zustandes zu den Konjunktiven.

Bei meinen Untersuchungen habe ich gefunden, dass in den Fällen der akuten oder subakuten Rhinitis die Ophthalmoreaktion sozusagen immer eine positive ist. Die chronische Rhinitis hingegen hat keinen grossen Einfluss auf sie.

In 186 Fällen (Tab. I) habe ich neben Ophthalmoreaktion auch Rhinoreaktion ausgeführt, und zwar so, dass ich die Nasenschleimhaut mit 50 prozentiger Alttuberkuline bestrichen habe, aber das hat ebenfalls keinen wesentlicheren Einfluss gehabt, sie hat aber einen interessanten Umstand bewiesen, dass von den 186 Fällen nur in 35 Fällen diese zwei Reaktionen voneinander abweichende waren, d. h. die eine war +, die

andere —, *welche Erscheinung beweist, dass die Ophthalmo- und die Rhinoreaktionen ganz ähnlichen Gesetzen unterworfen sind.*

TABELLE I

Ophthalmoreaktion.			*Rhinoreaktion.*
+	Rhinitis chron.	29	+
	Sine rhinitis	22	
—	Rhinitis chron.	32	—
	Sine rhinitis	68	
+	Rhinitis chron.	2	—
	Sine rhinitis	11	
—	Rhinitis chron.	9	+
	Sine rhinitis	13	
	TOTAL	186	

Beide Reaktionen gleich: 151. Beide Reaktionen verschieden: 35.

Wenn man diese 186 Fälle von dem Gesichtspunkte aus betrachtet, in welchem Grade die Tuberkulose bei denselben clinice festgestellt wurde, dann erhält man folgende Tabelle:

TABELLE II

Ophthalmoreaktion.			*Rhinoreaktion.*
+	Sine morbo: 1	I. u. II. Stad.: 19 III. Stad.: 30	+
+	Ophthalmoreaktion stark und dauerhaft. I. u. II. Stad.: 4	Ophthalmoreaktion sehr schwach. III. Stad.: 11	—
—	Rhinoreaktion stark und dauerhaft. I. u. II. Stad.: 7	Rhinoreaktion sehr schwach. III. Stad.: 15	+
—	I. u. II. Stad.: 23	III. Stad.: 76	—
	TOTAL: 186		

Nach meinen Untersuchungen ist also die Ophthalmo- und die Rhinoreaktion in den schweren Tukerkulosefällen entweder eine negative oder eine sehr schwache, und in den mittelschweren sowie leichtgradigen Fällen eine starke.

Im Verhalten der Ophthalmoreaktion tritt eine wesentliche Aenderung ein, wenn der akute Rhinitis künstlich hervorgerufen wird, derart, dass man mit 50-, 25-prozentigem Alttuberkulin oder 10-prozentigem Menthol getränkte Watte auf einige Stunden in die Nase einsetzt.

Während von 186 solchen Fällen, in welchen kein künstlicher Schnupfen herbeigeführt wurde, nur in 64 Fällen die Ophthalmoreaktion + war, wurde in 82 Fällen des künstlichen Schnupfens gerade bei der Hälfte der Fälle die Ophthalmoreaktion zu einer positiven.

TABELLE III

Ophthalmo-reaktion.		*Rhino-reaktion.*
+	Total 41	+
+	Total 8	—
—	Total 20	+
—	Total 21	—
	ZUSAMMEN . . 82	

Beide Reaktionen gleich: 62 Beide Reaktionen verschieden: 20

Tabelle III beweist, dass die Ausführung der Rhinotamponreaktion den Wert der Ophthalmoreaktion nicht nur nicht herabsetzt, sondern dadurch, dass sie die letztere empfindlicher macht, noch steigert, indem die Negativität der Ophthalmoreaktion bei gleichzeitiger Rhinotamponreaktion in erhöhtem Masse dafür spricht, dass man einer hochgradigeren, also eine schlechtere Prognosis bietenden Tuberkulose gegenübersteht.

TABELLE IV

Ophthalmo-reaktion.		*Rhino-reaktion.*
+	Beide stark. Sine mor. I. u. II. Stad.: 19	Beide schwach. II. u. III. Stad.: 22	+
—	Rhinoreaktion stark. Sine mor. I. u. II. Stad.: 10	Rhinoreaktion sehr schwach. II. u. III. Stad: 8	+
—	Sine mor. I. u. II. Stad.: 4	III. Stad.: 8	III. Stad. mit Tub. lar.: 11	—
	TOTAL: 82		

Die + Vermehrung der Ophthalmoreaktion bei Anwendung des Nasentampons bezieht sich zumeist auf die Fälle der leichtgradigeren tuberkulosen Aenderungen und zeigt genauer die leichteren Fälle an und trennt die schwierigeren Erkrankungen bestimmter von den weniger schwierigen. Diese beiden Reaktionen sind aber noch immer nicht genug genau, um an sich allein mit vollständiger Bestimmtheit den Grad der tuberkulösen Veränderung anzuzeigen.

Die von mir in dieser Form zuerst angewandte Rhinotamponreaktion kann wahrscheinlich auch die Subkutanmethode vorteilhaft ersetzen, indem sie, — die fieberige Reaktion ebenfalls einleitet und dadurch, dass ich bei dieser Methode beliebig konzentrierte Tuberkulinlösung verwenden kann, sowie dadurch, dass ich dieses Tampon beliebig lange Zeit in der Nasenhöhle belassen kann, kann ich eine beliebig tiefe Einsicht über den Verlauf der eintretenden Fieberreaktion und im allgemeinen über die Reagierungsfähigkeit des Patienten gewinnen.

Bei den Untersuchungen der Pirquetimpfung hingegen habe ich die

Erfahrung gemacht, dass sie an sich allein ebenfalls keinen genauen Aufschluss über die Prognose der Tuberkulose gibt, dass es sogar nicht genügt, wenn man gleichzeitig auch die Ophthalmo- und Rhinoreaktion anwendet, sondern, wenn man genaue Resultate erlangen will, auch an einer anderen Stelle der Hautfläche des Patienten, namentlich an der Brust, diese Kutanreaktion gleichzeitig ausführen muss.

Auf den Versuch der Brustkutanimpfungen hat mich übrigens der Gedanke geführt, ob man aus den verschiedenen Graden der an beiden Seiten der Brust bewirkten Impfungen nicht auf den vorgeschritteneren oder niedrigeren Grad des rechts- oder linksseitigen Lungen-Vorganges schliessen könnte ?

Mein Untersuchungsmaterial ist noch nicht hinreichend, so dass ich noch kein endgültiges Urteil fällen kann, soviel muss ich aber angeben, dass sowohl die einmaligen, als auch die wiederholten Impfungen eher dafür sprechen, dass an jener Hautfläche der Lungenhälfte, die mit einer leichteren Tuberkulose behaftet ist, auch die Reaktion eine ausgeprägtere ist.

TABELLE V

Ophthalmo-reaktion.		*Rhino-reaktion.*
	Tuberc. pulmon. later. utr.	
+	Fall : 70	+
—	Fall : 16	—
++	Stärker rechts. Fall : 22	+
+	Stärker links. Fall : 23	++
	Tuberc. pulmon. later. dextri.	
+	Fall : 14	+
—	Fall : 6	—
+	Stärker links. Fall : 14	++
++	Stärker rechts. Fall : 8	+
	Tuberc. pulmon. later. sinistri.	
+	Fall : 8	+
—	Fall : 5	—
+	Stärker links. Fall : 5	++
++	Stärker rechts. Fall : 12	+
	TOTAL : 203	

Von 203 Fällen war sie, wie dies die Tabelle zeigt, im Falle der clinice zweiseitigen gleichgradigen Veränderung, auch jene Fälle hieher gerechnet, bei welchen die Reaktion auf der Haut der clinice schwieriger erkrankten Lungenhälfte eine schwächere Positivität gezeigt hat, als auf der anderen Seite, in 112 Fällen eine entsprechende und in 91 Fällen eine verfehlte. Es ist aber eine grosse Frage, ob in den 45 Fällen, in welchen clinice in den beiden Lungenhälften eine gleiche Veränderung festgestellt wurde, mit Rücksicht auf die in dieser Richtung nicht übermässig verlässlichen klinischen Untersuchungen, die zweiseitigen Vorgänge tatsächlich gleiche waren. Wenn man diese 24 Fälle von den 91 Fällen in Abzug bringt, bleiben 112 Fällen gegenüber insgesamt 46 solche Fälle, in welchen

die Stärke der Reaktionen an den zwei Seiten von der normalen abweicht.

Die auf der der krankeren Lungenhälfte entgegengesetzten Brustseite aufgetretene Reaktion war von 39 Fällen in 26 Fällen stärker und in 13 Fällen, also gerade der Hälfte der vorerwähnten Fälle, übereinstimmend mit der auf der entsprechenden Seite aufgetretenen Reaktion.

Betrachten wir demgegenüber das Resultat der wiederholten Einimpfungen. Bisher kann ich leider nur über 13 Fälle berichten:

TABELLE VI

Kranke.	1913.	*Brust.*		*Larynx.*
		Rechts.	*Links.*	
1. *K.* Gaffky II. Cat. ap. l. utr. Impfung: 50% Alttuberkulin.	iv. 2	+(schwach)	+(schwach)	ulcer. chord. lat. utr.
	v. 26	+(mittelstark)	+(mittelstark)	
2. *Gy.* Gaffky 9. Cat. ap. l. utr. Impfung: 50% Alttub.	iv. 23	+(mittelstark)	+(mittelstark)	
	v. 21	+(mittelstark)	+(mittelstark)	
3. *D.* Gaffky III. Cat. ap. l. utr. Impfung: 50% Alttub.	iv. 30	+(sehr schwach)	+(sehr schwach)	ulcer. chord. l. u. Perich. Arytenoid.
	v. 17	+(mittelstark)	+(mittelstark)	
4. *S.* Gaffky III. Cat. ap. l. utr. Impfung: 50% Alttub.	v. 5	+(sehr schwach)	+(sehr schwach)	
	v. 15	+(mittelstark)	+(mittelstark)	
5. *R.* Gaffky II, III. Inf. ap. l. utr. Impfung: 50% Alttub.	iv. 2	+(sehr schwach)	+(sehr schwach)	
	iv. 19	+(sehr schwach)	+(sehr schwach)	
6. *W.* Gaffky —. Cat. ap. l. utr. Impfung: 50% Alttub.	v. 2	+(stark)	+(stark)	
	vi. 23	+(stark)	+(stark)	
7. *G.* Gaffky —. Cat. ap. l. utr. Impfung: 50% Alttub.	iv. 17	+(mittelstark)	+(schwach)	
	v. 29	+(sehr schwach)	+(schwach)	
8. *T.* Gaffky III. Cat. ap. l. dext. Impf.: 50% Alttub.	iii. 12	+(mittelstark)	+(stark)	Granul. et. utr. chord. l. dext.
	iv. 17	+(mittelstark)	+(stark) (!!)	
	v. 19	+(mittelstark)	+(stark) (!!)	
9. *V.* Gaffky —. Cat. ap. l. sin. Impfung. 50% Alttub.	iv. 2	+(mittelstark)	+(schwach)	
	v. 3	+(mittelstark)	+(schwach)	
10. *H.* Gaffky I, II. Inf. ap. pr. l. dext. Impf.: 50% Alttub.	iv. 2	—	—	
	iv. 12	+(schwach)	+(schwach)	
11. *B.* Gaffky II, III. Int. ap. pr. l. sin. Impf.: 50% Alttub.	iv. 2	+(mittelstark)	+)schwach)	
	iv. 29	+(schwach)	+(sehr schwach)	
12. *H.* Gaffky —. Cat. ap. pr. l. sin. Impfung: iv. 2, 50%; v. 26, 25 %.	iv. 2	+(sehr stark)	+(stark)	
	v. 26	+(schwach)	+(sehr schwach)	
13. *B.* Gaffky —. Cat. ap. pr. l. dext. Impfung: iv. 2, 50%; v. 26, 25%.	iv. 2	+(sehr stark)	+(mittelstark)	
	v. 26	+(sehr schwach)	+(sehr schwach)	

Auch hier ist es auffallend, dass in 11 *Fällen die Brustreaktion an beiden Seiten dasselbe Verhältnis zeigte, so oft ich die Impfung wiederholt habe.* Von den 13 Fällen war der Lungenverlauf in 6 Fällen auf der einen Seite stärker und von diesen haben 4 Fälle wiederholt so reagiert, dass auf der Haut der gesunderen Lungenhälfte auch die Reaktion eine ausgeprägtere war. Diesbezüglich verdient insbesondere der Patient No. 8 eine besondere Beachtung, bei welchem ich dreimal nacheinander dasselbe Resultat bekommen habe. *Die Stärke der Brustkutanreaktion steht in einem solchen Verhältnis zu der der Armkutanreaktion,* wie 3 oder 4 zu 1.

Diese erhöhte Empfindsamkeit macht die Brustkutanreaktion ausserordentlich wertvoll, ohne Rücksicht darauf, worin die Ursache dieser gesteigerten Reaktionsfähigkeit liegt. Dass keinesfalls in der grösseren Feinheit der Haut, beweisen meine auf der Haut des Oberarms, des Unterarms, des Halses und der Brust vorgenommenen gleichzeitigen Impfungen (siehe Tabelle VII).

TABELLE VII

OBERARM-, UNTERARM- UND HALSKUTANREAKTIONEN

Oberarm.	*Unterarm.*	*Hals.*	*Sine morbo Laryng.*	*Tuberc. Laryng.*	*Total.*
±	±	±	19	3	22
++	+	+	4	3	7
+	++	+	3	2	5
+	+	++	4	—	4
++	++	+	10	3	13
++	+	++	3	1	4
+	++	++	1	—	1
					56

Obzwar die Haut des Oberarms und die des Halses eine feinere ist, als die des Unterarmes, sogar die Haut des Halses oft eine feinere ist, als die der Brust, war die an den vorangehenden drei Stellen erfolgte Impfung gewöhnlich von gleicher Stärke, im Verhältnis war sogar am häufigsten die Halskutanreaktion die schwächste.

Meiner Ansicht nach besteht die Ursache der erhöhten Empfindlichkeit der Brustkutanreaktion in dem grösseren Blutgehalt der Brusthaut.

Die in den 203 Fällen erfolgten beiderseitigen Brustkutanimpfungen bezeigen, dass von allen Lokalreaktionen dies die verlässlichste ist, und zwar ist die Prognose eine umso bessere, je stärker die + Reaktion in den einzelnen Fällen ist.

Wie sich aus meinen Untersuchungen ergeben hat, vermag diese Armkutanreaktion die Richtigkeit der auf Grund der Brustkutan aufgestellten Prognose nur bekräftigen, nicht aber herabsetzen: Dasselbe kann auch von den Ophthalmo- und Rhinotamponreaktionen gesagt werden, aus welchem Grunde diese lokale Impfung immer an diesen vier

Stellen auszuführen ist, wenn man in Bezug auf Diagnose und Prognose genaue und verlässliche Resultate erlangen will (Tabelle VIII).

TABELLE VIII

GEMEINSAMES VERHALTEN DER KUTANEN BRUST-, ARM- UND OPHTHALMO-RHINOREAKTION IN 203 FÄLLEN

Reaktionen.	*Pulmo nihil.*	*Pulmo suspect.*	*I. Stad. Cat. ap. min. gr. Bacill.*	*II. Stad. Gaffky I, II. Cat. apic.*	*III. Stad. Gaffky III, IV. Inf. apic.*	*III. Stad. Gaffky I–X. Inf. ap. et. tuber. laryn.*	*Total.*
Alle vier Reaktionen stark ++	6	9	10	2			27
Alle vier Reaktionen mittelstark			6	2			8
Alle vier Reaktionen —		1			11	17	29
Alle drei Reaktionen + Armkutan —	1	4	9		1		15
Alle drei Reaktionen schwach + Armkutan —					3	9	12
Alle drei Reaktionen stark + Ophthalmoreaktion —	5	2	9	1			17
Alle drei Reaktionen schwach + Ophthalmoreaktion —					9	2	11
Brust- u. Armkutanreakt. stark + Ophthalmo-Rhinoreaktion —	3	3					6
Brust- und Ophthalmoreaktion stark + Arm- u. Rhinoreaktion —	2	3					5
Brust- u. Rhinoreaktion stark + Arm- u. Ophthalmoreaktion —			5	2			7
Brust- u. Armreaktion schwach + Ophthalmo-Rhinoreaktion —				4	3	9	16
Brust- u. Ophthalmoreaktion schwach + Arm- u. Rhinoreaktion —					3	3	6
Brust- u. Rhinoreaktion schwach + Arm- u. Ophthalmoreaktion —				4	4		8
Rhino-Ophthalmoreaktion schwach + Brust- u. Armreaktion —						3	3
Brustreaktion schwach + Arm-, Ophthalmo- und Rhinoreaktion —	3	3	4			1	11
Brustreaktion schwach + Arm-, Ophthalmo- und Rhinoreaktion —				1	9	9	19
Alle drei R. schwach + Ophthalmoreaktion stark +					1	1	2
Brust- u. Rhinoreaktion stark + Armreaktion schwach + Ophthalmoreaktion —	1						1
							203

Man wolle die Tabellen gut beachten. Auf den ersten Augenblick wird es auffallen, dass wenn alle vier lokale Reaktionen stark (+ +) sind, man es mit dem mildesten Grade der Tuberkulose zu tun hat, oder sie ist clinice gar nicht nachweisbar. Wenn alle vier Reaktionen negativ sind, so deutet dies auf die grosse Schwere der Krankheit hin. Je schwächer die vier lokalen Reaktionen sind, umso schwieriger ist der Fall. Auch in dem Falle, wenn die vier lokalen Reaktionen Kräftigkeitsgrade zeigen, kann man den Grad der Tuberkulose und die Prognose mit der grössten Wahrscheinlichkeit feststellen — aber auch in diesen Fällen ist das Verhalten der Brustkutanreaktion von grösster Wichtigkeit.

Schliesslich will ich noch erwähnen, dass ich in 195 Fällen wie ich glaube als erster die *Otoreaktion* versucht habe, jedoch ist wahrscheinlich das sehr blutarme Trommelfell Ursache, dass ich eine + Reaktion kaum in zwei bis drei Fällen bekommen habe.

TABELLE IX

Zustand.	+(*stark*)	+(*schwach*)	—	*Total.*
Sine morbo lar.	4	9	110	123
Laryng. chron.	—	—	42	42
Laryng. sub-acut.	3	2	4	9
Perichondr. lar.	7	4	24	35
Ulcer. chord.	1	2	24	27
Erosion et infiltr. lar. . . .	1	—	10	11
Chord. lat. dextri seu sinistri . .	—	2	7	9
Anaemie lar.	—	—	4	4
	16	19	225	260

Larynxreaktion (Tabelle IX) habe ich bei 260 Individuen versucht, jedoch ebenfalls die Erfahrung gemacht, dass sie bei einer Behandlung mit einer 1 prozentigen Lösung bis hinauf zur unverdünnten Alttuberkulinlösung — ob nun der Kehlkopf eingepinselt wurde, oder ob die Tuberkuline instilliert wurde — sehr selten eine + Reaktion ergab, sogar bei wiederholten Einimpfungen (Tabelle X) das Ergebniss zumeist ein negatives war. Von den 260 Fällen (siehe Tabelle) habe ich in 225 Fällen überhaupt keine Reaktion erhalten. Die meisten + Reaktionen habe ich in den Fällen der Perichondritis erhalten, von welchen 35 Krankheitsfällen 11 positiv reagiert haben, aber niemals in einem solchen Grade, dass die Reaktion den Kranken sonst irgend eine Unannehmlichkeit bereitet haben würde.

TABLE X

Kranke.	*Tuberkulin 2%. Reaktion.*	*Tuberkulin 10%. Reaktion.*	*Tuberkulin 25%. Reaktion.*	*Tuberkulin 50%. Reaktion.*	*Tuberkulin concentr. Reaktion.*
1912. *B.* Gaffky II, III. Inf. ap. d. Inf. chord. l. utr. Perichondr. aryten.	x. 14 —	x. 16 —	x. 18 —	x. 21 —	x. 30 —
1912. *N.* Gaffky IV. Cat. ap. utr. Infiltr. chord. l. utr. Perichondr. aryten.	x. 11 —	x. 14 —	x. 16 +	x. 23 ++	
1912. *Z.* Gaffky II. Cat. ap. utr. Ulcer. chord. l. utr.		xi. 6 - -	xi. 8 —	xi. 11 —	xi. 13 +(sehr schwach) xi. 15 —
1912. *O.* Gaffky IV. Inf. ap. lat. utr. Perich. aryten. l. d. Ulcer. chord. l. d.			x. 1 — x. 14 —		
1912. *G.* Gaffky V. Inf. ap. l. utr. Perich. aryten. l. sin. Ulc. chord. l. sin.			x. 18 —	x. 20 — x. 22 —	
1912. *K.* Gaffky III. Inf. ap. l. utr. Ulc. chord. l. utr.			xi. 4 —	xi. 6 +(schwach) xi. 8 +(schwach)	xi. 15 — xi. 18 —

SECTION XV

RHINOLOGY AND LARYNGOLOGY

INDEPENDENT PAPER

DIE THERAPEUTISCHE WIRKUNG DES BACTERIUM COLI COMMUNE IN FÄLLEN DER KEHLKOPF-TUBERKULOSE

Von Dr. ELEMÉR VON TÓVÖLGYI, Budapest

Geehrte Herren! Mit Rücksicht auf den Umstand, dass ich in der mir zur Verfügung stehenden Zeit — eigentlich das Material von zwei ganz separaten Vorträgen — die Resultate meiner seit beinahe drei Jahren in dieser Richtung fortgesetzten Tätigkeit dem sehr geehrten Kongresse bekannt zu machen wünsche, kann ich mich bloss darauf beschränken, dass in diesem ersten Teile meines Vortrages von jenen auffallenden Besserungen Rechenschaft ablege, welche ich bei der Kehlkopf-Tuberkulose mit der Bakterotherapie, und namentlich mit dem Bacterium coli commune, erzielte. All jenen geehrten Herren Kollegen, welche sich für meine diesbezügliche Forschungen interessieren, diene es hiermit, dass im Laufe des Herbstes 'die Resultate meiner Untersuchungen' in der *Monatsschr. f. Ohrenheilk.* in ihrem ganzen Umfange erscheinen werden.

Mit den therapeutischen Versuchen mit der Bakterie coli commune habe ich im Dezember des Jahres 1910 begonnen, nachdem ich schon vorher im Jahre 1902 als erster mit der Milchsäurebacille in den Fällen der Kehlkopf-Tuberkulose — und es sei gleich hier bemerkt, ohne Erfolg — therapeutische Resultate erreichen wollte. Dem folgten die mit den seitens Cohendys und Metchnikoffs stark empfohlenen Bacillus bulgaricus angestellten Versuche (*Orvosi Hetilap*, 1912), dessen wunderbare Wirkung North in den Fällen von Kehlkopf- und Nasenleiden so sehr hervorgehoben hat. Meine diesbezüglichen Versuche waren nicht nur in den Fällen der Kehlkopf-Tuberkulose, sondern auch bei anderen Kehlkopf- und Nasenleiden sozusagen ohne jeden Erfolg.

Welcher Gedanke mich auf den Versuch mit dem Bacterium coli commune geführt hat, kann ich zur Zeit nicht angeben, und will ich nur bemerken, dass die nach der Theorie Metchnikoffs bei den gesunden Menschen auffindbaren Darmbakterien die toxische Stoffe erzeugenden

Bakterien zugrunde richten und dass gleichzeitig die Darmbakterien die im Blute zirkulierenden Antikörper hervorrufen.

Dass die Anschauung Metchnikoffs eine sehr begründete war, beweisen meine Versuche, im Vereine damit, dass diese wohltätige Rolle der durch Metchnikoff näher nicht bestimmten Darmbakterien — mit der grössten Wahrscheinlichkeit — dem Bacterium coli commune zu verdanken ist. Gleich hier soll jener Teil der Metchnikoff-Theorie hervorgehoben werden, dass nur die in dem gesunden Darm auffindbaren Bakterien diese antagonistische Eigenschaft aufweisen. Meine Versuche haben auch dies bewiesen, indem ich mit dem aus dem katarrhalischen Darm gezüchteten, also schon als pathogen zu betrachtenden Coli commune nie eine Besserung erreichen konnte, sogar nach dem zweiten, dritten Einpinseln das Kehlkopfleiden sich verschlechtert hat, so dass ich die Behandlung in dieser Weise einstellen musste.

Bisher habe ich in mehr als 80 Fällen der Kehlkopf-Tuberkulose und in einem Falle des Gesichtslupus die therapeutische Wirkung des Bacterium coli commune ausprobiert.

Zu Beginn meiner Versuche — als ich noch über keine genügende Praxis in dieser Hinsicht verfügte — habe ich mich angesichts der anfänglichen, wirklich auffallenden Besserungen der Hoffnung hingegeben, dass ich die Kehlkopf-Tuberkulose in ein bis zwei Monaten gänzlich zu heilen vermag. Obzwar sich diese Hoffnung in den meisten Fällen nicht zur Gänze eingestellt hat, sind meine Besserungsergebnisse derartige, wie solche bisher — vielleicht mit Ausnahme des in letzterer Zeit in Verkehr gebrachten Ulsanin — mit keinem Mittel erreicht werden können. Die anfängliche rasche und auffallende Aenderung besteht in erster Reihe darin, *dass der Kehlkopf auffallend rein wird.*

Uns allen, die wir die Kehlkopf-Tuberkulose leider genug oft vor Augen bekommen, ist das Bild nicht unbekannt, auf welches der Ausdruck am besten passt, in Siechheit begriffen.

Als wenn der Kehlkopf von tausenden Schimmel-Pilzen überflutet wäre, ein solches Bild zeigen die mit schmutzig grauem Niederschlag belegten, eine träge Granulation besitzenden, oedematöse, rostfarbigen Schleimhäute. Die scharfen Grenzlinien des Kehlkopfes sind verschwommen, sowohl die wirklichen Stimmbänder, wie auch die Taschenbänder sind geschwollen, mit einem Worte, was wir sehen erweckt in uns immer den ersten Eindruck, dass der Widerstand des Organismus der Tuberkulose gegenüber in diesen Fällen sozusagen dem Nichts gleich steht. Dieses Bild erfährt an dem der ersten Coli-Einpinselung folgenden zweiten bis dritten Tage eine vollständige Veränderung.

Die Absonderung verschwindet zur Gänze, die Farbe der Schleimhäute nähert sich der gesunden, die Schwellungen verringern sich oft schon nach der ersten Pinselung in einem solchen Grade, dass die wirklichen Stimmbänder, die zufolge Schwellung der Taschenbänder nicht sichtbar waren, zum Vorschein kommen und dabei bessert sich das allgemeine Wohlbefinden des Kranken ausserordentlich. Diese letztere Verbes-

serung ist *dem raschen Aufhören der Dysphagie zu verdanken; dies ist das zweite wichtigste Zeichen der Wirkung des Coli.* In den vierundachtzig Fällen bin ich kaum auf eine solche Dysphagie gestossen, die nicht in Kürze aufgehört hätte oder nach den ersten zwei bis drei Pinselungen sich wesentlich nicht gelindert hätte. Und wunderbarerweise war in den Fällen, wo die Decrepidität die grösste war, wo die perichondritische Veränderung in dem grössten Ausmass bestand, die rasche Herabsetzung der Dysphagie am auffallendsten. Das rasche Aufhören der Dysphagie hat sich auch dann eingestellt, wenn sich die Schwellung nicht in auffallender Weise verringert hat. In diesen Fällen haben die Kranken behauptet, dass ihnen das Schlucken schwer fällt, dass der Bissen oft in dem Kehlkopfe stecken bleibt, aber keine Schmerzen verursacht. In den ganz schweren Fällen — in welchen der Kranke dem Exitus nahe steht — währenddem die Dysphagie auffallend rasch aufhörte, tritt diese nach vier bis fünf Tagen oder nach einer Woche ebenso rasch wieder auf und das Coli hat nun, von ganz unwesentlichen Besserungen abgesehen, all seinen wohltätigen Einfluss bereits verloren. In den Fällen, wo diese Krankheit nicht derart vorgeschritten ist, kehrt die Dysphagie oft nach einigen Wochen oder nach ein bis zwei Monaten wieder zurück, wenn der allgemeine Zustand sich während dieser Zeit verschlechtert hat, hört aber bei der neuerlichen Coli-Einpinselung auch in den letzteren Fällen oft von neuem auf oder verringert sich in hohem Masse.

Die dritte auffallende Veränderung, die nach den ersten zwei bis drei Einpinselungen gewöhnlich schon auftritt, besteht *in der wesentlichen Besserung der Stimme.* Kranke, die bisher ganz aphonisch waren, können nach zwei bis drei Tagen, selbstverständlich heiser, aber laut sprechen.

Die vierte Veränderung betrifft die Geschwüre. Wie bereits erwähnt, *zeigt sich die Reinigung der Geschwürgrunde am ehesten,* darauf — und in dieser Hinsicht ist die Zeitdauer eine sehr schwankende, von zwei bis drei Wochen bis zu einem halben Jahre — beginnen *auch die Geschwüre langsam zu heilen,* die oberflächlichen werden rascher, die tiefer liegenden langsamer kleiner, die ersteren verschwinden auch ganz, ohne eine Narbe zu hinterlassen. Dieser letztere Fall ist aber ein ziemlich seltener. In den Fällen der hochgradigen Lungeninfiltrationen und der ausgedehnten Kehlkopf-Tuberkulose werden die Geschwüre nach ihrer der Coli-Pinselung folgenden Reinigung — wie ich die Erfahrung gemacht habe — oft noch rascher zerfallen, an den Stellen des Geschwüres werden Substanzverluste auftreten und wenn sich der Zustand des Kranken mittlerweile verschlechtert, steigert sich die Zerfallung, während bei unverändertem oder verbessertem Zustand diese Substanzverluste zwar verbleiben, jedoch begrenzt werden.

Also die Kehlkopfschleimhaut ist bestrebt, sich von der Geschwürenveränderung zu befreien, wenn sich aber der Widerstand des Organismus sehr geschwächt hat, dann leitet der Zerfall des Geschwürs nicht die Heilung, sondern eher eine hochgradige Verschlechterung ein. Auch bei

den Ulsanin-Einpinselungen heilen die Geschwüre derart — ich habe es wenigstens in mehreren Fällen so erfahren — dass wenn sie den Rändern nahe liegen, z. B. an dem Rande der Stimmbänder sitzen, zur Gänze sich absondern. Das Ulsanin wird — gleich hier will ich es bemerken, wenn von solitären Geschwüren die Rede ist und die perichondritische Veränderung keine hochgradige ist — in vielen Fällen die tuberkulotischen Geschwüre rascher und gründlicher heilen, als Coli; bei grösseren Veränderungen hingegen habe ich Coli bisher immer besser gefunden als Ulsanin. Die therapeutische Wirkung erreichen die zwei Mittel auf verschiedene Weise, was mit Rücksicht darauf, dass das eine lebende Bakterie, die andere hingegen eine chemische Verbindung ist, sozusagen selbstredend ist. Die beiden Verfahren können, wie ich es später erfahren habe, einander gründlich unterstützen, und zwar wo Ulsanin nicht mehr nützt, wird Coli-Pinselung vorgenommen und umgekehrt, was zumeist erfolgreich ist.

Die zufolge der Coli-Einpinselungen eintretende anfänglich auffallende Besserung wird sich aber im Verlaufe der weiteren Behandlung einstellen oder es wird später die Besserung nicht mehr so rasch fortschreiten, ja sogar bisweilen ein Rückfall eintreten. Was die Ursache hievon ist — ich muss es gestehen — konnte ich bisher nicht ausfindig machen, am wirksamsten zeigen sich gewöhnlich die ersten zwei oder drei Behandlungen und wenn die Besserung in diesem Masse fortschreiten würde, dann müsste — wie ich dies zu Anfang meiner Versuche auch gehofft habe — innerhalb vier oder sechs Wochen die vollkommene Heilung eintreten. Ob es im Falle der Aufhörung oder der weiteren Verlangsamung der Besserung eine Rolle spielt, dass sich der Organismus an die Colibakterien gewöhnt hat oder ob die wohltätige Heilwirkung des Coli damit verschwindet, dass sich der Schutz des Organismus eben der Coliinfektion gegenüber einsetzt und dadurch das in die Zellen neuerdings hineingelangende Coli, bevor dieses seine Wirkung ausüben würde, rasch vernichtet, kann ich zur Zeit noch nicht entscheiden.

Die diesbezüglich angestellten Versuche haben gar keinen Aufschluss gegeben. In fünfundzwanzig Fällen habe ich — wie die Tabelle zeigt — parallel mit den Pirquetimpfungen auch Colikutanimpfungen versucht, und obzwar das Colikutan in einzelnen Fällen positiv, in den anderen Fällen negativ war, konnte ich zwischen dem Verhalten des Kehlkopfleidens dem Coli gegenüber und zwischen der Reaktionsfähigkeit des Organismus auf das Colikutan gar keinen Zusammenhang finden.

Aus der Tabelle I, bei welcher ich neben der 50 prozentigen Tuberkulinimpfung wiederholt auch mit dem lebenden Coli Kutanimpfungen ausgeführt habe, geht nur hervor, dass in den schwersten, dem Exitus nahestehenden Fällen auch das Colikutan immer negativ und nie positiv sein wird, wie dies in den leichteren Fällen, wenn auch nur selten, jedoch vorkommt. Dies würde jedenfalls dafür sprechen, dass der Organismus auf das Coli etwas ähnlich reagiert, wie auf die Tuberkuline. Es ist aber zu beachten, dass die mit dem Toxin der Bakterie der Tuberkulose

und mit dem lebenden Coli erfolgte gleichzeitige Impfung, wie dies in diesen Fällen geschehen war, einen ausschlagenden Beweis in dieser Richtung niemals geben kann. Dies kann nur so möglich sein, dass entweder mit dem Toxin beider Bakterienarten oder mit den lebenden Formen beider Arten zu gleicher Zeit geimpft wird. Ich habe auch dies versucht, indem ich in sechszehn Fällen an der einen Hälfte der Brust des Kranken eine lebende Kochbacille und an der anderen eine lebende Coli eingeimpft habe. Auf die lebende Kochbacille hat der Organismus viel schwächer reagiert, als auf die Tuberkuline. Beinahe so schwach als auf die Coli. Von den 16 Fällen hat in 6 Fällen die Brusthaut auf die Kochbacille und in 4 Fällen auf die Coli in Spuren reagiert, und zwar in drei Fällen ist die zweiseitige + Reaktion zusammengefallen. Wo die Reaktion + war, das waren alle Fälle der leichteren Erkrankung. Dies würde rein dafür sprechen, dass die Coli ähnliche Reaktionen im Körper verursacht, als die Tuberkuline. Dies alles will ich nur nebensächlich erwähnt haben, da aus der Anzahl meiner Fälle noch keine bestimmte Folgerung gezogen werden kann.

Ich verfüge auch über 279 solcher Fälle, wo ich insgesamt einmal zugleich mit dem lebenden Coli und der 50 prozentigen Tuberkuline geimpft habe, und zwar immer am Unterarm. Von den 279 Fällen waren in 31 Fällen beide Reaktionen positiv, in 134 Fällen negativ, in 94 Fällen die Coliimpfung und in 20 Fällen die Tuberkulinimpfung eine negative. Ich habe mich aber sogar mit diesem nicht begnügt und zwecks Studiums der Einwirkung der zweierlei Arten von Bakterien auf den Organismus auch mit der Mischung des lebenden Coli und der 50 prozentigen Tuberkuline in 205 Fällen Kutanimpfung ausgeführt; von dem Ergebnis hievon werde ich zufolge Zeitmangels nachträglich im Rahmen eines Artikels Rechenschaft abgeben.

Vorläufig will ich nur soviel bemerken, dass von den lebenden Coli-Kutanimpfungen weder auf die Schwere des Leidens, noch darauf, ob die positiv Reagierenden besser durch Colipinselungen heilen würden, keine Folgerung gezogen werden kann.

Zurückkommend auf meine mit dem Bacterium coli commune erreichten Resultate, will ich wiederholt betonen, dass die anfängliche grosse Besserung rasch aufhört, sogar bei meinen anfänglichen Fällen, wo ich noch über wenig Praxis verfügte, der Zustand öfters sich verschlechtert hat. Aber nur dort, wo ich die Coli-Einpinselungen jeden zweiten Tag, also zu oft, vollführte. Später habe ich es erlernt, dass eine wiederholte Einpinselung sogar anfänglich nicht innerhalb einer Woche ratsam ist. Es ist immer der Zeitpunkt abzuwarten, bis der Besserungsprozess zum Stillstand kommt.

Von der dritten bis vierten Pinselung an darf man in den Fällen, in welchen sich die Kehlkopf-Tuberkulose nicht mehr bessern will oder von neuem sich zu verschlechtern beginnt, zwischen zehn Tagen bis drei Wochen keine neuerliche Behandlung anstellen. Ob während dieses Zeitraumes nicht eine Ulsaninpinselung zu empfehlen ist, darüber

TABELLE I

DAS VERHALTEN DER WIEDERHOLTEN PIRQUET'SCHEN UND COLIKUTANEN IMPFUNGEN IN 25 FÄLLEN DER LUNGEN- UND KEHLKOPF-TUBERKULOSE

Kranke.	*Datum.*	*Pirquet 50%.*	*Coli commune.*	*Wirkungslos oder kleine Besserung.*	*Anfangs grosse Besserung, später Heilung.*	*Anfangs grosse Besserung, später Stagnation.*	*Anfangs grosse Besserung, dann Verschlimmerung.*	*Fortwährende Besserung.*
Frau Kl.	1912 :							
	ix. 6	−	−			+		
	ix. 13	+	−					
	ix. 27	+	−					
	x. 16	+	+					
	x. 28	+	−					
	xi. 18	+	−					
H. Bar	Bei fünfmaliger Impfung beide Reaktionen negativ.						+ Exitus	
H. Czeb	1912–13 :							
	ix. 20	+	+		+			
	x. 14	+	+					
	xi. 8	+	+					
	xi. 18	+	−					
	ii. 5	+	−					
	ii. 17	+	+					
Frau Kirsch	1912 :							
	x. 14	−	+			+		
	x. 28	−	+					
	xi. 18	+	−					
Frau Bi.	1912 :							
	x. 14	+	−			+		
	x. 30	+	−					
H. Ge.	1912 :							
	ix. 30	+	−	+				
	x. 4	+	+					
	x. 28	+	−					
	xi. 8	+	+					
H. Hc.	1912 :							
	ix. 17	−	−				+	
	ix. 27	+	−				Exitus	
	x. 4	−	−					
Fr. Hel.	1912 :							
	ix. 4	−	−			+		
	ix. 16	−	+					
	x. 9	+	+					
	x. 28	+	+					
Fr. Be.	1912 :							
	x. 16	−	−				+	
	x. 18	−	−				Exitus	
	x. 28	−	−					
H. Par.	1912 :							
	x. 18	−	−				+	
	x. 28	−	−				Exitus	
	xi. 8	−	−					
H. Szol.	1912 :							
	x. 28	−	−				+	
	xi. 16	+	−					
	xi. 18	+	−					
Fr. Kop.	1912–13 :							
	xi. 4	+	−			+		
	xi. 18	+	+					
	iii. 3	+	−					
	iv. 12	−	−					

TABELLE I (*Forts.*)

Kranke.	*Datum.*	*Pirquet 50 %.*	*Coli commune.*	*Wirkungslos oder kleine Besserung.*	*Anfangs grosse Besserung, später Heilung.*	*Anfangs grosse Besserung, später Stagnation.*	*Anfangs grosse Besserung, dann Verschlimmerung.*	*Fortwährende Besserung.*
H. Ol.	1912 :	Conc. T.						
	x. 16	—	—				+	
	xi. 11	+	—				Exitus	
	xi. 18	—	—					
H. Ga.	1913 :	25% T.						
	i. 5	+	—			+		
	ii. 19	+	—			Nach der		
	ii. 28	+	—			Provinz		
						verzogen		
H. K.	1913 :	50% T.						
	ii. 28	+	+		+			
	iv. 30	+	—					
Fr. Veib.	1913 :							
	iii. 31	—	—				+	
	iv. 7	—	—					
	v. 19	—	—					
H. Iá.	1913 :							
	iii. 12	+	—					+
	iv. 7	—	—					
	v. 19	+	—					
Fr. Lak.	1913 :							
	iii. 29	—	—					+
	iv. 28	—	—					
Fr. Re.	1913 :							
	iv. 2	—	—	+				
	iv. 19	—	—					
H. Dr.	1913 :							
	iv. 30	+	—	+				
	v. 17	+	—					
H. Klt.	1913 :							
	iii. 17	—	—			+		
	v. 26	—	—					
Fr. Dep.	1913 :							
Cat. ap.	iv. 2	—	—			Sine morbo lar.		
	iv. 30	+	—					
H. Holt	1913 :							
Inf. ap. l. d.	iv. 2	—	—			Sine morbo lar.		
	iv. 19	+	—					
Fr. Bert.	1913 :							
Inf. ap. sin.	iv. 2	+	—			Sine morbo lar.		
	iv. 19	+	+					
H. Fi.	1913 :							
	iii. 28	—	—	+				
	iv. 23	—	—					

stehen mir noch nicht eingehende Erfahrungen zur Verfügung, soviel kann ich aber sagen, dass es besser ist, in der Zwischenzeit gar keine Einpinselungen vorzunehmen, als mit Milchsäure, Menthol, Phenolum, Natriosulforicinicum oder mit ähnlichen Mitteln Versuche anzustellen. Dies würde die vom Coli geschaffene Besserung zunichte machen.

Am wirksamsten erweist sich immer die frisch gezüchtete Coli, und zwar deren eher die sich viel indolbildende Form. Die einige Tage alten Kulturen sind schon sehr abgeschwächt. Eine Wirkung haben

zwar auch diese, jedoch eine viel schwächere und sie kann dementsprechend auch öfters Verwendung finden. Die zweitäglich vorgenommenen Impfungen der frischen Colikultur sind acht bis zehn Tage verhältnismässig noch genügend stark, schwächen aber von dieser Zeit an rapid ab. Die Wirkung der nicht indolbildenden und auch keine selbstständige Bewegung ausführenden Coliarten ist ebenfalls eine geringere. Ich muss es gestehen, dass in der Wirkung — in Anbetracht der ausserordentlichen Variationen der Coliarten — ebenfalls auffallende Aenderungen eintreten. Oft zeitigt die eine oder andere Zucht die eklatanteste Wirkung, andersmal dauert es auch zwei Wochen, bis mir wieder eine Kultur zu Händen kommt, die ebenfalls eine derart grosse Wirkung zeigt. Man muss auch sehr darauf achten, dass man immer mit reinen Kulturen arbeitet, auf diesen Umstand haben sowohl ich, als Kollege Assistent Tibor Kern von der I. Innern Klinik, welcher die Züchtungen durchgeführt hat und dem ich auch an dieser Stelle wärmsten Dank ausspreche — sehr sorgfältig geschaut.

In den Fällen, in welchen die sehr virulente Coli nicht die erhoffte Besserung brachte, oder das Leiden eventuell noch eine Verschlechterung erfuhr, ist es ratsam, eine paar Tage alte Kultur anzuwenden. In dieser Beziehung hat man es auch mit dem Coli nicht anders, als mit allen anderen Medikamenten, auf die die verschiedenen Individualitäten verschieden reagieren.

Professor H. Krause, den ich stolz als meinen Meister bekenne und welcher mit regem Interesse meine Versuche verfolgte, hat mir letzthin geraten, neben der Colibehandlung auch Arsen den Kranken innerlich zu verabreichen. In den Tagen habe ich auch mit dieser Modifikation die Versuche angestellt, über diese kann ich jedoch wegen der Kürze der Zeit selbstverständlich noch nicht berichten.

Die schönsten Erfolge habe ich bei den tuberkulösen Aenderungen des Schlundes gefunden und die hier gefundenen grossen Besserungen habe ich auch Herrn Professor August von Székely, dem Direktor des Pasteur-Instituts in Budapest, vorgelegt, derselbe hat auch mehreremals den Fall des Gesichtslupus kontrolliert, bei welchem jede Einpinselung nach einigen Tagen eine eklatante Besserung bewirkte.

Das Gesicht trägt zwar noch immer Lupusveränderungen, jedoch in viel schwächerem Grade, als früher, nur in Gestalt von roten Flecken, aber auch diese sehen stark ihrer Heilung entgegen. Leider vermag der überaus arme Patient nicht so oft aus der Umgebung hereinzukommen, als dies für die Behandlung wünschenswert wäre, es verstreicht oftmals ein halbes Jahr zwischen zwei Pinselungen. Zu dieser Zeit steht er zwei Jahre unter meiner Behandlung und während sich die an anderen Stellen seines Körpers zeigenden vielen tuberkulösen Knoten alle verschlechtern (der eine Arm musste auch amputiert werden), ist der Gesichtslupus der einzige, welcher geheilte Stellen zeigt, während derselbe sich zufolge der früheren Röntgen-, Finsen- und anderen Therapien fortwährend verschlechterte. Ich weiss gut, dass aus diesem einen Falle keine Folgerung

gezogen werden kann, jedoch in Anbetracht der auffallenden Besserungen, die ich in den Fällen der Kehlkopf- und der Pharynx-Tuberkulose erreicht habe, dass sogar alle meine Fälle sehr schwierige waren und ich trotzdem auch über Heilungen berichten kann, macht es wünschenswert, dass die mittels Colipinselungen gemachten Heilversuche ebenfalls in je breiteren Kreisen angestellt werden.

Meinen Resultaten gegenüber bin ich absolut nicht eingenommen und habe daher eher weniger, als mehr gesagt, — die nachfolgenden exakten Forscher sollen es entscheiden, wie weit ich im Rechte war.

Beschuldigungen, welche gewöhnlich die Bahnbrecher neuer Heilmethoden treffen, dass auch in den Fällen der Kehlkopf-Tuberkulose oft ohne jeden Eingriff nicht nur Besserungen, sondern auch Heilungen eintreten können, solche Beschuldigungen können mich nicht treffen, wenn ich über mehr als achtzig solche Fälle berichten kann, in welchen ich Bacterium coli Monate hindurch anwenden konnte und bei welchen ich während einiger Tage oder Wochen eine derartige wesentliche und rasche Besserung erzielte, die von selbst nie so rasch einzutreten pflegen.

Und abgesehen hievon waren die meisten mit Coli behandelten Patienten mit einer derart schweren Tuberkulose behaftet, in welchen Fällen die von sich selbst eintretende Heilung eine derart seltene ist, dass sie gar nicht in Betracht kommen kann.

Schliesslich will ich noch bemerken, dass ich die Colibouillon-Kultur bei den Pinselungen angewendet habe, und dass diese letztere Operation genügend energisch den kranken Teilen des Kehlkopfes mitgeteilt werden muss. Ich habe auch die Milchkultur des Coli versucht, diese aber nicht so gut befunden, als die Bouillonkultur.

Einige Krankengeschichten sollen an dieser Stelle angeführt werden.

Mein in der *Monatsschrift für Ohrenheilkunde, Laryngologie* etc. erscheinender Artikel wird mehr als 80 Krankengeschichten enthalten.

Mit Coli behandelte Fälle der Kehlkopf-Tuberkulose

I.—1911. i. 17. Zs. M., 22 jährige Kranke. Gaffky VI. Infiltr. apic. l. utr. Ulcer. pharyng. Perichondr. aryten. et epigl. Infiltr. chord. l. utr. Ulc. chord. spur. l. utr. Dysph. + + +. Die Hälfte des Rachens und der Kehlkopf mit grünlich schmutzigem graulichen Niederschlag besetzt.

1911. i. 17. Mit dreitägigem ½ Stunde bei 60° C. erhitztem Bact. coli commune ausgeführte Rachen- und Kehlkopfpinselung.

i. 24. Besserung auffallend. Im Rachen starke Reaktion. Absonderung weder im Rachen, noch im Kehlkopf.

i. 31. St. idem. Lebende Coli im Rachen und Kehlkopf.

ii. 4. Die Schwellung des Rachens vermindert, Infiltration verkleinert, Ulceration rein; perichondritische Schwellung des Kehlkopfes gelindert.

ii. 21. Patientin sagt mit Freude, dass seit 2 Wochen die Dysphagie vollkommen aufgehört hat. Rachen ist rein, allgemeines Befinden sehr gut. Ulceration kleiner, gesund aussehende Granulationen. Kehlkopf heilt langsam, jedoch ununterbrochen.

vi. 21. Gegenwärtiger Zustand. Im Kehlkopf Ulceration überhaupt nicht sichtbar. Schlund-Infiltration ein Viertel des alten. Ulceration in der Mitte der Infiltratio nur linsengross. Während der Behandlung hat sie 4 kg zugenommen. Ihr Appetit ist ein guter, das Schlucken ein freies.

vi. 26. Ihre Stimme wird immer reiner. Patientin hat sich auf sieben Wochen aufs Land begeben.

viii. 12. Allgemeines Aussehen gut. 1 kg zugenommen. Von neuem gewachsene Infiltration. Auch Uvula ist infiltriert. Zustand des Kehlkopfes st. idem. Die Lungeninfiltration hat sich gesteigert. Auf die neuerliche Colibehandlung tritt, wenn auch nicht so rasch als früher, von neuem eine Besserung ein. Der infiltrierte Teil der Uvula hat sich abgetrennt, die dort entstandene Ulceration vernarbt. Die Schwellung des Kehlkopfes hat ganz aufgehört. Geschwürwunden sind nicht zu sehen. Die Schleimhaut des Kehlkopfes sieht zusammengeschrumpft aus — und ist sehr rein. Am 27. xii. ist die Patientin weggeblieben. Am 2. vii. 1912 ist sie von neuem erschienen. Ihr Aussehen ist ein gutes. Lungen in gleichem Zustand. Kehlkopf status idem. Auf der Rachenwand eine erbsengrosse Infiltration. Allgemeines Befinden gut. Keine Dysphagie. Patientin hat nach einigen Monaten geheiratet ! ! ! Seit dieser Zeit hat sie sich nicht gezeigt. 1913. ix. 16. Patientin zeitdem ganz geheilt.

II.—1911. ii. 28. F. E., 26 jährige Patientin. Seit 3 Monaten heiser. Cat. apic. lat. utr. Infiltr. chord. l. utr. Ulcer. chord. spur. l. sin. Der geschwollene und geschwürige Teil der Taschenbänder hat die vordere Hälfte der Stimmbänder ganz bedeckt. Während der Behandlung hat die Infiltration des rechtseitigen Stimmbandes gänzlich aufgehört. Die linkseitige Taschenbänderulceration ist geheilt und auch die Schwellung der Taschenbänder hat sich so gelegt, dass auch die vordere Hälfte der Stimmbänder sichtbar wurde und nun hat es sich gezeigt, dass die Ulceration auch auf sie übergangen ist. Diese letztere Ulceration wollte nicht heilen.

Am 24. v. hat sich die Patientin entfernt. Am 4. vii. ist sie von neuem erschienen. Der Zustand hat sich verschlechtert. Stimmbänder zeigen um das Geschwür Granulationen. Auf den Taschenbändern von neuem beginnende oberflächliche Ulceration. Patientin hat sich wegen dringenden Angelegenheiten noch an demselben Tage entfernt.

III.—1912. ix. 4. B. G., 55 jähriger Patient. Gaffky V. Infiltr. ap. l. utr. Perichondr. epigl. et aryten. l. utr. Chord. ulcer. pr. l. sin. Dysphagie + + +. Bis 18. xi. ausserordentliche Besserung. Dysph. ganz verschwunden. Vom 18. xi. an verschlechtert sich aber der Zustand rapid und wird nach einigen Wochen Exitus.

IV.—1912. ix. 9. C. G., 45 jähriger Patient. G. VIII. Infiltr. ap. l. utr. Perichondr. aryten. l. utr. Ulcer. chord. l. utr. Viel Absonderung. Keine Dysphagie. 1913. vi. 2. Perichondritis geheilt, Ulceration verkleinert.

V.—1912. x. 14. B. H., 45 jährige Patientin. G. II, III. Cat. ap. l. utr. maj. gr. Ulcerat. chord. l. utr. Arytenoid. Oberflächliche Ulcerationen haben in Kürze ganz aufgehört.

VI.—1912. x. 16. B. K., 24 jährige Patientin. G. VIII. Phth. pulm. Decrepidität. Perich. diff. lar. Ulc. chord. verae et spur. aphon. Dysph. + + +. Zu Beginn der Behandlung Dysphagie aufgehört. Patientin spricht laut. Kehlkopf ganz gereinigt, die Schwellung auf die Hälfte herabgesetzt. Fortwährende grosse Fieber.

xi. 13. Dysphagie zurückgekehrt. Nach einigen Tagen Exitus.

VII.—1912. x. 18. P., 25 jähriger Patient. G. V. Inf. ap. lat. utr. Decrepidität. Perich. aryten. maj. gr. Ulcer. chord. lat. utr. Tbc. miliaris lar. Sehr viel Absonderung. Dysph. + + + ! Schlucken sozusagen unmöglich. Am 21. x. Dysph. ! Kehlkopf sehr rein. Bis 22. xi. d. der Patient befindet sich sehr wohl. Von dieser Zeit an Dysphagie zurückgekehrt — rasch verschlechtert. Am 22. i. 1913 Exitus.

VIII.—1913. 4. ix. Hell., 29 jährige Patientin. G. V. Inf. ap. l. utr. Perich. aryten. l. d. Ulcer. chord. l. d. Erosion. et gran. int. aryten. Dysph. + +. Bis 30. x. Dysphagie gänzlich aufgehört. Patientin ist von neuem in Arbeit getreten.

IX.—1912. 9. xii. S., 31 jährige Patientin. G. III. Infiltr. ap. pr. l. d. Perichondr. aryten. ab cricoid. Ulc. chord. pr. lat. sin. Stenos. lar. Dysph. + +. Dieselbe kann zufolge schweren Atmens nur sitzend schlafen.

1913. ii. 5. Bisher hat sich ihr Zustand fortwährend gebessert; das Atmen erfolgt viel leichter, sie schläft schon liegend. Dysphagie 0. Die Stimme ist sozusagen ganz klar. Am 14. ii. ein kleiner Rückfall, sodann von neuem eine wesentliche Besserung, worauf sie von neuem in Arbeit treten kann.

X.—1913. i. 3. V. J., 28 jähriger Patient. G. I. Cat. ap. l. utr. Infiltr. chord. spur. l. d. Ulcer. chord. lat. sin. Dysphagie 0. Am 2. vi. 1913 Stimmbänder: Bild von Chorditis sonst nihil. Behandlung eingestellt.

XI.—1913. iii. 7. F. N., 45 jähriger Patient. G. IV. Inf. apic. l. utr. Perich. diff. lar. Dysph. + + +. Am 14. iii. 1913 Schwellung sehr vermindert. Stimmbänder wurden sichtbar. Dysphagie 0. Patient ausgeblieben.

XII.—1913. iii. 17. S. S., 23 jähriger Patient. G. —. Cat. ap. l. d. Infiltr. chord. l. utr. Ulcer. chord. vocal. l. d. Infiltr. chord. spur. l. sin. et ulcer. chord. sp. l. sin. Die linke Stimmband- und Taschenband-Infiltration hat aufgehört. Linkseitiger Taschenbandulcus ebenfalls geheilt. Rechtseitiger Stimmbandulcus halblinsengross. An den übrigen Stellen des Ulcus Granulation. Steht unter Behandlung.

XIII.—1913. iv. 21. C. M., 21 jährige Patientin. G. III. Inf. ap. l. utr. Perich. aryten. Pachid. chord. voc. l. sin. Infiltr. chord. lat. utr.

v. 21. Perichondritis aufgehört. Stimmbänder nicht geändert. Am 4. vi. plötzliche Verschlechterung. Dysphagie + + +. Probe mit Ulsanin. Nach ersten drei Behandlungen schlechter — sodann plötzlich besser. Dysphagie wesentlich verringert. Rechtseitiger Ulcus rein — besteht aber auch noch am 29. vi. unverändert. Perichondritis von neuem auf der rechten Seite aufgetreten. Steht unter Behandlung.

XIV.—1913. iii. 7. P. G., 35 jähriger Patient. G. IV. Inf. apic. Ulc. chord. lat. utr. Arytenoiditis. Ulcer. phar. l. utr. Ulc. chord. rechts geheilt; links verkleinert. Ulcer. phar. wesentlich verkleinert.

XV.—1913. iii. 7. Pr. T., 32 jährige Patientin. G. III, IV. Inf. ap. pr. l. sin. Ulcer. chord. l. sin. Ulc. geheilt.

XVI.—1913. iii. 17. Pa. Gy., 52 jähriger Patient. G. —. Inf. ap. l. sin. Perich. diff. lar. Ulcer. epigl. Dysph. + +. Während der Behandlung riesige Besserung. Ulceration geheilt. Dysphagie aufgehört. Perichondritis hat sich auf ¼ zurückentwickelt.

XVII.—1913. ii. 21. Za. J., 35 jähriger Patient. G. IV. Infiltr. apic. Ulc. chord. l. d. Geheilt.

SECTION XV

RHINOLOGY AND LARYNGOLOGY

DISCUSSION No. 5

THE PATHOLOGY AND TREATMENT OF MALIGNANT GROWTHS OF THE NOSE AND NASO-PHARYNX (FIBROMA TO BE EXCLUDED)

(For Report by Professor G. Ferreri, see Part I, pp. 149–202)

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Die Pathologie und Therapie der malignen Tumoren der Nase und des Nasenrachenraumes (mit Ausschluss der Fibrome)

Die ungeahnten Fortschritte der neueren Rhino-Laryngologie der letzten zwei Dezennien hat unsere Spezialwissenschaft zu einer zusehends im Ansehen steigenden Disziplin gemacht. Nicht nur, dass die Rhino-Laryngologie aus einem Teilfach der internen Medizin zu einem solchen der Chirurgie geworden ist, so dass ein moderner Rhino-Laryngolog einer gewissen speziellen Ausbildung in der allgemeinen Chirurgie nicht mehr entraten kann, hat sie sich immer mehr und mehr zu einer selbständigen Wissenschaft aufgeschwungen und nach und nach alles, was zum Schleimhautrohr der oberen Luft- und Speisewege in Beziehung tritt oder treten kann, an sich gezogen. Man würde es heute wohl einem Rhino-Laryngologen, der an die chirurgische Behandlung der Erkrankungen der oberen Luftwege herangeht, verübeln, wenn er nicht die gesamte Chirurgie dieses Gebietes beherrschte, wo er in jedem einzelnen Falle in die Lage kommen kann, Schädeltrepanationen, umfangreiche Plastiken oder fernabliegende Hilfsoperationen (Gastrostomie, etc.) ausführen zu müssen. Und gerade die Chirurgie jener Organe verdankt ihre schönsten Erfolge in der neueren Zeit der wertvollen Befruchtung durch die Rhino-Laryngologie, deren kontinuierliches Studium, Beschäftigung und Erfahrung in der Physiologie und Pathologie jener komplizierten Partien des Kopfes und Halses auch die Chirurgie derselben mehr und mehr den feineren physiologischen Ansprüchen anzupassen vermocht hat. Ich erinnere nur an die Entwicklung der radikalen Behandlung der Nebenhöhleneiterungen, der Methode der Hypophysenoperation, der Resektion des Pharynx und Larynx.

Angesichts aller dieser Erfolge ist der Tiefstand umso bedauerlicher, auf dem sich trotz mancher ermunternder Fortschritte das Problem der malignen Tumoren der inneren Nase befindet, und es muss die vorwärtsstrebende Rhino-Laryngologie mit Befriedigung erfüllen, dass das Komitee eines internationalen Kongresses dieses Problem als Verhand-

lungsthema gewählt hat. Die Prognose der malignen Nasen- und Nasenrachenraum-Tumoren ist ja leider noch immer eine sehr schlechte, ja in manchen Statistiken eine so verzweifelte, dass namhafte Rhinologen ‘das Bestreben zeigen, die Radikaloperation, deren Erfolg in den meisten Fällen ein äusserst zweifelhafter ist, überhaupt zu verlassen und sich der konservativen Therapie zuzuwenden’ (Harmer, 1907). Fälle, wie der von Knight (1911), in dem ein endonasal entferntes Sarkom erst nach 7 Jahren rezidivierte und dann nach der zweiten Operation wieder geheilt blieb, oder der von Wishart, Sarkom der Nase bei einem 17 jährigen Mädchen, welches über acht Jahre rezidivfrei war, oder gar der Salzburg'sche, in dem ein histologisch einwandfrei festgestelltes Plattenepithelkarzinom der unteren Muschel nun schon, wie ich vom Autor persönlich erfahren habe, durch 14½ Jahre rezidivfrei ist, gehören noch immer zu den Seltenheiten, und wenn ein Chirurg wie König unter 48 Fällen von Oberkieferkarzinom mit Stolz von 8 Fällen berichten kann, die durch 10 bis 26 Jahre rezidivfrei geblieben sind, so ist sein Stolz nur zu berechtigt. Die düstere Prognose dieser Tumoren hat verschiedene Ursachen, nicht nur, dass der Gesichtsschädel mit seinen unzähligen Buchten und Knochenlücken und -kanälen einer vielgliedrigen Ausbreitung des Neoplasmas förderlich ist, ist auch infolge der Nähe lebenswichtiger Organe schon oft bei mässiger Ausbreitung in kurzer Zeit des Bestehens der Tumor inoperabel geworden, und bietet die Operation sehr geringe Aussicht auf unmittelbare und dauernde Heilung.

Als mir von dem Komitee des internationalen Kongresses die Ehre zuteil wurde, mit Professor Gherardo Ferreri die malignen Tumoren der Nase und des Nasenrachenraums behandeln zu dürfen, verhehlte ich mir nicht die Schwierigkeiten, die mir hiebei erwachsen würden. Sie beginnen schon bei der Begrenzung und Präzisierung des Themas. Wenn wir auch die malignen Tumoren der äusseren Nase, die zum grossen Teil die Domäne der Dermatologie sind, ausschalten, gleichwie die sekundären Tumoren, so werden wir doch nicht umhin können, ihrer kurz Erwähnung zu tun, da ja auch ein Epitheliom der äusseren Nase gelegentlich in die Nebenhöhlen einwuchern und bis zur Keilbeinhöhle vordringen kann und dann natürlich unter denselben Gesichtspunkten zu behandeln ist, wie ein primär in der Nasenhöhle entstandenes Neoplasma, geradeso, wie nicht so selten ein der inneren Nase angehöriger Tumor seinen Ursprung ausserhalb dieses Gebietes genommen hat, wie bei vielen Oberkiefertumoren. Noch schwerer ist die Bestimmung der Zugehörigkeit bei den Nasenrachenraum-Tumoren, da an der Bildung der Wand des Nasenrachenraums eine ganze Anzahl von Gesichtsknochen und die Halswirbelsäule beteiligt ist. Ferner wird der von der Schleimhaut resp. vom Innern der Nase und ihrer Nebenhöhlen ausgegangene Tumor von den Chirurgen noch immer zu wenig beachtet und mehr auf die Erkrankungen der einzelnen die Nase und Nebenhöhlenwandungen bildenden Gesichtsknochen Rücksicht genommen. Es werden daher in allen chirurgischen Hand- und Lehrbüchern und Sammelwerken die

Krankheiten der Kiefer separat besprochen, womöglich mit dem Unterkiefer zusammen und dabei sehr häufig keine Unterscheidung gemacht zwischen dem Tumor, der von der Schleimhautauskleidung der Kieferhöhle, und dem, der vom Knochen ausgegangen ist, dem primären und dem sekundären Tumor, ein Uebelstand, auf den schon Bellotti in seinem Referat auf dem XI. Italienischen Otolaryngologischen Kongress mit den Worten aufmerksam gemacht hat: 'Die Nebenhöhlen-Tumoren sind immer ein wenig stiefmütterlich behandelt worden. Man kannte nur den klassischen Oberkieferkrebs und die klassische Oberkieferresektion von Nélaton, kümmerte sich aber wenig um die übrigen Tumoren der Nebenhöhlen. Die Rhinologie, die Spezialwissenschaft, erscheint berufen, diesen Teil der Chirurgie mehr aufzuklären und vor allem die triste Prognose zu verbessern.'

Zu diesen Schwierigkeiten der Präzision des Begriffes maligner Nasentumor kommt nun noch die geradezu trostlose Verwirrung und Ungenauigkeit der Literaturangaben, welche schon Citelli auf dem oben erwähnten Kongress bitter beklagt. Man begnügt sich vielfach mit der Diagnose: maligner Tumor, verwechselt Sarkom mit Karzinom, Epitheliom mit Endotheliom und speziell das Endotheliom, jenes Schmerzenskind der pathologischen Histologie, werden häufig mit Karzinom oder Sarkom zusammengeworfen. Diese Ungenauigkeit beruht zum Teil darauf, dass ja leider die pathologische Anatomie selbst sich über die Nomenklatur und Einteilung, ja über die Histogenese mancher Geschwülste noch nicht im Klaren ist, worauf ich noch später zurückkommen werde, besonders wieder, was die Endotheliome anbelangt, und es ereignet sich nicht zu selten, dass man von verschiedenen pathologischen Anatomen über ein und dasselbe Präparat völlig differente Befunde bekommt. Ja nicht einmal der Begriff Malignität ist sichergestellt. Borst, einer der grössten Kapazitäten auf dem Gebiete der Geschwulstlehre, sagt selbst, dass junges undifferenziertes Granulationsgewebe oft nicht von einem Sarkom zu unterscheiden ist. Die Grenzen zwischen zellreichem Fibrom und fibrösen Sarkom sind oft verzweifelt undeutlich. Wenn wir unter dem Begriff, 'maligner Tumor' Neubildungen von den bekannten histologischen Formen subsummieren, die mehr oder weniger rasch wachsen, schrankenlos mit Zerstörung der Umgebung wuchern, und Metastasen setzen, so sind wir um keinen Schritt weiter gekommen. Wir kennen echte maligne Geschwülste, welche ungemein langsam wachsen, welche oft während des ganzen Wachstums die Umgebung nicht substituieren, sondern höchstens verdrängen oder usurieren und welche enorme Ausbreitung gewinnen können, ohne Metastasen zu setzen. Hinwiederum kennen wir histologisch exquisit benigne Tumoren, welche rasch wachsen und schrankenlos die Umgebung zerstören, ja sogar Metastasen setzen. Speziell was den Verlauf anbelangt, so erlebt man da oft die seltsamsten Ueberraschungen, worüber später noch einmal die Rede sein wird. Es ist also den betreffenden Beobachtern nicht zu sehr zu verübeln, wenn sie oft oberflächliche

oder ungenaue Angaben machen. Besonders bei einer Gruppe von benignen Tumoren, den Angiofibromen oder Nasenrachenfibromen wird viel gesündigt, worauf schon Mikulicz in seinem Artikel über die Rachenneubildungen in *Heymanns Handbuch* aufmerksam gemacht hat, und nach ihm mehrere andere. Viele von diesen Tumoren segeln unter falscher Flagge (Angiosarkom, Fibrosarkom, Fibroangiosarkom, etc.), so dass ich bei allen derartigen Angaben von malignen Tumoren des Nasenrachenraums, welche jugendlich männliche Individuen betreffen, zur Vorsicht raten möchte. Ich selbst habe mich in einer Anzahl von Fällen genötigt gesehen, Beobachtungen von Fibrosarkom, etc., aus diesem Grunde in meiner Statistik unberücksichtigt zu lassen.

Endlich haben wir Kliniker nicht zuletzt mit dem Begriff der sogenannten *klinischen Malignität* zu rechnen, die für uns oft viel mehr bedeutet, als die pathologisch-anatomische. So stehen gerade die Nasenrachenfibrome in dieser Hinsicht in der Mitte zwischen echten benignen und echten malignen Geschwülsten, da sie bis auf die Metastasen alle Symptome eines malignen Tumors machen können und oft genug dem Patienten mehr Gefahr bringen, als mancher echte maligne Tumor. Wir wissen auch, dass das histologische Bild dieser Tumoren ein sehr wechselndes sein kann, manchmal von wirklichem Sarkom kaum mehr zu unterscheiden. Es zeugt für die Auffassung des Komitees von der klinischen Malignität, dass es, um das Thema einigermassen abzugrenzen, die Nasenrachenfibrome ausdrücklich ausgeschaltet wissen wollte.

Wenn ich also im Folgenden um das Thema sogleich genau zu präzisieren, die Sarkome, Karzinome und Endotheliome der Nase und des Nasenrachenraums zu besprechen habe, u. zw. deren Pathologie einschliesslich der Diagnose, während die Prognose und Therapie mein geehrter Herr Korreferent, Prof. Ferreri, zu übernehmen die Freundlichkeit hatte, so werde ich die Tumoren des Nasenrachenraums im zweiten Teil gesondert behandeln. Nicht nur, dass sie weniger Beziehungen zur eigentlichen Nasenhöhle haben — werden sie doch in allen Lehrbüchern mit den Tumoren des Rachens zusammen behandelt — so ist auch ihre Symptomatologie eine deutlich differente von denen der Nase und der Nebenhöhlen, vor allem wegen der Nachbarschaft des Gehörorgans und der Hirnnerven. Dagegen erscheint es mir nicht opportun, auf die reinliche Scheidung zwischen Nasen- und Nebenhöhlentumoren ein zu grosses Gewicht zu legen. Denn einerseits sind die meisten Nasentumoren zur Zeit ihrer Beobachtung schon Tumoren der Nase und der Nebenhöhlen ; andererseits ist in vielen Fällen der Ausgangspunkt überhaupt nicht mehr nachweisbar und dann auch für die Diagnose und Therapie belanglos. Ich folge damit dem Vorgehen von Harmer und Glas in ihrer klinischen Studie aus dem Jahre 1907, die ihre Arbeit ' Die malignen Tumoren der inneren Nase ' betiteln. Nur die einwandfrei nachgewiesenen primären Nebenhöhlen-Geschwülste seltenerer Art, namentlich die der Stirnhöhle und der Keilbeinhöhle, werde ich gesondert behandeln.

I. Die malignen Tumoren der Nase und ihrer Nebenhöhlen

Gehen wir von dem Artikel Kümmels in *Heymanns Handbuch* aus, der erst 69 Sarkome und 40 Karzinome der inneren Nase aus der Literatur sammeln und demnach von einer auffallenden Seltenheit der malignen Nasentumoren sprechen konnte, so hat sich das Verhältnis in den 15 Jahren bis heute wohl schon bedeutend geändert. Als Sammel- und Uebersichtswerke dienen uns die Arbeiten von Finder, Killian, Harmer und Glas, Maljutin, Gordiat, Ruaud, Safranek, Sendziak, sowie die Lehrbücher von Chiari, Zarniko, Denker-Brünings, etc., und die Lehr- und Handbücher der Chirurgie. Ueber das Sarkom der Nase erschienen grössere Sammelwerke von Watson, Grimm, Sonnenschein, über das Karzinom der Nase von Donogany und Lenart, Gibb und Katz. Ueber die Erkrankungen des Oberkiefers stehen uns Artikel von Hammer, Martens, Perthes und Sébileau zur Verfügung. Unter den Einzelbeobachtungen stellen die Engländer ein grosses Kontingent. Gemäss der Teilung unseres Referats erscheinen hier natürlich nicht die Sammelarbeiten, die sich vornehmlich mit Behandlungsverfahren und Operationsmethoden befassen.

Vorbehaltlich der schon erwähnten Ungenauigkeit und Verwirrung der Literaturangaben, die eine genaue Statistik von vornherein illusorisch machen, habe ich bis jetzt ca. 800 Beobachtungen von malignen Tumoren der Nase und ihrer Nebenhöhlen feststellen können. Darunter finden sich die schon erwähnten 109 Fälle von Kümmel, 79 von Maljutin aus verschiedenen Moskauer Spitälern gesammelt, darunter 62 Sarkome und 17 Karzinome; Gibb sammelte im Jahre 1902, 78 Nasenkarzinome aus der Literatur; Lenart und Donogany (1904) 80 Nasenkarzinome aus der Literatur und 7 eigene Fälle; Harmer und Glas berichten über 32 eigene Beobachtungen, i. e. 25 Karzinome, 6 Sarkome und 1 Zylindrom (Endotheliom). Es folgen Katz (1911) mit 8 eigenen Fällen von Nasenkarzinomen. Von Sarkomen der Nase konnten Watson 150 Fälle aus der Literatur zusammenstellen, Sonnenschein 142, Grimm 168, doch scheint letzterer manche Nasenrachenfibrome mitgezählt zu haben. An *Oberkiefertumoren* sammelte Hammer 22 Fälle. Aus Perthes' Artikel, 'Ueber die Verletzungen und Krankheiten der Kiefer,' im *Handbuch der pr. Chir.* erfahren wir, dass C. O. Weber 84 Sarkome und 125 Karzinome, Gurlt 69, Küster 15, Batzaroff (aus der Züricher Klinik) 33, Sébileau 30, und Martens 85 Tumoren des Oberkiefers zusammengestellt hat. Perthes selbst verzeichnet im ganzen 330 Beobachtungen von Karzinom und 225 von Sarkom des Oberkiefers. Endlich erschien im März-Aprilheft der *Archives internationales de Lar.*, etc., dieses Jahres von dem durch seine erschöpfende Statistik des Larynx-Karzinoms berühmt gewordenen Sendziak der Anfang einer eben so gross angelegten Monographie über die malignen Tumoren der oberen Luftwege, u. zw. vorderhand die Abhandlung über die Nebenhöhlentumoren. Danach ist es dem wahrhaft bewunderungswürdigen Eifer und Spürsinn Sendziaks gelungen, von den

ältesten Zeiten bis 1913, 643 maligne Nasentumoren, darunter 351 Sarkome und 224 Karzinome zu sammeln und 224 Nebenhöhlentumoren mit 7 eigenen Fällen, darunter 99 Sarkome und 113 Karzinome (12 Fälle ohne genaue Bezeichnung), eine Statistik, wie man sieht, die die meine noch bei weitem übertrifft. Sie wird ergänzt in Fortsetzung der aus der Klinik Chiari hervorgegangenen Arbeit von Harmer und Glas durch die seither weiter beobachteten Fälle, welche ich hiermit in der Lage bin, mit 44 (Nase und Nebenhöhlen) anzugeben, darunter 30 Karzinome, 12 Sarkome und 2 Endotheliome. Ueber die Endotheliome der Nase und der Nebenhöhlen existieren ausser einer grossen Zahl von Einzelbeobachtungen vornehmlich 3 Uebersichtsarbeiten, und zwar von Althoff und Broeckaert aus dem Jahre 1907 und von Pollak aus dem Jahre 1912. Letzterer weiss bereits über 54 Fälle zu berichten. Wenn man bedenkt, dass noch zu Anfang dieses Jahrhunderts nur einige wenige Fälle bekannt waren und bekanntlich ja vielfach diese Tumoren zu den Sarkomen oder Karzinomen gerechnet werden, eine gewiss schon recht stattliche Zahl. Nach meinen Nachforschungen ergänzt sich die Zahl bereits auf ca. 60. Wir sehen also, dass die malignen Nasentumoren nur mehr relativ selten sind.

Da nun bei den statistischen und anderen vergleichenden Angaben die einzelnen Tumorformen immer wieder getrennt erscheinen, wollen wir gleich hier in die *Histologie* der Geschwülste eingehen. Ich verweise zunächst auf die Lehr- und Handbücher der pathologischen Anatomie, Ribbert, Ziegler, Kaufmann, Aschoff, Borst u. a., und möchte nur kurz erwähnen, dass wir bekanntlich über die eigentliche Ursache der Geschwulstbildung noch immer im Unklaren sind, speziell über die der uns so sehr interessierenden bösartigen Geschwülste. Als bekannt kann ich auch voraussetzen, dass man in neuerer Zeit einer Art Kompromisstheorie huldigt, die die Ursache der Entstehung der Geschwülste vor allem in die Zellen selbst verlegt (Beneneke, Hertwig, v. Dungern, Werner, E. Albrecht), nebenbei eine gewisse Disposition zur Geschwulstbildung im Organismus überhaupt annimmt, dagegen der Annahme einer angebornen Grundlage (Cohnheim) nur bedingte Bedeutung, jedenfalls nicht gleichmässig für alle Geschwülste, beimisst, und den sog. ätiologischen Faktor, wie Trauma, chronische Reizung nur als veranlassendes Moment der prädisponierten Geschwulstbildung gelten lässt. Diese erfolgt auf Grund eines Wachstumsexzesses, der bei den malignen Geschwülsten die höchsten Grade erreicht. Der enge Zusammenhang zwischen gut- und bösartigen Blastomen zeigt sich auch darin, dass Uebergänge vorkommen. Nach Borst (in seiner *Zelltheorie des Karzinoms*), beruht Krebs- und Geschwulstbildung überhaupt auf einer primären zellulären Entartung. Albrecht nimmt an, dass in den malignen Geschwülsten embryonale Eigenschaften erhalten geblieben seien, was er sich durch eine abnorme Bildung organogener Stoffe ermöglicht denkt. Borst nimmt diese Hypothese insoweit an, als er zugibt, dass die Anlage zu solcher chemischer Entartung angeboren sein muss, jedenfalls bleibt die Annahme einer

tiefgreifenden Aenderung des Zellcharakters bestehen. Arzt und Kerl konnten in einer Publikation über die Ergebnisse der Freund-Kamierschen Karzinomreaktion (1912) mit Hilfe der zellzerstörenden Wirkung des Serums oder des Mangels einer solchen experimentelle Beweise für die Karzinomdisposition im pathologischen Gewebe erbringen, welcher *lokalen* Umstimmung späterhin erst eine solche im Blut folge. Auch Hochenegg ist des öfteren schon für die *Disposition* bei der Entstehung des Karzinoms als ursächlichen Faktor eingetreten. Auch die bekannte und zweifellos festgestellte Familienerblichkeit (Batzaroff, Winiwarter, Guillaumes, Bergmann, Trendelenburg) ist eine weitere Stütze für die Lehre von der Disposition. Von der Theorie eines bakteriellen oder parasitischen Erregers (Gussenbauer, J. J. Clarke, Doyen) scheint man sich mehr und mehr zu entfernen, zumal, da künstliche Züchtung derartiger Parasiten immer nur entzündliche Neubildungen, aber nie echte Geschwülste erzeugen konnte. Diese sind experimentell nur immer wieder aus echten Geschwulstelementen zu gewinnen. An spezifischen Erregern, die für die Entstehung maligner Geschwülste verantwortlich gemacht worden sind, ist kein Mangel. Erst 1911 hat S. Löwenstein die *Trichodes crassicanda specifica* als eine causa directa in der Aetiologie der Tumoren gefunden. Dieser Wurm, den der Autor im Urogenitaltrakt von Ratten gefunden hat, regt die Nieren-, Ureter- und Blasenepithelien direkt durch Einwanderung der Eier und Jungformen in das Epithel zur atypischen Zellteilung und Wucherung an. Es entstehen Papillome, die manchmal malignen Charakter zeigen, während bei Bilharzosis durch den Parasiten erst Cystitis und chronische Entzündung erzeugt wird. Borst sagt: 'Es mag sein, dass für einzelne Neubildungen, die wir heute noch unter die echten Geschwülste rechnen, z. B. für manche "Sarkome" einmal eine parasitäre Noxe gefunden wird; für die Gesamtheit der Geschwülste aber ist ein parasitärer Ursprung im höchsten Grade unwahrscheinlich.' Was die sogenannte *Alterstheorie* (Antagonismus) anlangt, so lehren Werners Transplantationsversuche mit Sicherheit, dass die Art des Weiterwachsens der Gewebe von der Beschaffenheit seiner Zellen abhängt und nicht vom Antagonismus der Umgebung. Ferner zeigt die klinische Erfahrung, dass jugendliches Gewebe weniger zur Tumorbildung disponiert ist, als gealtertes, wofür Werners Reizversuche die Erklärung gegeben haben.

Die Lehre von der ätiologischen Bedeutung des Traumas im weitesten Sinne hat besonders auch in dem hier verhandelten Gebiet Geltung. So existieren genug Beobachtungen von malignen Geschwulstbildungen nach einmaligem Trauma oder der einem oft wiederholten Trauma gleichzusetzenden chronischen Irritation. Was das erstere betrifft, so ist es am häufigsten als veranlassendes Moment bei Sarkomen gefunden worden, so hat D. Grant, 1908, ein Lymphosarkom der Stirnhöhle bei einer 25 jährigen Frau beobachtet, wo sich im Anschluss an ein Trauma der Tumor entwickelt hat; desgl. Johnson, 1911, ein alveoläres Spindelzellensarkom bei einer 33 jährigen Frau im Anschluss an ein Trauma

der Wange; Wishart, 1909, maligner Nasentumor, einige Jahre vorher Fall auf die Nase. Bei so versteckten Partien, wie es die Nase und ihre Nebenhöhlen sind, wäre natürlich auch denkbar, dass der Tumor zur Zeit des Traumas schon bestanden hatte, doch ist die Erfahrung von der Bedeutung des Traumas an zahlreichen oberflächlich gelegenen Tumoren einwandfrei erwiesen. Der Frage der chronischen Irritation, die namentlich für das Karzinom als ätiologisches Moment herangezogen wurde, sind auf unserem Gebiete schon Kümmel und Killian und später Harmer und Glas nähergetreten. Es handelt sich entweder um wiederholtes Trauma (häufige Polypen-Operationen) oder chemische Reizung (Chromarbeiter) oder um chronisch entzündliche Prozesse. Als solche werden genannt: Ozäna, chronische Eiterungen, Syphilis und Tuberkulose; endlich ist aus der allgemeinen Pathologie die Entwicklung von malignen Tumoren, besonders Karzinomen, in alten Geschwürsnarben bekannt.

Uns interessiert besonders die Frage der *Entstehung maligner Tumoren bei chronischen Empyemen*, da sie auch diagnostische Bedeutung beanspruchen. Harmer und Glas studierten die Frage eingehend und stellen drei Möglichkeiten des Zusammenhanges fest: (1) Zufällige Koexistenz, (2) der Tumor ist das primäre, (3) Die Eiterung ist das primäre. Fälle von malignen Tumoren, in denen langdauernde Eiterungen bestanden hatten, sind zahlreich beschrieben worden (Pepper, Dreyfuss, Day, Halstedt, Wolkowitsch, Harmer, Manasse, Labarre). Aber schon Harmer will in den Empyemen in den meisten Fällen nur ein prädisponierendes Moment zur malignen Tumorbildung erblicken und warnt davor, stets ein ' Post hoc, ergo propter hoc ' konstruieren zu wollen, da dann eine nicht geringe Zahl von Trugschlüssen zustande kommen kann. Schon mit Rücksicht auf das Missverhältnis der Zahl der malignen Tumoren zu den ungeheuer vielen chronischen Eiterungen ist anzunehmen, dass wohl noch andere Ursachen zur Tumorbildung vorhanden sein müssen, weshalb den Eiterungen im allgemeinen keine prinzipielle Bedeutung zum Zustandekommen der malignen Neubildungen zuzuschreiben ist.

Eine weitere, die Geschwulstlehre in hohem Masse interessierende Frage ist die der Umwandlung benigner Tumoren in maligne. In unserem Gebiet speziell die Umwandlung von Polypen, oder, wie sie heute pathologisch-anatomisch richtiger heissen, oedematösen Fibromen. Auch damit haben sich Kümmel und Harmer und Glas eingehend beschäftigt. Die letzteren Autoren stellen vier Möglichkeiten des gemeinsamen Vorkommens von Polypen und malignen Neubildungen zusammen: (1) zufälliges Zusammentreffen; (2) durch entzündlichen Reiz oder Stauung entstehen in der Umgebung des Neoplasmas benigne Polypen; (3) durch Abschnürung einzelner Tumoranteile können polypenähnliche Bildungen entstehen. Diese letztere Möglichkeit ziehen die Autoren zur vermutlichen Erklärung mancher einschlägiger Literaturangaben heran, von Fällen, welche angeblich längere Zeit an Polypen gelitten hatten und daran operiert worden waren. Endlich (4) direkte Umwandlung von Polypen in maligne Geschwulst. Nach genauem Studium der

einzelnen diesbezüglichen Beobachtungen kommen sie zum Schluss, dass eigentlich keiner der verzeichneten Fälle einer strengen Kritik standhält, nicht einmal der vielzitierte Fink'sche Fall, und betonen das *Fehlen von histologischen Uebergangsbildern.* Auch Borst vertritt die Ansicht, dass bei der sog. malignen Entartung einer gutartigen Geschwulst nicht die fertig differenzierten Teile eines Blastoms sich in krebsiger oder sarkomatöser Weise umwandeln, sondern die bösartige Wucherung geht von den weniger differenzierten Proliferationszonen aus; von hier werde im Falle der malignen Entartung destruierend wucherndes Gewebe gebildet. Wahrscheinlich unter dem Zwange gewisser lokaler und allgemeiner Wachstumshemmungen entwickelt sich eine Geschwulst, *die von vornherein die Qualität der Bösartigkeit latent in sich trug* und mit dem Wegfall der Hemmungen destruierend zu wachsen beginnt.

Die Begriffe Sarkom und Karzinom als bekannt voraussetzend, werde ich mich nur bezgl. der Endotheliome mehr verbreitern. Sie sind gegenüber den typischen Sarkomen oder Karzinomen so selten, dass sie bei den Zahlenverhältnissen nicht in Betracht kommen. Bezgl. der Häufigkeit der Sarkome und Karzinome gehen die Ansichten der einzelnen Autoren in den einzelnen Zeitepochen auseinander. Kümmel fand im *Heymann'schen Handbuch* die Sarkome der inneren Nase überwiegend, stellt hingegen in seinem Artikel im *Handbuch der pr. Chir.* die Karzinome wegen ihrer 'relativen Häufigkeit' an erste Stelle. Maljutin fand in seiner Zusammenstellung die Sarkome weitaus überwiegend. Zu demselben Ergebnis kommen Harmer und Glas (in der Literaturübersicht), Sonnenschein, Katz und endlich Sendziak. Dieser letztere fand Vorherrschen der Sarkome der eigentlichen Nase gegenüber dem Karzinom (351 : 224), bei den Nebenhöhlen dagegen Vorherrschen der Karzinome (112 : 99). Dieses Verhältnis verschiebt sich etwas bei der Statistik der einzelnen Nebenhöhlen. Die Sammlungen über die Kiefertumoren ergeben nach Perthes Ueberwiegen der Karzinome im Verhältnis von 3 : 2. Dasselbe fanden Hammer, Martens, Sébilau. Mit diesen Resultaten stehen die Statistiken von solchen Autoren, welche an dem einheitlichen Material einer Klinik genaue histologische Untersuchungen der einzelnen Fälle anführen, im merkwürdigen Gegensatz. So konnten Harmer und Glas gegenüber 25 Karzinomen nur 6 Sarkome verzeichnen. Auch die von mir vorhin angeführte Statistik der Fälle der Wiener Klinik nach 1907 ergibt ein auffallendes Prävalieren der Karzinome. Schon dieser Umstand gibt zu denken, und ich kann nur meine schon einmal ausgesprochene Mahnung zur Vorsicht bei den meisten Literaturangaben wiederholen.

Bezüglich des *Alters* der malignen Nasentumoren sind uns, wenn wir die Erfahrungen über das Alter der malignen Tumoren an anderen Körperstellen vergleichen, die relativ häufigen Beobachtungen von Sarkomen bei jugendlichen Individuen nichts Verwunderliches. Ja, das jugendliche Alter ist sogar mit einer gewissen Vorsicht für die Differentialdiagnose verwertbar. Citelli (1908) gibt für die Endotheliome und

Sarkome das Alter als gewöhnlich unter 40 Jahren, für die Karzinome über 40 Jahren stehend an. Perthes (Oberkiefertumoren) fand: das Sarkom wird in jedem Lebensalter beobachtet, am meisten im 3. und 4. Dezennium; verhältnismässig stark ist die Beteiligung des jugendlichen Alters. Watson gibt das 5. Dezennium als Prädilektionsalter für das Nasensarkom an, Grimm das 6. Dezennium, H. Klein und Sonnenschein finden merkwürdigerweise zwischen 10 und 20 Jahren die meisten Angaben. Darin stimmen aber alle Statistiken so ziemlich überein, dass Karzinome des jugendlichen Alters eine grosse Seltenheit sind. Von Sarkomen existieren Beobachtungen von 3 jährigen Patienten (Ross, Myxosarkom der Nase; Finsterer, Chlorom oder myelogenes Sarkom des Oberkiefers; Paludetti, Sarkom der Nase), 5 jährigen (Scholtz, Osteosarkom des rechten Siebbeins), 10 jährigen (Finder, Riesenzellensarkom der Kiefer; Fitzwilliams, Myelom des Oberkiefers), 13 jährigen (Walker Downie), 16 jährigen (Tilley, Chondrosarkom der Nase; Uffreduzzi, Endotheliom der Nase), über Karzinome von Willis und Tawse (16 jähriger Bursch, raschwachsendes Karzinom der Nase). Watson fand vier Angaben, Sonnenschein 6 Angaben von Sarkom der Nase unter 10 Jahren.

Was das *Geschlecht* der Kranken anbelangt, so finden wir übereinstimmend Ueberwiegen der Männer, wie überhaupt bei den malignen Tumoren der oberen Luftwege.

Wenden wir uns nun der Besprechung der einzelnen *Geschwulstformen* zu, so finden wir ziemlich allgemein die Angabe, dass bei den Sarkomen die Spindelzellen- und Rundzellensarkome weitaus die häufigsten sind, von diesen wieder die kleinzelligen Formen häufiger als die grosszelligen; nach Kümmel, Sonnenschein und Harmer die spindelzelligen häufiger als die rundzelligen, nach Watson und Gerber umgekehrt. Im übrigen sind alle Formen von Sarkomen beobachtet worden, klein- und grosszellige Spindel- und Rundzellensarkome, Fibrosarkome. Riesenzellensarkome, eine relativ häufige und bekannte Erscheinung am Processus alveolaris, die als sekundäre Geschwulst auch mit den Nebenhöhlen und der inneren Nase in Beziehung treten können, sind in der inneren Nase sehr selten. Kümmel kennt 2 Fälle (Watson und Nélaton). Von neueren Beobachtungen wären 1 Fall von C. J. Kœnig (Paris) zu erwähnen, in welchem es sich um einen 60 jährigen Mann mit einem blutroten Tumor nahe der rechten Choane handelt, dessen endonasale Abtragung zur Heilung führte, und ein Fall der Wiener Klinik, vom Referenten beobachtet; schliesslich einer von Finder, in dem bei einem 10 jährigen Knaben wegen eines alle Wände der Kieferhöhle auftreibenden Riesenzellensarkoms Oberkieferresektion gemacht werden musste. Ueber Alveolärsarkome publizierten Knight, über solche mit Spindelzellen, Johnson, Ssamoylenko. Von den höher organisierten Sarkomen, welche Beziehungen zu den fertigen mütterlichen Bindesubstanzgeweben noch nicht ganz vermissen lassen, wurden beobachtet: Myxosarkome (Morelli, Ross, Schmiegelow); Myxofibrosarkome (Dougherty, Hajek und Polyák),

letzterer Fall ein mächtiger Tumor, der von der Keilbeinunterfläche ausgegangen war und die ganze Schädelbasis zerstört hatte; Chondrosarkome (Tilley); Osteosarkome (Freudenthal, Rydygier, Scholtz), letzterer bei einem 5 jährigen Knaben; Osteoid-Sarkome (Watson-Williams); Cystosarkome (Heyfelder, Petroff); ferner die den Endotheliomen sehr nahestehenden Angiosarkome (Robertson, Sendziak, Zenker u. a.), diese Tumoren können oft so gefässreich sein, dass sie pulsieren; malignes Fibroadenom (Lehm). Die Melanosarkome sind relativ selten und auch entgegen dem Verhalten in anderen Körperregionen gutartig. Nach Perthes sind bisher 17 Melanosarkome des Oberkiefers beobachtet. Weiter von Nicolai, Schurmann, Wilkinson, Kassapian (sekundär von der Chorioidea aus in den Tränensack und in die Nase gewandert). Einen wertvollen Beitrag zur Geschwulstlehre bildet der vom Referenten beobachtete Fall von kleinzelligem Spindelzellensarkom mit Drüsenmetastasen, bei einer 66 jährigen Frau, die vor 5 Monaten wegen Carcinoma simplex mammae derselben Seite mit Ausräumung der Axillardrüsen operiert worden war. Ein weiterer interessanter Fall ist von Manasse publiziert: Spindelzellensarkom der mittleren Muschel, die Wand einer Mukokele durchsetzend und Cholesteatom-Massen durchsetzt. Man sieht nach der Beschreibung des Referats von der Oberfläche Schläuche in das Sarkom hineingehen, das Flimmerepithel der Oberfläche ist metaplasiert und gleichzeitig gewuchert, u. zw. anscheinend stärker als das Sarkom. Daher die Gutartigkeit und das langsame Wachstum der Geschwulst. Eine ungeheure Rarität ist das Rhabdomyo-Sarkom der Nase. Kümmel nennt es ein Unikum, da er damals nur den Fall von Richardson kannte, mittlerweile hat Vail (Cincinnati) im *Laryngoscope*, 1908, einen zweiten Fall publiziert. Der Tumor gehörte der lateralen Nasenwand an, rezidivierte im Anschluss an diverse endonasale Eingriffe mehrmals und die Patientin erlag schliesslich der Radikaloperation. Vail kann sich die Entstehung nur durch Keimversprengung auf einem der quergestreiften Muskeln der äusseren Nase denken. Die Malignität hängt von dem sarkomatösen Anteil ab. Perthes erwähnt auch die hin und wieder beobachtete Aenderung der histologischen Struktur, so von Chondrosarkom in Rundzellensarkom u. dgl. Walker Downie berichtet von einem beide Siebbeine erfüllenden blassen, leichtblutenden, schmerzhaften Tumor, der in mehreren Sitzungen endonasal entfernt worden war. Die histologische Untersuchung der einzelnen Tumorstücke zeigte die Umwandlung von reinem Myxom in Myxosarkom und wieder zurück in einfaches Myxom. Bei Rezidiven oder im weiteren Verlauf, wenn der Tumor rascher wächst, kann man oft auffallende Vermehrung der Zellen gegenüber dem Zwischengewebe nachweisen. In dem Fall Sonnenscheins handelte es sich um ein 11 Jahre dauerndes Sarcoma nasi, welches bei der ersten Operation kernreiches Bindegewebe gezeigt hatte, nach einer zweiten Operation 7 Jahre verschwunden war, um dann wiederzukommen. Die entfernten Tumormassen boten jetzt ein an Karzinom erinnerndes Bild. Nach 4 weiteren Jahren hatte der Tumor

die ganze Nase ergriffen und bestand jetzt aus kleinzelligem Rundzellensarkom. Das Lymphosarkom, im Nasenrachenraum relativ häufig, ist in der inneren Nase und in den Nebenhöhlen eine seltene Erscheinung, meist ist es vom Nasenrachenraum gegen die Nase und die Nebenhöhlen zu vorgedrungen (Kümmel). M. Schmidt will 5 derartige Fälle gesehen haben. Die Unterscheidung zwischen kleinzelligem Rundzellensarkom und Lymphosarkom scheint aber nicht so leicht zu sein. Ich fand eine Publikation von D. Grant, *Lympho-Sarkom der Stirnhöhlen bei einer 25jährigen Frau*, und von Menzel, welcher eine sehr seltene Form des Lympho-Sarkoms beschreibt. Der Tumor hatte in der Nase oder im Nasenrachenraum begonnen und war eigentümlich infiltrierend, wie eine Leukämie in die vordere Nase und nach abwärts in den Rachen, Kehlkopf, bis in die Bronchien vorgedrungen. Bei der Sektion fand man die Rachenhinterwand vom Kehlkopf bis zum Rachendach mächtig infiltriert, ferner setzte sich die Infiltration in die Trachea und Bronchien fort, welche sich über und über mit hirsekorngrossen Knötchen besetzt zeigte. Es handelte sich um die überaus seltene Form von diffusem, fibrösem Lymphosarkom sämtlicher Teile der oberen Luftwege. Der Tumor zeigte sich stellenweise besonders reich an zellarmem, fibrösem oder direkt als Hyalin zu bezeichnendem Bindegewebe. Einen sehr merkwürdigen Tumor des Oberkiefers hat Finsterer im Jahre 1911 operiert, bei einem 3jährigen Knaben. Der Tumor zeigte nach der Herausnahme grasgrüne Verfärbung der Schnittfläche ; es handelte sich entweder um Chlorom, wofür allerdings ein strikter Beweis nicht zu erbringen war, oder um myelogenes Sarkom, mit Grünfärbung ; Finsterer studiert die Entstehungsarten dieser Grünfärbung, man hat verschiedene Erklärungen dafür versucht, die aber sämtlich nicht voll befriedigen konnten (Pigmentkörnchen u. a.). Die Grünfärbung verändert sich bei längerem Verweilen an der Luft ins schmutzig Graugrüne.

Die Sarkome können entweder von dem Bindegewebe der Schleimhaut, von dem periostalen Ueberzug der Nase und der Nebenhöhlen, vom Periost der Aussenseite der Gesichtsknochen, oder, wie bei vielen Oberkiefertumoren, vom Innern des Knochens (myelogene Sarkome) ihren Ursprung nehmen. Ein sicher festgestellter Fall von originärem Schleimhautsarkom scheint nur Duplays von Kümmel erwähnter Fall zu sein, zumeist kommen die anderen Entstehungsmöglichkeiten vor. Die Lokalisation der Sarkome ist vorzugsweise am Septum, u. zw. im vorderen Anteil und an der lateralen Nasenwand am vorderen Ende der mittleren Muschel. Selten sind die Nebenhöhlen der Ausgangspunkt. Die Sarkome der Nebenhöhlen sind zum grossen Teil sekundär in sie eingewucherte Tumoren (Killian). Charakteristisch für die Sarkome ist die Neigung zur Tumorbildung und die geringe Tendenz zu Metastasen, so dass speziell die Oberkiefersarkome ganz enorme Dimensionen annehmen können, ohne dass allgemeine Sarkomatose eintreten müsste. Doch zeigt meine vorhin erwähnte Beobachtung (grosser Drüsentumor bei Spindelzellensarkom in wenigen Monaten gewachsen), dass dies nicht

immer zutrifft. Eine weitere Eigentümlichkeit, die ihnen gegenüber den Karzinomen auch mehr Gutartigkeit verleiht, ist die verhältnismässig längere Dauer. Nach Perthes und Martens dauern die Oberkiefersarkome von einem Monat bis 4 Jahren, durchschnittlich 14 Monate. Birnbaum berechnet für die Rundzellensarkome 3 Monate, für die periostalen Riesenzellensarkome 6 Monate, für die Spindelzellensarkome 14 Monate, und für die zentralen Riesenzellensarkome $4\frac{1}{2}$ Jahre. Nach Sonnenscheins und meiner Zusammenstellung dauerte ein Fall von Chiari und Marschik 6 Jahre, desgleichen ein Fall von I. Braun, je 1 Fall von Huetlin und Gerber 7 Jahre, 1 Fall von Watson $7\frac{1}{2}$ Jahre, einer von Michael 10 Jahre, Sonnenscheins Fall 11 Jahre und ein Fall von Higgens angeblich gar 14 Jahre, doch sind die Angaben dieses letzteren etwas zweifelhaft. Im Falle Sonnenschein ' vermochte eine bösartige Geschwulst, durch entsprechende Eingriffe in Schranken gehalten (eine Radikaloperation war abgelehnt worden), 11 Jahre hindurch weder dem befallenen Organ, noch dem Gesamtorganismus etwas besonderes anzuhaben, und doch handelte es sich um die bösartigste Form (kleinzelliges Rundzellen-Sarkom).'

Ich fahre in der Besprechung der Histologie fort, um Symptomatologie und Diagnose aller Tumoren später vereint zu besprechen. Die *Karzinome* der Nase und Nebenhöhlen, in der Nase seltener, in den Nebenhöhlen häufiger, sind die wichtigsten malignen Tumoren, besonders wegen ihrer Bösartigkeit. Sie zeichnen sich gegenüber den Sarkomen durch mehr destruktives, weniger tumorbildendes Wachstum aus, machen mehr Defekte, weniger Verdrängungserscheinungen, verlaufen rascher und unterscheiden sich noch besonders durch die Neigung zu oberflächlichem Zerfall, weshalb, wie noch später auszuführen sein wird, die Jauchung in dem Vordergrund des Krankheitsbildes tritt. Während nach Borst für die Sarkome einstweilen eine Histogenese nicht herzustellen war, unterscheidet dieser Autor bei den Karzinomen eine formale und kausale Genese.

Ad 1. (Ribbert) Postembryonale Lösung organischer Verbindungen durch Bindegewebswucherungen.

Entweder durch Trauma und chronische Entzündung oder als Krebsbildung an Stelle komplizierter Entwicklungsvorgänge. Hervorgehen von Krebs aus Gewebsmissbildungen oder sicheren embryonalen Gewebsverlagerungen mit angeborener oder vererbter Disposition zur Krebsentwicklung.

Ad 2. Epithelausschaltungen, Aberrationen bei der embryonalen Entwicklung.

Die Karzinome gehen entweder von dem Deckepithel der Schleimhaut oder von den Drüsen aus. Ihr Lieblingssitz ist nach Kümmel ungefähr in gleichem Verhältnis der vordere Anteil des Septum und der unteren und mittleren Muschel. Nach Donogany und Lenart stellt sich der Prozentsatz wie folgt: Septum 14 %, untere Muschel 21 %, mittlere Muschel 50 %. In den Nebenhöhlen kommen sie besonders häufig

in der Kieferhöhle zur Entwicklung, dies wahrscheinlich zum Teil infolge der verschiedenen sekundären Tumoren, die oft mitgerechnet werden, da sich der Ausgangspunkt nicht mehr bestimmen lässt. Um weiterer Konfusion vorzubeugen, will ich hier nur die Zahlen der Sendziak'schen Statistik anführen. Er berechnet das Verhältnis von Sarkomen zu Karzinomen in den einzelnen Nebenhöhlen wie folgt: Stirnhöhle 13 zu 12, Kieferhöhle 49 : 84, Siebbein 22 : 16, Keilbeinhöhle 15 : 12. Was diese Zahlen, namentlich für die Kieferhöhle und das Siebbein anbelangt, würde ich kein zu grosses Gewicht darauf legen. Sehr häufig sind die Tumoren primär in der Nasenhöhle entstanden und derart in die Nebenhöhlen eingewuchert, dass sie dann klinisch ihnen anzugehören scheinen (Killian). Citelli leugnet überhaupt für die meisten Kieferhöhlen-Karzinome ihre primäre Entstehung. Killian hat im *Heymannschen Handbuch* auf die Mangelhaftigkeit diesbezüglicher Angaben aufmerksam gemacht und auch Sendziak schliesst sich dieser Ansicht an.

Sehen wir uns nun die Formen an, in denen das Karzinom der Nase und der Nebenhöhlen am häufigsten erscheint, so finden wir wohl häufig genug Zylinderzellenkrebs oder Adenokarzinom, was ganz plausibel erscheint, aber noch weit häufiger *Plattenepithelkrebs*, und zwar nicht nur am Naseneingang oder sonst an Stellen, wo sich schon normaler Weise Plattenepithel vorfindet, sondern gerade dort, wo sonst typisches Zylinderepithel vorhanden ist, zum Beispiel die Gegend der mittleren Muscheln, ja in den Nebenhöhlen selbst. Ich verweise hier auf das, was ich schon bei den Sarkomen gesagt habe, dass nämlich meiner Meinung nach (wie auch von anderen Autoren schon ausgesprochen worden ist), das einheitliche Material eines und desselben Forschers aus einer Klinik oder Station hervorgegangen, mehr Wert hat als umfangreiche Sammlungen von Einzelbeobachtungen, insbesondere wenn dieses Material gleichmässig und sorgfältig ausgeführte pathologisch-anatomische und histologische Untersuchungen erfahren hat. So fanden Harmer und Glas 14 Plattenepithelkarzinome, 10 Zylinderzellenkarzinome und ein Medullarkarzinom. Aus der Statistik derselben Klinik nach 1907 kann ich über 24 Plattenepithelkarzinome und 6 Zylinderkarzinome berichten. Als Erklärung hierfür ist nun schon ziemlich frühzeitig die *Epithelmetaplasie* gefunden worden. Nachdem schon Kümmel 1898 darauf hingewiesen, aber für diese Theorie damals noch wenig Belege vorhanden waren, haben seither eine Reihe von Autoren das Vorhandensein von Plattenepithelien bei der Schleimhaut in der Nähe von malignen Tumoren als Grundlage für die gleichartigen Karzinome in der Nase und in den Nebenhöhlen nachgewiesen (Bartha und Onodi, Iwanoff, Schwenn, Manasse u. a.), ferner durch das Vorkommen von Cholesteatom in der Kieferhöhle. Harmer und Glas haben 4 Entstehungsarten der Plattenepithelkarzinome angegeben:

1. Das Karzinom nimmt seinen Ursprung vom Introitus nasi. Diese Fälle sind allerdings ziemlich selten.

2. Das Karzinom entwickelt sich aus Plattenepithel, welches durch

Metaplasie aus Zylinderepithelien hervorgegangen ist, und zwar in der Regel durch chronische Entzündung.

3. Aus den schon von Killian zur Erklärung der Kieferhöhlenplattenkarzinome herangezogenen paradentären Zahnkeimen (Malassez,) die schon von Bruns und Partsch zur Erklärung der Entstehung der Zahnwurzelcysten verwertet worden sind.

4. Die Entstehung aus embryonalen vorgebildeten primär entstandenen Inseln von Plattenepithelien (Cohnheim). Diese letztere Annahme wird schon von den genannten Autoren als die unwahrscheinlichste bezeichnet und das grösste Gewicht auf die unter 2. angeführte Erklärung gelegt. Hiezu kommt noch das Vorkommen von Plattenepithelkarzinomen der Kieferhöhle, sekundärer Tumoren, die von der Schleimhaut des Gaumens oder Zahnfleisches ausgegangen sind.

Die Metaplasie des Zylinder- in Plattenepithel ist früher mehrfach bei Ozaena nachgewiesen worden. Siebenmann und Schönemann ist das Hauptverdienst an der endgiltigen Erledigung dieses Problems zuzuschreiben. Die Disposition zur Metaplasie nimmt vom Naseneingang nach hinten zu proportional ab. Der häufigste Ort derselben ist das vordere Drittel des Septums. Hier spielt sich auch die von Siebenmann zuerst beschriebene Rhinitis sicca anterior ab, ferner das vordere Ende der beiden Muscheln, erfahrungsgemäss auch ein Lieblingssitz von Karzinomen. Siebenmanns Schüler Oppikofer fand 1907 an zweihundert untersuchten Nasen intranasales Plattenepithel als regelmässiges Vorkommnis, u. zw. untere und mittlere Muschel gleich häufig, vorn ist es häufiger und ausgedehnter als hinten. Die Inseln von Plattenepithel gehen immer allmählich in das Zylinderepithel über, selten sind scharfe Grenzen vorhanden. An der Hinterfläche der unteren Muschel fand Oppikofer Plattenepithel in 11 %, Verhornung fand er in 20 %. *Somit ist für alle Formen des Plattenepithelkarzinoms in der Nase die Entstehungsmöglichkeit geklärt.*

Oppikofer fand auch, dass ausgedehnte Metaplasie sich in ganz normalen Nasen finden könne. Im Jahre 1909 hat dann dieser Autor diesen überraschenden Befund der Nasenschleimhaut auch für die Nebenhöhlen bestätigen können. An 165 untersuchten Schleimhäuten fand er Epithelmetaplasie bei 41 % aller Kieferhöhlenfälle, 31 % aller Stirnhöhlen, 33 % aller Siebbeine, nur bei 3 Keilbeinhöhlen nicht. Er nimmt selbst an, dass das Plattenepithel wahrscheinlich noch häufiger ist als in dem obigen Verhältnis. Ausgedehnte Strecken sind selten, mehrere kleinere Inseln dagegen häufig. Für die Metaplasie bezeichnet er als häufigste Entstehungsursache chronische Eiterungen und Katarrhe. Bei 70 akuten Eiterungen fand sich nie Plattenepithel. Nachdem er schon an der ersten Serie von 200 Nasen die Häufigkeit der Nebenhöhlenentzündungen namentlich der Kieferhöhle pathologisch-anatomisch nachgewiesen hatte, kommt er auf Grund genauer mikroskopischer Untersuchungen an 165 Fällen der zweiten Serie zu dem Ergebnis, dass die Nebenhöhlenentzündung, namentlich auch der leichtere nicht eiterige

Katarrh, ungemein häufig sei, vielfach von selbst ausheile, als Residuum aber bleibt die Epithelmetaplasie. Auch für die Kieferhöhle will Oppikofer die Genese der Plattenkarzinome aus der durch chronische Entzündung hervorgerufenen Metaplasie erklärt wissen und meint, dass auch in der Stirnhöhle und Keilbeinhöhle die Plattenkarzinome überwiegen und nach seiner Untersuchung eine Metaplasie des Nebenhöhlenepithels wenigstens auf kurze Strecken durchaus nichts seltenes ist. So ist die bereits von Krug, Hansemann, Martens, Zarniko, Uffenorde u. a. ausgesprochene Annahme näherliegend, dass sich das Karzinom aus dem metaplasierten Epithel entwickelt hat. Später hat Goetjes 31 Keilbeinhöhlenschleimhäute von Lebenden untersucht, dazu noch 100 Leichenpräparate und gleichfalls die Häufigkeit des metaplasierten Epithels feststellen können. Auch er fand die Häufigkeit pathologischer Befunde bei den untersuchten Nebenhöhlen auffallend. Sie bestätige nicht die Auffassung, dass die in den Nebenhöhlen nachgewiesenen Veränderungen akute terminale Leiden darstellen. Die Theorie von den primären in den Nebenhöhlen ohne Nasenerkrankung entstehenden Entzündungen ist wahrscheinlich nicht richtig. An einer Stelle bleibt die Erkrankung bestehen, an einer anderen Stelle heilt sie aus. Endlich untersuchte Neumann auf Grund des Befundes zweier Plattenepithelkarzinome der Stirnhöhle der Wiener Klinik 12 Schleimhäute von Stirnhöhleneiterungen und 43 Schleimhäute von Leichen und konnte bei den Eiterungen in allen Fällen mehr minder ausgebreitete Metaplasie feststellen, bei den Leichenpräparaten nur in 8 Fällen keine Metaplasie finden. Das Plattenepithel fand er manchmal mehrschichtig, mit und ohne Verhornung, in einzelnen Fällen sogar niedere papillomartige Bildungen mit interpapillären Zapfen. Da nur bei wenigen von den untersuchten Leichenpräparaten mit Sicherheit Entzündung nachzuweisen war, so muss man annehmen, dass ganz geringe Reize genügen können, um bei genügend langem Bestande Metaplasie hervorzurufen. Die Entstehung der Kieferhöhlenkarzinome aus den Malassez'schen Keimen scheint jedenfalls sehr selten zu sein. 1908 hat Moure über zwei Fälle von malignen Kieferhöhlentumoren berichtet, die aus Wurzelcysten entstanden waren.

Einer interessanten Hypothese zur Lehre von der Entwicklung des endonasalen Karzinoms müssen wir noch Erwähnung tun, die von Katz in seiner schönen Studie über das endonasale Karzinom entwickelt wird. Er kommt nämlich auf Grund vergleichender Forschungen zu der Folgerung, dass alle Organe, die selten Sitz primärer Tumoren sind, sich durch grossen Reichtum an *adenoidem Gewebe* auszeichnen, so die Milz, der Dünndarm, die Lymphdrüsen, die Rachen-, Gaumen- und Zungenmandel. Die Zunge z. B. wird dort befallen, wo die wenigstens lymphfollikulären Elemente vorhanden sind, d. i. an der Seitenfläche, am Rücken, an der Spitze. Ebenso zeigt die Nasenschleimhaut eine sehr minimale Tendenz zur Entwicklung epithelialer Tumoren überhaupt, aber auch dort entwickelt sich das Karzinom an den Stellen geringsten adenoiden Gewebes (Septum, untere und mittlere Muschel, die anderen Stellen kommen

kaum in Betracht). Katz kommt zu dem Schluss, dass durch die konstante Funktion der adenoiden Schicht, durch das ununterbrochene Durchströmen der Leukozyten nach der Schleimhautoberfläche, durch das gewaltsame Durchbrechen derselben durch die sie bedeckende Epithelschicht und durch das damit verbundene ständige Vergehen und Entstehen neuer epithelialer Zellen, das atypische, vor allem ungebundene, exzessive, aus dem Organverbande losgerissene Wachstum epithelialer Elemente hintangehalten wird, weil der Mutterboden schon eine maximale Arbeitsleistung in der Ersetzung des normalen Epithels zu leisten hat. Oppikofer und Referent konnten an Präparaten von Nasenkarzinomen die Entstehung von Plattenepithelkarzinom auf dem Boden von Metaplasie nachweisen. Besonders interessant ist der Oppikofer'sche Fall, wo in der Schleimhaut einer wegen chronischer Kieferhöhleneiterung radikal operierten 66 jährigen Patientin im Bindegewebe massenhaft Karzinomnester gefunden wurden (Plattenepithel auf kurze Strecken).

Die Zylinderzellenkarzinome nehmen zum Teil von dem Schleimhautepithel, zum Teil von den Drüsen ihren Ursprung, wobei ich der durch Zarniko, Boenninghaus, Citelli, Glas, Hajek, Cordes und Oppikofer selbst studierten intraepithelialen Drüsen kurz Erwähnung tue, welche sich nach den Untersuchungen des letztgenannten Autors auch regelmässig auf normaler Schleimhaut finden. Harmer und Glas befürworten für das endonasale Karzinom Zylinderzellen- und Adenokarzinom nicht zu streng auseinanderzuhalten, indem manchmal die Entscheidung, welcher der beiden Typen vorliegt, wirklich schwierig ist. Dieselben Autoren konnten in einem ihrer Fällen von Zylinderzellenkarzinom die Abstammung des Tumors von den Drüsen direkt beobachten. Sie finden in der Umgebung echter Neubildung vermehrte, aber noch typische Wucherung der Drüsentubuli. Diese Beobachtung, welche den direkten Uebergang von anscheinend normalen Drüsenzellen in Elemente des Karzinomparenchyms zeigen, sprechen für die Hauser'sche Lehre, dass es eine primäre zur Geschwulstbildung führende Epithelerkrankung gebe, welche darauf beruhe, dass ursprünglich normales Epithel ohne vorherige Vorlagerung in das Bindegewebe sich direkt in Geschwulstepithel umwandelt.

Von anderen Karzinomformen wäre zu erwähnen das papilläre Karzinom oder der sogen. Zottenkrebs. In den meisten dieser Fälle handelt es sich um die benigne Form des Papilloms der Nase, welche noch später besprochen werden wird. Ueber Fibroadenom des Siebbeins mit karzinomatöser Entartung hat Preysing berichtet. Harmer und Glas beobachteten zwei Fälle von sog. osteoplastischem Karzinom (Recklinghausen). Ich verweise noch ausserdem auf die von den Autoren erörterte kleinzellige Infiltration und das Vorkommen von Riesenzellen in den Geschwülsten, sowie das von Alagna nachgewiesene Fehlen von Plasmazellen im Geschwulstparenchym. Auch Aenderungen der histologischen Struktur werden beobachtet. So berichtet Katz über

ein Basalzellenkarzinom (nicht verhornendes flaches Hautkarzinom sec. Donogany und Lenhart) der äusseren Nase bei einer 53 jährigen Frau, welches in das Naseninnere durchgewuchert war und die untere Muschel teilweise zerstört hatte. Auf dem Tumor der unteren Muschel waren mehrere, wenn auch verwandte Gewebsarten zu sehen, speziell auch Uebergang vom Platten- zum Zylinderzellenkarzinom. Es handelte sich höchstwahrscheinlich um sekundäres Karzinom (Abklatschkarzinom) von dem in das Innere der Nase einwuchernden Tumor der äusseren Nase entstanden.

Um die als *Endotheliome* bezeichneten eigenartigen Tumoren schwankt derzeit noch der Kampf der Meinungen. Während die einen sie entweder zu Sarkom oder Karzinom (Hansemann und Lubarsch) rechnen, die anderen zu den Sarkomen (Burkhard und Harmer) oder zu den epithelialen Geschwülsten (Ribbert, Krompecher, Perthes), oder endlich sie von der Entstehung aus pathologischen während des embryonalen Lebens verlagerten Epithelkeimen herleiten (Ribbert, Coenen, Fischer), glaubt eine Anzahl von pathologischen Anatomen und Rhinologen, ihnen einen festen Platz in der Gruppe der malignen Tumoren einräumen zu müssen. Die Lehre dieser interessanten Geschwülste ist besonders von Volkmann und Borst gefördert worden. Der Nachweis von Plattenepithelien mit charakteristischer Epithelfaserung, Bildung von Keratohyalinkörnchen und Verhornung würde entschieden gegen eine rein bindegewebige Herkunft sprechen, so dass dann nur noch die Frage der Entscheidung harrte, ob man diese Geschwulst als ‘Mischgeschwulst’ oder ‘Adenom’ auffassen und bezeichnen soll (Perthes). Alle diese untrüglichen Stigmata für die Herkunft aus Plattenepithel werden aber von Volkmann, der sich am meisten mit Endotheliomen beschäftigt hat, geleugnet. Die sog. Verhornung ist nach Althoff und Pollak nur Schichtung (Pseudoverhornung). Die geschichteten Zellen besitzen aber weder Keratohyalin, noch Protoplasmafasern, noch Interzellularbrücken. Zweifellos ist die Entstehung der Endotheliome von den Endothelien der Blut- und Lymphgefässe und Saftspalten histologisch nachgewiesen und ebenso zweifellos ist es, dass diese Tumoren nicht nur wegen ihres eigenartigen histologischen Baues, sondern auch wegen ihres differenten *klinischen* Verhaltens eine gewisse Sonderstellung in der Pathologie der malignen Nasentumoren nie verlieren werden. Althoff definiert Endotheliom als eine bösartige Geschwulst, welche von den Endothelien ausgeht. Letztere bestehen aus platten, grossen, protoplasmareichen Zellen, welche in epithelartiger Anordnung die Blut- und Lymphgefässe und die Bindegewebsspalten und serösen Höhlen auskleiden. Die Geschwülste zeichnen sich dadurch von den anderen malignen Tumoren aus, dass nicht lediglich die einmal entarteten Endothelzellen in die Umgebung eindringen, sondern dass immer aufs Neue normale präexistierende Endothelzellen der Spalten- und Lymphgefässe an der Geschwulstbildung in hohem Masse teilnehmen. Für das histologische Bild der Endotheliome ist charakteristisch der Aufbau aus Zellsträngen,

die teils Lumina bilden, teils solid verlaufen, und einem bindegewebigen Stroma, das exquisite Neigung zu hyaliner oder schleimiger Degeneration aufweist, welche beiden Degenerationsvorgänge sich oft in Uebergängen in einer und derselben Geschwulst finden. Die Zellstränge können die verschiedenartigsten Gruppierungen annehmen und die zierlichen netzartigen Verzweigungen, die man manchmal sieht, gehören zu den reizvollsten histologischen Bildern. Nach Borst ist vor allem wichtig das Vorkommen von soliden schmalen Zellsträngen in netzartiger Anordnung, die in ihrer ganzen Verteilung und Konfiguration an die Lymphspalten und Gänge des Bindegewebes erinnern, ferner der Habitus der Geschwulstzellen und die innige Zusammengehörigkeit und feste Verbindung zwischen Stroma und Geschwulstzellen. Die Unentscheidenheit der histologischen Einteilung der Endotheliome liegt zum Teil darin, dass eben auch die Zugehörigkeit der Endothelzellen noch unentschieden ist, ob zum Epithel oder Bindegewebe.

Zu den Endotheliomen sind auch die meisten der sog. *Zylindrome* zu rechnen. Der Begriff Zylindrom wurde bekanntlich von Billroth in die pathologische Anatomie eingeführt, da durch Herausfallen der zerstörten oder aufgelösten Zellstränge das hyaline Stroma in Form von Zylindern übrig bleibt. Borst definiert das Zylindrom als eine gallertige Geschwulst, die als charakteristische Bestandteile Kugeln, Kolben und Zylinder aus einer durchsichtigen hyalinschleimigen Substanz hervorbringt. Es kann sich dabei um Geschwülste verschiedener Gattung, wie Adenome, Sarkome, Karzinome und Endotheliome handeln. Die echten eigentlichen Zylindrome sind eine Varietät des Endothelioms. Bei ihm kommen die hyalinen Zylinder als besonders charakteristischer Bestandteil vor. Pollak konnte wie gesagt bereits 54 sichere Fälle zusammenstellen, darunter 1 Haemangioendothelioma intravasculare, 6 Haemangioendotheliomata perivascularia (Peritheliome), 31 Lymphangioendotheliome, 13 Zylindrome, 1 Psammom und 1 Parotistumor, dazu einen Fall von Zylindrom eigener Beobachtung.

Von den Tumoren sassen vier am Septum (Heimendinger und Hirose) und Nasengerüst, alle übrigen an der lateralen Nasenwand, resp. in den Nebenhöhlen, besonders in Kieferhöhle und Siebbein, seltener in Stirnhöhle und Keilbein. Das Alter variierte zwischen 16 und 77 Jahren. Als klinische Charakteristica sind vor allem ihre relative Gutartigkeit, langsames Wachstum hervorzuheben. Sie rezidivieren sehr leicht, machen aber nie Metastasen, nur in einigen Fällen kam es zu raschen Rezidiven und rapidem Wachstum. Manchmal bleibt der Tumor jahrelang klein und abgekapselt um dann plötzlich rapid zu wachsen und malignen Charakter anzunehmen. Referent hat zwei Fälle beobachtet, einen Oberkiefertumor bei einem 47 jährigen Mann seit 3 Jahren bestehend, auf die andere Seite übergreifend und einen Keilbeinhöhlentumor bei einer 47 jährigen Frau mit beiderseitiger Erblindung.

Nun zur *Symptomatologie* der malignen Tumoren der Nasen- und der Nebenhöhlen. Schon zu Beginn meines Vortrags habe ich die Malignität

und vor allem die düstere Prognose dieser Tumoren zum Teil mit der mangelhaften Diagnose erklärt. Leider sind erfahrungsgemäss die Anfänge der Tumoren derart symptomlos, dass viele von den Patienten erst zum Arzt kommen, wenn der Tumor die Nase oder die Nebenhöhle bereits durchbrochen hat und seine Fortsätze in unberechenbarerweise in die benachbarten Knochenlücken und Spalten vorgedrungen sind. Trotzdem muss es unser unentwegtes Streben sein, gerade dieses Anfangsstadium der Tumoren auf das Genaueste zu studieren und den geringsten Anhaltspunkt festzuhalten, der uns die Mittel in die Hand gibt, die radikale Operation zu einer Zeit auszuführen, in der noch mit Sicherheit in Gesunden operiert werden kann, was ja bei den meisten der zur Operation kommenden Fälle nicht mehr der Fall ist. Es hat nun im Verlaufe der Zeit die Erfahrung gezeigt, dass die Tumoren je nach dem Sitz eine bessere oder schlechtere Prognose bilden. So haben schon Harmer und Glas die Einteilung der malignen Tumoren in die relativ gutartigen Tumoren des vorderen Septumanteils und die gestielten Karzinome der unteren Muschel als Tumoren der unteren Etage gegenüber den prognostisch ungünstigeren der oberen Etage, zu welchen vor allem alle vom Siebbein ausgehenden Tumoren zu rechnen sind, befürwortet. An denen der ersten Gruppe sind sogar endonasale Eingriffe mit radikalem Erfolg ausgeführt worden (Bylau, Baber, Anari u. v. a.). Auch Moure unterschied im Jahre 1909 auf dem Budapester Kongress zwei Typen: (1) die Tumoren im vordern Drittel der Nasenhöhle, Septumboden und untere Muschel; (2) die Tumoren, die sich im oberen Teil der Nasenhöhle gegen die Nebenhöhlen zu ausbreiten, manchmal in den Nebenhöhlen geradezu entstehend. Aber schon 1910 unterscheidet er bereits 3 Typen: die Tumoren der unteren, oberen und mittleren Etage. Die der ersten Gruppe sind ziemlich selten und leicht endonasal zu operieren. Die der zweiten (Septumtumoren) ziemlich häufig, die Operation meist von aussen zu machen. Die der dritten mittlere und obere Partie der Nase sehr häufig, die Radikaloperation stets von aussen bietet trotzdem wenig Aussicht auf Erfolg. Eine weitere Schwierigkeit in der Deutung der Symptome liegt in der Maskierung durch andere Erkrankungen. Wir erinnern nur an die schon erwähnten und bei malignen Tumoren häufig genug gleichzeitig angetroffenen Polypen und chronischen Eiterungen. Ueber den Zusammenhang zwischen Tumor und diesen beiden Erscheinungen ist schon früher gesprochen worden. Gewöhnlich das erste Symptom des Wachstums maligner Geschwülste ist gleich wie beim benignen Tumor langsam zunehmende Verstopfung der Nase, eventuell Herabsetzung des Geruchsvermögens. Durch Verdrängung des Septums und weiteres Wachstum des Tumors (mehr beim Sarkom), durch Durchwucherung des Septums und Ausfüllung der anderen Nasenseite wird die Nasenatmung vollständig aufgehoben und es stellt sich Rhinolalia clausa und mehr weniger reichliche Sekretion aus der Nase ein. Bei den Sarkomen mehr wässerig schleimig, bei den Karzinomen bald blutig serös, schliesslich eiterig und jauchig. Handelt es sich um einen Tumor

der unteren Etage oder des Septums, so kann er besonders bei Sarkom noch ganz in Beginn sein, gut operabel, und schon diese Symptome machen; anders sind die Tumoren der oberen Etage, namentlich aber die Nebenhöhlentumoren. Diese können lange Zeit überhaupt symptomlos verlaufen. Aus diesem Grunde hat Citelli in seinem Referat 1908 drei Phasen der malignen Nebenhöhlentumoren aufgestellt: 1. Die Phase der Latenz; sie ist bei ihnen mehr ausgeprägt als bei den eigentlichen Nasentumoren und dauert länger. Sie besteht entweder in Symptomlosigkeit oder höchstens in leichten Druckerscheinungen in der befallenen Nebenhöhle und dem Gefühl der 'Gêne'. Dagegen kann schon in der Latenzperiode der rhinologische Befund positiv sein und Harmer und Glas zitieren eine Anzahl von höchst instruktiven Krankengeschichten, wo nur kleine verdächtige Granulationen im mittleren oder oberen Nasengang das einzige Symptom waren und bei der Operation der Tumor sich schon von erschreckender Ausdehnung gezeigt hat. Sehr frühzeitig ruft der Tumor besonders des oberen Nasenanteils sowie der Nebenhöhlen Polypenwucherungen hervor. Man wird bei einseitigen Polypen, welche trotz radikaler Operation immer wieder rezidivieren, stets vorsichtig sein müssen. Häufig kommt es vor, dass bei der Extraktion von Polypen einerseits eine profuse Blutung auftritt, welche keine Tendenz hat von selbst zu stehen. Ist die Blutung nicht so mächtig, dass die Rhinoskopie verwehrt ist, dann wird man als Basis der entfernten Polypen anstatt der normalen lateralen Nasenwandknochen der mittleren Muschel, eine weissliche, von den Polypen durch das saturierte Aussehen gekennzeichnete Aftermasse sehen. Probeexzision oder auch histologische Untersuchung der entfernten Polypen ergeben dann die eindeutige Diagnose. Es können auch reine Tumormassen polypenähnliche Gebilde ergeben und daher kommen Harmer und Glas zu der Forderung, alle Polypen, zumal jene, bei deren Entfernung es zu einer starken Blutung gekommen ist, einer mikroskopischen Untersuchung zu unterziehen. Die zweite Phase, die der Manifestation, ist für Nasentumoren die Nasenverstopfung, Rhinolalie, vermehrte Sekretion, endlich Auftreibung des Nasengerüstes. Durch Wachstum des Tumors in die Tiefe kommt es zur Arrosion grösserer Gefässe und es könnten schon jetzt stärkere Blutungen auftreten. Diese Blutungen werden im allgemeinen beim Sarkom häufiger als beim Karzinom beobachtet. Bei letzterem nur nach operativen Eingriffen (Probeexzision), doch hat Harmer auch bei einer Reihe von Karzinomen spontane Blutungen beobachtet. Es entspricht das seltenere Auftreten der Blutungen beim Karzinom dem Aufbau der Geschwulst, der durch seine Gefässarmut bekannt ist. Bei den Sarkomen kann das Gefässreichtum so gross sein, dass der Tumor pulsiert und die Blutungen spontan und bei Berührung entstehen, dann nicht durch Arrosion der normalerweise vorgebildeten Gefässe, sondern der neugebildeten zartwandigen Tumorgefässe.

In dieser Periode erscheint auch gewöhnlich schon ein weiteres Hauptsymptom, die Schmerzen. Diese Schmerzen sind in der Nase meist

nicht lokaler Natur, sondern entweder neuralgiform durch Druck auf die in der Umgebung verlaufenden Nerven oder, wie besonders bei den Tumoren der oberen Etage, diffuse Kopfschmerzen. Diese Kopfschmerzen erklärt Harmer durch das Vorhandensein von Nebenhöhleneiterungen als Drucksymptom, als reflektorisch von einem komprimierten Nerven ausgelöst, durch Meningen-Hyperämie. Letztere fand sich bei der Obduktion in einem Falle dieses Autors und in einem anderen von Biehl als Erklärung der hochgradigen Kopfschmerzen der letzten Lebenstage. Die Neuralgieschmerzen treten vorzugsweise beim Karzinom auf durch direkte Zerstörung der benachbarten Nervenstämme. Typisch sind die neuralgiformen Schmerzanfälle der Zahnnerven des Oberkiefers, oft als Frühsymptom eines Kieferhöhlen-Tumors lange Zeit allein bestehend. Häufig kommt es endlich zur Extraktion sämtlicher Zähne des Oberkiefers, natürlich ohne dass die Schmerzen sistieren. Zahnschmerzen sämtlicher Zähne ohne Organerkrankung der Zähne wäre also bei Erwachsenen und älteren Personen namentlich wenn sie einseitig auftritt, als verdächtiges Symptom zu vermerken. Durch Druck auf die Nn. supra- und infraorbitales entstehen die sogenannten neuralgiformen Kopfschmerzen, welche nach Moll besonders bei Karzinom beobachtet werden. Hinterkopfschmerzen werden von Sendziak als pathognomonisch für Keilbeinhöhlentumoren bezeichnet. Differential-diagnostische Bedeutung hat der erste Punkt (Schmerzen durch Nebenhöhleneiterung), da der Tumor wenigstens im Anfang durch die Nebenhöhleneiterung, an deren Symptomen nichts auffällig ist, völlig maskiert werden kann. Harmer macht auf die Häufigkeit des Kieferhöhlenempyems bei malignen Nasentumoren aufmerksam, dadurch entstanden, dass die Kieferhöhle durch ihre Grösse und auch infolge ihres Baues als Reservoir für alle von oben kommenden Sekrete dient. Eine Eiterung, die auf konservative Behandlung sich nicht bessert, wäre als verdächtig zu bezeichnen, namentlich, wenn das Sekret nicht das typische Aussehen der gewöhnlichen Nebenhöhleneiterung hat, sondern das Spülwasser diffus getrübt und fötid bleibt, eventuell mit Blutstreifen vermengt ist.

Eine weitere Symptomenreihe kommt zustande, wenn der Tumor die Knochenwände ausdehnt oder durchbricht und die Umgebung zuerst komprimiert, dann in dieselbe einwuchert. Dieses wichtigste Symptom, welches oft als erstes auf einen Tumor aufmerksam macht, erzeugt bei den Nasentumoren Auftreibung der einen Seite des Nasenrückens oder der Nasenwurzel, bei der Kieferhöhle die charakteristische derbe Wangenauftreibung mit den typischen erweiterten Hautvenen des Oberkiefertumors, bei den Stirnhöhlentumoren eine buckelförmige Vorwölbung im innern Augenwinkel. Bei Siebbein- und Kieferhöhlentumor kommt es wegen der Dünnheit der Orbitalwände relativ frühzeitig zu Kompressionserscheinungen von Seiten der Orbita, Verdrängung des Bulbus, Lid- und Konjunktivalödem und Exophthalmus. Auch Blenorrhöe (non lacrimalis) wird von Sendziak in den Fällen von Stirnhöhlen- und Siebbeintumoren als diagnostisch wertvolles Symptom angegeben. Die Beweglichkeit des Bulbus ist

anfangs noch erhalten, geht aber infolge der nicht lange ausbleibenden Affektion der Augenmuskelnerven verloren. Es ist das Verdienst Onodis uns mit der überraschenden Mannigfaltigkeit der anatomischen Beziehungen der Hirn- und speziell der Augennerven zu den Nebenhöhlen bekannt gemacht zu haben. Onodi gebührt auch das Verdienst, sich als Pathologe mit den klinischen Beziehungen zwischen Augen- und Nebenhöhlen beschäftigt zu haben und den Anstoss gegeben zu haben, dass sich auch die Rhinologen eingehender mit den Augenveränderungen bei Nebenhöhlenerkrankungen befassen, was wieder auf den Fortschritt der Ophthalmologie günstigen Rückschlag ausübte. Auch Schmiegelow verdanken wir eine wertvolle Studie über die Bedeutung von Nebenhöhlenerkrankungen für die Augenpathologie. Ich nenne weiter die Namen von V. d. Hoeve, A. de Kleyn, Wallis, Edin, Whinnie, Markbreiter, Birch-Hirschfeld. Weitere Beiträge zu den Beziehungen zwischen Keilbeinhöhle und Hirnnerven verdanken wir Sluder, Halasz und Onodi jun. Diese anatomischen Arbeiten erwiesen in zahlreichen Fällen, dass die Augennerven und anderen Hirnnerven von den Nebenhöhlen, vorzugsweise Keilbein und Siebbein, aber auch Kieferhöhle und Stirnhöhle oft nur durch papierdünne Knochenlamellen getrennt sind. Manchmal aber auch fehlen diese Wände ganz und der *Nerv bildet einen Teil der Wandung der Höhle.* Durch diese Studien ist also einerseits die Häufigkeit und die Klinik der durch Nebenhöhlenerkrankungen hervorgerufenen Augenaffektionen erklärt, andererseits aber auch die manchmal oft erschreckend rasche Affektion von Hirnnerven, besonders aber von Augennerven im Verlaufe des Wachstums eines malignen Tumors. Speziell über die Affektion von Hirnnerven bei Keilbeintumoren haben Hunter Tod, Behring und Wichenkiewicz, Sluder, Hall, Adam, Schmiegelow, Ziem berichtet. Die Keilbeinhöhle ist in dieser Beziehung am ungünstigsten daran, wegen ihrer Einschachtelung inmitten der Gesichts- und Schädelknochen und der von den oben genannten Autoren studierten Nachbarschaft der Hirnnerven, und zwar des II., III., IV., V., VI., aber auch des IX., X., XI., XII. Durch Onodi u. a. ist aber auch nachgewiesen worden, dass die hinteren Siebbeinzellen sehr häufig in dem Keilbeinkörper eingelagert sind und dann die physiologische und pathologische Rolle der Keilbeinhöhle übernehmen können, ja auch dass die Keilbeinhöhle und die Siebbeinzellen der einen Seite durch Ausdehnung auf die andere Seite Hirnnervenerkrankung der contralateralen Seite hervorrufen können. Nach Kümmel kommt es bei Keilbeinhöhlentumoren manchmal zur Schädigung des zweiten Trigeminusastes am Foramen ovale, später auch anderer Zweige, meist auch des Opticus, und zwar in Form der Neuritis retrobulbaris mit konsekutiver Atrophie, endlich zu Neuralgien des Bulbus und Keratitis neuroparalytica, während für Siebbeingeschwülste häufiger frühzeitige Erkrankung des ersten Trigeminusastes in der Fissura orbitalis superior, Exophthalmus und Lähmung einzelner Augenmuskeln typisch ist.

Bei den Tumoren der hinteren Nase, sowie denen von Kieferhöhle,

hinterem Siebbein und Keilbein kann es auch frühzeitig zur Beeinflussung des Gehörs kommen, und zwar ist es zunächst nur durch Druck auf die Tube oder Verschluss derselben durch Tumormassen hervorgerufen. Erst später und vor allem bei der sekundären Affektion der Schädelbasis kann es zu vollständiger Taubheit durch Einwucherung des Tumors in das innere Ohr oder durch Affektion des Akustikus kommen. Diese letztere und Facialiserkrankung werden im allgemeinen mehr bei den sekundären Tumoren beobachtet. Zahlreiche Beobachtungen der letzten Zeit haben uns gelehrt, dass das Nervensystem, besonders aber die Sinnesnerven wohl sehr empfindlich gegen mechanische und toxische Schädigung sind, dass aber eine vitale Schädigung oder Zerstörung der Nervenfasern erst relativ spät erfolgt, ja dass die Nerven in dieser Beziehung sogar ziemlich widerstandsfähig sind, und zwar nicht nur gegen Entzündung, sondern auch gegen maligne Neubildungen. Es ist also zunächst immer die Funktion, welche gestört ist, und welche, wenn die Noxe rechtzeitig behoben wird, trotz anscheinender schwerster Schädigung des Organs vollständig sich wieder restituieren kann. So kann durch Stauungsödem der hinteren Orbita, welches durch Kompression der Venen durch einen in der Nähe wachsenden Tumor erzeugt wird, eine vollständige Amaurose entstehen, welche nach der Operation des Tumors und Behebung der Stauung wieder verschwinden kann, ohne dass dabei der Tumor schon in die Orbita eingebrochen zu sein braucht. In dieser Beziehung zeigt sich ebenfalls wieder die Gutartigkeit der Sarkome, die zunächst die Wandung der Nebenhöhlen mehr ausdehnen und mehr verdrängend wuchern gegenüber den mehr destruierend wachsenden, die Knochen frühzeitig durchbrechenden Karzinomen. Ueber die Widerstandsfähigkeit der peripheren Nerven gegenüber malignen Neoplasmen hat uns die Erfahrung bei unseren zahlreichen Drüsenoperationen maligner Tumoren am Hals, wo oft die eine Halsseite einen starren Tumor bildet, der schon die grossen Gefässe in sich aufgenommen hat, belehrt und trotzdem die Nerven wie in einem Kanal durch ihn hindurchlaufen, welche, wenn der Druck oder toxische Schädigung fehlt, auch vollständig in der Funktion erhalten sein können. Es würde das für die Prognose namentlich bei Sarkomen eine gewisse Bedeutung haben, da wir hoffen können, auch bei schon eingetretener Nervenlähmung noch ein radikales Resultat zu erzielen.[1]

Eine gleichfalls mehr bei sekundären Tumoren, bei primären Kieferhöhlentumoren nur dann wenn sie mehr an der lateralen Kieferhöhlenwand wuchern und diese zerstören, beobachtete Erscheinung ist *Ankylostoma*, durch Beeinflussung des Kiefergelenks, und zwar ist es auch hier wieder zunächst durch Stauungsödem erst später durch wirkliche Zerstörung des Gelenkes erzeugt und zeigt sich das Karzinom durch frühzeitige Affektion der Umgebung der Nebenhöhlen in seiner ganzen Bösartigkeit.

[1] Behring und Wichenkiewicz, Rimpler, Onodi, haben Fälle berichtet bei denen, trotzdem die Geschwulst der Keilbeinhöhle den Sehnerven vollkommen einbettete, von seiten desselben keine auffallende Störung beobachtet werden konnte.

Wir kommen nun zu den Erscheinungskomplexen, die meist in der dritten und letzten Phase nach Citelli zu beobachten sind, der Phase der Kachexie, die aber auch manchmal überraschend früh innerhalb weniger Monate auftreten können, das sind die *endokraniellen Symptome*. Sie sind naturgemäss eine typische Erscheinung bei den Tumoren der oberen Etage, also Nasendach und besonders Stirnhöhle und Siebbein. Sie können entweder bedingt sein durch Einwachsen des Tumors in die Schädelhöhle, meist durch die Lamina cribrosa, seltener durch das Siebbeindach hindurch, oder durch endokranielle Entzündungen infolge der durch den Tumor gesetzten Nebenhöhleneiterungen oder Infektionen. Die letztere Form ist vor allem durch ihren mehr akuten Verlauf und das gleichzeitige Vorhandensein von Fieber gekennzeichnet. Es sind alle Arten beobachtet worden: Meningitis serosa und suppurativa, circumscripta und generalis, Extradural- und Hirnabzess. Harmer und Glas haben ausserdem auf die Häufigkeit der Meningitis bei operativ und konservativ behandelten Nasentumoren aufmerksam gemacht. Uns interessiert hier mehr die erste Form, weil gerade von der genauen Kenntnis dieser Symptome die Prognose und eventuell auch die Indikation für den operativen Eingriff abhängt. Das Einwachsen des Tumors erfolgt entweder durch direkte Zerstörung aller im Wege liegenden Gewebe beim konzentrischen Wachstum oder durch infiltratives Wachstum in Verfolgung der präformierten Knochenlücken der Blut- und Lymphwege. Speziell die Lamina cribrosa ist in dieser Beziehung eine beliebte Einbruchspforte der Tumoren. Aber auch auf *metastatischem* Wege, durch die Lymphbahnen kann die Einwanderung von Tumorelementen erfolgen. Die Frage des Zusammenhanges der Lymphgefässe der Nase mit dem Schädelinnern, vor allem den subarachnoidealen Lymphräumen, haben, nachdem man ihn früher hauptsächlich den Blutgefässen zugeschrieben hatte, Schwalbe, Axel Key, Retzius, Zuckerkandl, in neuerer Zeit Poli, Broeckaert, Hartz und Grünwald geklärt und die Kommunikation der Saftspalten und des Lymphgefässnetzes der Nase mit den intermeningealen Räumen sichergestellt. Direkte Lymphwege ziehen auf dem Wege der Olfactoriusscheiden durch die Lamina cribrosa ins Gehirn. Zwillinger hat im oberen Viertel des Septums Lymphbahnen gefunden, welche unabhängig von den perineuralen Olfactoriusscheiden die Verbindung zwischen Nase und Gehirn herstellen, und führt auf diese ausser den Blutbahnen die postoperativen und andersartigen cerebralen Komplikationen zurück. Die Existenz von Lymphwegen zwischen der Stirnhöhlenschleimhaut und dem Gehirn direkt durch die Stirnhöhlenhinterwand hindurch hat Falconer festgestellt. Durch Vermittelung aller dieser Lymphbahnen kann es, wie in dem lehrreichen Harmer'schen Fall, zu einer Einwanderung des Tumors in die Schädelhöhle und mit Umgehung der Lamina cribrosa von da auf die andere Seite zur Etablierung des Tumors in der anderen Nasenhöhle kommen. Von den Symptomen, die das Einwachsen des Tumors in die Schädelhöhle anzeigen, werden von allen Autoren in der Regel dumpfer Kopfschmerz, Stupor, Schlafsucht und Apathie angegeben,

seltener Herzsymptome, Schwindel, Krämpfe. Intrakranielle Drucksteigerung kommt begreiflicherweise, da der einwuchernde Tumor ja geradezu den Schädel trepaniert, meist nicht zur Beobachtung, dagegen kann es durch Eröffnung des subarachnoidalen Raumes neben der oft beobachteten Meningitis zum Abfluss von Liquor cerebro-spinalis und zur Rhinorrhöe kommen, wie in dem Fall von Boyd, in dem ein Hypophysentumor in der Schädelbasis eine Lücke gesetzt hatte, die mit der Kieferhöhle kommunizierte.

Die Frage der Lymphwege führt uns zum letzten Kapitel der Symptomatologie, das sind die *Metastasen*. Die Erfahrung hat gezeigt, dass die Nasen- und Nebenhöhlentumoren recht selten Metastasen setzen, von manchen Autoren werden sie direkt geleugnet. Es zeigt sich auch hier der Wert der Erfahrung grösseren einheitlichen Krankenmaterials im Gegensatz zu vereinzelten Beobachtungen. Anscheinend scheint in den meisten früheren Fällen eine Untersuchung aller Lymphdrüsen der Umgebung unterlassen worden zu sein. Diese Seltenheit der Metastasen, und zwar sowohl der Lymphdrüsen als insbesondere der Fernmetastase verschafft den Nasentumoren, auch den bösartigeren, ihre relative Gutartigkeit. Worin die Seltenheit der Lymphdrüsenmetastasen teilweise wenigstens beruht, haben Harmer und Glas, später Poli und Broeckaert, gezeigt. Nicht die in der Regel untersuchten und beobachteten Submaxillardrüsen sind die regionären Drüsen der inneren Nase, sondern die retropharyngealen Drüsen an der Seitenwand des Nasenrachenraums und des Pharynx, von diesen erst kommt es sekundär zur Infektion der Parotisdrüsen und der tiefen Cervikaldrüsen; die submaxillaren Drüsen werden am seltensten und erst in zweiter Linie infiziert. Harmer und Glas haben besonders auf die Wichtigkeit dieser retropharyngealen Drüsen in Bezug auf die Radikalbehandlung hingewiesen, und auch ich möchte besonders die Operateure von Nasentumoren darauf aufmerksam machen, dass ein vorgeschrittener Nasentumor ohne Ausräumung dieser Drüsen nicht radikal operiert werden sollte. Unter denselben Gesichtspunkten wäre der von Poli betonte Zusammenhang der Nasenlymphbahnen mit den Tonsillen zu beachten.

Nach Most richtet sich der Hauptstrom der Lymphe des Naseninnern nach den Choanen hin und sammelt sich in der seitlichen Pharynxwand, direkt unter dem harten Gaumen nach den pharyngealen Tubenostium und etwas unterhalb desselben, auch vom Septum. Von da zieht die Lymphe in zwei Hauptströmen, erstens nach aussen und unten zu den vorderen und tiefen Cervikaldrüsen, zweitens der wichtigste Strom aber zieht nach hinten zu der seitlichen Retropharyngealdrüse. Diese endonasalen Lymphbahnen sind relativ lang bis zu den nächsten Drüsen, das Karzinom (Katz) führt daher durch sein infiltratives Einwachsen in lebenswichtige Organe früher zum Tod, bevor die Lymphdrüsen erheblich erkranken können.

Wie uns die in neuerer Zeit an der Wiener Klinik Professor Chiaris besonders häufig geübte Ausräumung und Untersuchung der regionären Lymphdrüsen bei malignen Neoplasmen gezeigt hat, handelt es sich bei

vielen der makroskopisch als malign erkrankt und infiltriert befundenen Lymphdrüsen nur um einfache Entzündung, Vereiterung oder Tuberkulose.

Von den Fernmetastasen sagt Broeckaert, dass die Lungen in der Regel verschont bleiben, obwohl die Keime, wenn sie in die Blutbahn kommen, doch notwendigerweise dieses Organ passieren müssen. Sie siedeln sich mit Vorliebe in gewissen Organen an; manche Metastasen können nur durch 'retrograde Invasion' (Recklinghausen) erklärt werden.

Eine *Beeinflussung des Gesamtorganismus* durch die malignen Nasen- und Nebenhöhlentumoren kommt in der weitaus grössten Zeit des Tumorwachstums nicht vor, wenn wir von den cerebralen Komplikationen absehen. Daher auch die relativ lange Krankheitsdauer, über die wir schon bei den Sarkomen gesprochen haben. Bei den Karzinomen ist die Dauer allerdings wegen der destruktiven Tendenz des Tumors, der früher die grösseren Gefässe arrodiert, Lymphdrüsen und Fernmetastasen setzt und auch infolge der Jauchung den Allgemeinzustand mehr beeinträchtigt, etwas kürzer als beim Sarkom, doch hat u. a. Nicolai von einem Karzinom berichtet, das 7 Jahre gedauert haben soll. Die Nahrungsaufnahme ist meist nicht gestört, wenn nicht der Tumor den weichen Gaumen affiziert oder durch Infiltration der Schädelbasis, die Zungen- und Schlundnerven lähmt. Der Tod erfolgt daher auch, von den operativen Todesfällen abgesehen, meist vor der Krebskachexie durch cerebrale Komplikationen oder Pneumonie infolge von Aspirationen infektiösen Sekrets während des Schlafes.

Als Kuriosum der Beeinflussung eines malignen Tumors durch den Gesamtorganismus möchte ich den von Lazarraga 1910 berichteten Fall erwähnen, wo bei einer 33 jährigen Frau ein ulceriertes Sarkom des Gaumens und der Nase mit Zerstörung des Septums zu Beginn der *Menses* jedesmal verstärkte Blutungen und entzündliche Schwellung zeigte.

An dieser Stelle muss über die diversen Beobachtungen berichtet werden, welche als wahre Kuriosa unsere ganzen Erfahrungen von der Klinik der malignen Tumoren umzustossen scheinen, aber meines Erachtens doch nichts anderes als die nirgends fehlenden Ausnahmen von der Regel der keinen Absolutismus noch Schematismus duldenden Natur zu betrachten sind. Da ist zunächst der schon im Anfang kurz erwähnte Fall von Knight, in dem eine endonasal entferntes Sarkom erst nach 13 Jahren rezidiviert wird, und dann nach der zweiten Radikaloperation wieder durch $3\frac{1}{2}$ Jahre rezidiv frei blieb. Der Autor sagt, nachdem er auf das auch in anderen Gebieten der Pathologie häufig beobachtete Verschwinden der malignen Tumoren hinweist: Diese späte Recidiv gibt zu denken. F. Semon hält ein Jahr Dauerheilung für genügend zur definitiven Heilung, Ruault verlangt sieben Jahre, gewöhnlich sagt man drei Jahre: man wird die Zeit der Radikalheilung noch mehr ausdehnen müssen. Bekannt ist ferner die Einwirkung des Erysipels auf gewisse maligne Tumoren. Diese Fälle sind nach Ramon y Cajal, der über eine eigene

Beobachtung berichtet, besonders bei Sarkomen ziemlich zahlreich, so dass man sogar auf die Impfung mit dem Micrococcus Fehleisen Hoffnungen gesetzt hat, welche sich aber nicht erfüllt haben, obwohl Coley neben glänzenden Resultaten sogar Heilungen erreicht haben will. In anderen Fällen wurde aber der Verlauf hierdurch geradezu beschleunigt. Ferner wäre hier die spontane Rückbildung maligner Tumoren zu nennen, ohne irgend nachweisbaren Anlass. Solche Fälle unseres Gebietes haben unter anderen Robertson und Coban gesehen; letzterer einen Tumor des Septums und einen solchen der Rachenhinterwand. Eine interessante Studie aus dem Jahre 1912 stammt vom Theilhaber, in der der Autor 200 derartige Beobachtungen zusammenstellt. Ja er versteigt sich geradezu zu der Behauptung, dass viele Aerzte solche Beobachtungen verschweigen, in der Annahme vorher eine falsche Diagnose gemacht zu haben. Seine Untersuchungen führen ihn zu merkwürdigen Folgerungen: Das Karzinom entwickelt sich nicht im normalen Gewebe, vorerst ist das subepitheliale Gewebe atrophisch, die Zahl und Grösse der Zellen ist verringert, es besteht Anämie (Spärlichkeit und Obliteration der Gefässe). Manches Rezidiv tritt nur deshalb ein, weil die Operationsnarbe ein im schlechten Ernährungszustande befindliches Gewebe ist. Man hebe die Ernährung der Narbe etwa durch Hebung des allgemeinen Zustandes, Gebirgsluft, Seebäder, durch Stauung und Hyperämisierung der Narbe (Heissluftbehandlung, Fibrolysin, Sonnenbäder). Vielleicht regt auch der starke Blutverlust bei der Operation die Resorption zurückgebliebener Karzinomzellen durch die konsekutive Reaktion des Organismus an. Theilhaber hat daher im letzten Jahre zwei Wochen nach der Operation stets einen Aderlass von 400 Kubikcentimeter folgen lassen. Ich kann nicht umhin, obwohl ich damit eigentlich schon in das Gebiet der Prognose mich verirre, einige interessante Bemerkungen anzuführen, die Koschier anlässlich der Publikation zweier ungewöhnlich verlaufener Fälle von Nasenrachenraumtumoren über das Tumorwachstum im allgemeinen gemacht hat: Sarkom und Karzinom wachsen bei jugendlichen Individuen viel rascher, auch die Metastasen treten früher auf. Einen ähnlichen Einfluss auf die Wachstumschnelligkeit übt die Beschaffenheit des engeren Trägers, das ist das befallene Organ. Auch wachsen die Tumoren viel schneller wenn sie wiederholten, wenn auch kleineren mechanischen Insulten ausgesetzt sind. Endlich zeigt Zellreichtum beim Sarkom, Verhornung beim Karzinom die relative Bösartigkeit an. Wir können den Verlauf auf Grund der statistischen Erfahrung im Einzelfall mehr oder weniger abschätzen. Manchmal aber lässt uns die Prognose ganz im Stich. Maligne Neoplasmen fangen plötzlich rapid zu wachsen an, spontan oder durch interkurrente Erkrankung, eingreifende Behandlung, operative Eingriffe; manchmal aber ist auch das gerade Gegenteil der Fall. Der alte Organismus ist viel wiederstandsfähiger als der junge, entschieden sind die einzelnen gleichartigen Tumoren von verschiedener Virulenz; eine Sonderstellung nimmt das Lymphosarkom ein.

Ueber die *Diagnose* der malignen Nasen- und Nebenhöhlentumoren

kann ich mich mit Beziehung auf die schon besprochene Symptomatologie kürzer fassen. Zunächst sei die Förderung hervorgehoben, die die Diagnostik in neuerer Zeit durch die Vervollkommnung der Untersuchungsmethoden erfahren hat. Um einen malignen Tumor zu diagnostizieren, stehen uns, wofern auf Grund der eben besprochenen Symptome im Einzelfall der Verdacht eines solchen rege geworden ist, ausser der (1) Rhinoskopie noch folgende Mittel zur Verfügung: (2) die Durchleuchtung, (3) das Röntgenverfahren, (4) die Probeexzision, (5) die Serumreaktion.

ad 1. Die Rhinoskopie hat, namentlich wenn es sich um die Diagnose maligner Tumoren handelt, immer auch durch die hintere Rhinoskopie ergänzt zu werden, welch letztere mit allen Mitteln durchzuführen ist, d. h. man darf sich nicht damit begnügen, ein flüchtiges Bild eines Teiles der Choanen zu bekommen. Abgesehen von den seit jeher angegebenen Methoden der Fixation des weichen Gaumens besitzen wir in den diversen Pharyngoskopen nach Flatau, Schmuckert, Hay-Kahler-Leiter etc., besonders aber in dem von Zaufal zuerst angewendeten, dann von Valentin und Hirschmann, auch von Leiter verbesserten Salpingoskop wertvolle Untersuchungsinstrumente. Während aber die Pharyngoskope mehr Demonstrationszwecken dienen und eigentlich das Choanenbild nicht wesentlich klarer und heller bringen als der postrhinoscopische Spiegel, auch gleich diesem von der Konfiguration, Beweglichkeit und Reizbarkeit des weichen Gaumens mehr weniger abhängig sind, scheint mir das Salpingoskop eine grössere Bedeutung für die Nase zu haben, als man gewöhnlich annimmt. Nicht nur, dass es durch die Vergrösserung des Bildes und durch die Nähe der Lichtquelle eine grelle und äusserst deutliche Beleuchtung der Choanen herstellt, für den Patienten auch viel angenehmer ist als manche hintere Rhinoscopie oder Untersuchung mit dem Pharyngoskop, können wir vor allem auch während der Einführung des Instruments die ganze Nasenhöhle von unten her beobachten, und schon bei normalen Nasen ist man von der Fülle unbekannter Details überrascht, die man mit dem gewöhnlichen Nasenspiegel nie gesehen hat. Die Untersuchung gerade der für die malignen Tumoren so wichtigen oberen Nasenpartie ist viel eingehender möglich. Ich selbst habe mit dem Instrument in den letzten Jahren viel gearbeitet und kann es für die Rhinologie nur dringendst empfehlen. Es leistet verhältnismässig mehr als das Pharyngoskop, ist billiger und ist auch für die Untersuchung der Kieferhöhle durch eine Oeffnung im unteren Nasengang brauchbar.

ad 2. Die Durchleuchtung ist in neuerer Zeit fertig ausgebildet worden und Vohsen, Gyergyai u. a. haben das Instrumentarium bedeutend verbessert. Letzterer hat ein Instrument angegeben, welches, in den Nasenrachenraum eingeführt, genauen Vergleich der beiden Seiten bezüglich ihrer Helligkeit gestattet. Immerhin sind die Untersuchungsergebnisse dieser Methoden Irrtümern und Paradoxen ausgesetzt; sie werden dem

ad 3. Röntgenverfahren gegenüber immer inferior bleiben. Ueber

dessen Fortschritte auf unserem Gebiet brauche ich wohl die rhinologische Welt nicht eingehender zu unterrichten. Allen bekannt sind die Leistungen von Killian, Kuttner, Scheier, Spiess, Winckler, Goldmann, Wassermann, Caldwell, Coakley u. a. Referent selbst hatte in mehrjähriger Zusammenarbeit mit dem Röntgenologen Schüller, der seine Erfahrungen und Forschungen jetzt in einem klassischen Werk niedergelegt hat (*Röntgenologie des Schädels*), Gelegenheit, auch viele benigne und maligne Nasen- und Nebenhöhlentumoren zu untersuchen und das Ergebnis des Röntgenverfahrens mit dem pathologisch-anatomischen Befunde zu vergleichen. Das Material stammte zum grössten Teil aus der Wiener rhinolaryngologischen Klinik. Einige von diesen Erfahrungen haben wir in einem Vortrag auf dem Internationalen Kongress 1911 in Berlin publiziert. Ungeachtet der Ueberzeugung von dem grossen Wert des Verfahrens möchte ich aber doch vor einer allzu optimistischen Verfolgung seiner Ergebnisse warnen. Man wird dann Enttäuschungen leichter vermeiden. Es ist schon bei den katarrhalischen Erkrankungen oft genug das Röntgenbild mit dem anatomischen Befunde schwer in Einklang zu bringen, umsomehr ist Vorsicht bei der Diagnose eines malignen Tumors am Platze, wenn wir auch dazu noch die Seltenheit gegenüber den katarrhalischen Entzündungen bedenken. Eine sichere röntgenologische Diagnose ist nach unseren Erfahrungen nur bei Knochen- und kalkhältigen Tumoren möglich, also meist benignen Formen. Immerhin können wir Jansen zustimmen, der sagt: 'Bisweilen steht das Resultat der Röntgendurchleuchtung in striktem Gegensatz zu dem der elektrischen Durchleuchtung. Meist liefert das Röntgenverfahren das zuverlässigere Ergebnis, gewöhnlich aber ergänzen sich beide, indem das eine die Durchleuchtung von hinten, das andere die von unten besorgt.' Bei malignen Tumoren kann das Röntgenbild falsche Diagnosen verhüten, aber auch direkt erzeugen. Manchmal kann der Tumor in der Orbita sitzen und die anliegenden Nebenhöhlen sind nur katarrhalisch oder ödematös. Beweisend für die malignen Tumoren ist in der Regel der Nachweis von Wanderkrankung. Ich habe in dem vorerwähnten Vortrag auch über unsere Erfahrungen bezüglich der Differenzialdiagnose zwischen Keilbein und Hypophysentumoren berichtet.

ad 4. Probeexzision und histologische Untersuchung. Auch hier müssen wir, indem wir ihr einen Hauptanteil an der exakten Diagnosestellung zumessen, vor allzu gläubiger Verwertung des Ergebnisses derselben warnen. Zahlreich sind die Berichte, die diese scheinbar exakteste und untrüglichste diagnostische Methode in anderem Licht erscheinen lassen. Um nur einige wenige hier zu nennen, so hat W. J. Howe in der Royal Society of Medicine in London verschiedene Präparate von Sarkomen demonstriert und auch von Fällen, welche Sarkom vortäuschen, und kommt zum Ergebnis, die Histologen machen öfters die Diagnose Sarkom als es in der Tat vorliegt. Nur wenn die pathologisch-histologische Diagnose Sarkom negiert, kann man sicher sein, dass auch keines vorhanden ist. Hingegen berichtet wieder Ruault über einen Fall von Sarkom der Nase

bei einem Kind, das alle klinischen Anzeichen eines malignen Tumors bot, und trotzdem konnte die histologische Diagnose Sarkom nicht nachweisen. Zwei Jahre nach der Operation kam es wieder zu Rezidiv, das Kind überlebte die Operation noch 6 Jahre, starb aber schliesslich an unzweideutigen Erscheinungen eines malignen Tumors. Auch Ruault meint, man dürfe sich nicht immer auf das Mikroskop verlassen. Mériel schreibt über entzündliche Tumoren der Gliedmassen, welche Sarkome vortäuschen und meint, dass unter der Bezeichnung Sarkom noch verschiedene pathologische Zustände zusammengefasst werden. Die histologische Diagnose gestatte nicht immer die Unterscheidung zwischen Sarkom und entzündlichem Tumor. Wahrscheinlich beziehen sich auch die als Heilwirkung des Radium, der Röntgenstrahlen etc. bei Sarkom beschriebenen Beobachtungen in Wirklichkeit auf entzündliche Tumoren. Durch die neueren Forschungen seien Beziehungen zwischen Entzündung und Neoplasma, sowie das Vorhandensein von Uebergangsformen festgestellt. Katz spricht von der Schwierigkeit der histologischen Diagnose der endonasalen Tumoren. Man soll das Stück zur Probeexzision immer möglichst weit weg von der Schleimhautbedeckung des Tumors entnehmen, weil die Schleimhaut desselben, welche dem Naseneingange benachbart ist, manchmal Veränderungen eingeht, die dem Sarkom sehr ähnlich sind. Wir können Katz nach unseren reichen Erfahrungen der Wiener Klinik nur beipflichten. Weiter möchte ich empfehlen, stets Stücke von mehreren Stellen zur gleichzeitigen Untersuchung zu entnehmen. Einen lehrreichen Beitrag liefert Zenker:

60 jähriger Mann, maligner Tumor der Nase seit einem Jahr. Erste Probeexzision ergibt Fibrom mit starker kleinzelliger Infiltration. Zweite Probeexzision, Grundzellensarkom. Operation. Dritte histologische Untersuchung vom hinteren Nasendrittel: Adenokarzinom; also drei histologische Untersuchungen an drei scheinbar örtlich differenten, jedoch klinisch einem und demselben Tumor angehörigen Gewebsstücken brachten jedesmal eine andere Diagnose. Primär war offenbar nur das maligne Adenom, sekundär die fibromatöse Wucherung und kleinzellige Rundzelleninfiltration von sarkomähnlichen Massen.

Auch von derartigen Fällen haben wir mehrere an unserer Klinik beobachtet. Besonders die im Gefolge von sekundärer Entzündung entstehende kleinzellige Infiltration und entzündliche Neubildung von Gewebe kann zu den grössten Irrtümern führen. Ich erinnere mich speziell an einen Tumor des Siebbeins und der Orbita, bei dem eine Inzision in die Orbita zur Vereiterung geführt hatte. Der durch die Radikaloperation gewonnene Tumor veranlasste den Pathologen zum grössten Teil zur Diagnose: Rundzellensarkom. Erst die vollständige Durcharbeitung des Tumors führte zu der Entdeckung, dass es sich um Karzinom handelte. Das kleinzellige Rundzellensarkom war alles entzündliche Infiltration. Noch eine Bemerkung zur Frage der Probeexzision: unsere Erfahrung hat gelehrt, dass die malignen Tumoren auf Infektion, die sich in der Nase ja kaum verhindern lässt, viel heftiger als das normale Gewebe reagieren und dass z. B. durch eine Probeexzision

phlegmonöse Entzündung und Vereiterung des ganzen Tumors hervorgerufen werden kann, mit den gefährlichsten Folgeerscheinungen oder mindestens auffallender Wachstumsanregung. Wie ich schon bei den Lymphdrüsen erwähnt habe, ist dann das Meiste an dieser oft enormen Schwellung entzündliches Infiltrat. Aber dieses Infiltrat erstreckt sich rasch in die Umgebung und verhindert dann oft die sichere Abgrenzung des Neoplasmas bei der Operation, abgesehen von der Gefahr der Wundinfektion durch den infizierten Tumor. Wir sind daher auf dem Standpunkt angelangt, die Diagnose maligner Tumoren nach Möglichkeit *klinisch* zu stellen und die Probeexzision nur in zweifelhaften Fällen auszuführen. Wenn sie aber ausgeführt werden soll, so geschehe sie unter peinlichster Asepsis und möglichster Desinfektion der Tumoroberfläche, event. nach vorheriger Salvarsanbehandlung (nach Gerber) durch einige Tage, was die Nekrobiosen und Mischinfektionen günstig beeinflussen soll.

ad 5. Berechtigtes Aufsehen hat vor einigen Jahren die Mitteilung von Freund und Kaminer in der Wiener Gesellschaft der Aerzte über Beeinflussung von Karzinomzellen durch Serum erregt. Die beiden Forscher sind auch derzeit noch unermüdlich am Werke, ihre Entdeckung weiter zu verfolgen und neue Reaktionen zu gewinnen. Ich verweise bezüglich der Details auf die entsprechenden Berichte. Auch wir haben uns auf diese Mitteilung von experimentellen Erfolgen begeistert an der Sammlung von Belegmaterial am Lebenden beteiligt; die Fälle der Klinik Chiari erschienen in einer Arbeit von Arzt und Kerl im Jahre 1912 aus der Dermatologischen Klinik zu Wien. Sie fanden (im allgemeinen) unter 53 Fällen von nachgewiesenem Karzinom 44 mal eine richtige Diagnose (83 %) und 9 mal (17 %) eine Fehldiagnose. In späteren Statistiken scheint sich der Prozentsatz durch Ausschaltung von Fehlerquellen noch vergrössert zu haben. Doch kann die Reaktion auch bei diversen anderen Zuständen eintreten, bedarf daher anscheinend noch genauer Differenzierung. Eine andere ebenfalls erst vor kurzem angegebene Methode — die der Wassermann'schen Reaktion auf Syphilis analog ist — ist die Komplementbindungs-Reaktion zur Diagnose maligner Geschwülste nach von Dungern. Beide Methoden haben gewiss eine grosse Zukunft, wenn es gelingen sollte sie noch eindeutiger zu gestalten, sie werden aber nie imstande sein, die anderen, vor allem die älteren, längst geübten Untersuchungsmethoden entbehrlich zu machen. Immer mehr kommt die Medizin trotz aller sensationellen Entdeckungen auf dem Gebiete exakter Untersuchungsmethoden auf den gewiss einzig richtigen Standpunkt zurück, eine exakte eindeutige Reaktion, wie in der Chemie, gibt es nicht. Eine exakte Untersuchung und Diagnose aber ist nur möglich durch echt klinische Verwertung des mit allen uns zu Gebote stehenden Mitteln gefundenen symptomatischen Materials unter der ordnenden Leitung eines erfahrenen medizinisch denkenden Geistes.

Nicht unerwähnt sei hier noch eine diagnostische Methode der Kieferhöhlenerkrankung, die von Mink 1909 angegeben worden ist, die sogenannte Auskultation der Kieferhöhle. Mittels eines Ohrtrichters,

der mit einem in die Ohren des Untersuchers geleiteten Gummischlauches verbunden ist und an die Fossa canina angesetzt wird, hört man bei lufthältigen Kieferhöhlen ein amphorisches Geräusch, welches verschwindet, wenn sie luftleer sind, auch durch Störung der Luftpassage der untersuchten Seite. Das Geräusch wird bei Schleimhautverdickung der Höhle abgeschwächt, auch wenn sie luftleer ist. Durch entsprechende Neigung des Kopfes kann man auch Flüssigkeitsgeräusch an der natürlichen Oeffnung erzeugen. Tumoren heben das Geräusch ebenfalls auf.

Ich werde hier nicht die jedem Mediziner bekannten diagnostischen Merkmale des klassischen Oberkiefertumors schildern, oder das Symptomenbild des ausgebildeten Nasentumors; auch bezüglich der Differenzialdiagnose zwischen malignem Tumor und anderen Erkrankungen, sowie zwischen Sarkom und Karzinom kann ich mich auf das vorhingesagte und auf die schon zitierten Lehr- und Handbücher und Sammelarbeiten berufen. Eingehend haben sich auch Harmer und Glas, meine Vorgänger an der Wiener Klinik, in ihrer hier viel zitierten Arbeit mit der Differenzialdiagnose zwischen malignem Tumor und den verschiedenen Formen einfacher chronischer Periostitis und Ostitis, Tuberkulose und Syphilis, Aktinomykose, Sklerom, Zahncysten und anderen gutartigen Tumoren, den verschiedenen Ursachen des Exophthalmus etc. beschäftigt. Auch Hydrops der Nebenhöhlen und einfache Höhleneiterungen (geschlossene Empyeme) können ganz unter dem Bilde eines malignen langsam und reaktionslos wachsenden Tumors verlaufen. Probatorische Eröffnung der Nase oder der Nebenhöhlen ist daher mangels einer Probeexzision mit positivem Befund mindestens dann unbedingt indiziert, wenn man einen Fall als inoperabel erklären will, und auch von Kümmel, Harmer, Citelli, Sendziak und vielen anderen gefordert worden. Citelli hat besonders die Diagnostik der Nebenhöhlen-Tumoren (Keilbeinhöhle) behandelt.

Anhangsweise möchte ich noch kurz auf einige Tumoren der Nase und der Nebenhöhlen zu sprechen kommen, die eigentlich nicht in mein Thema einschlagen. Es sind das zunächst die *sekundären Tumoren*, welche erst durch Einwachsen in die Nebenhöhlen oder die Nase zu ihnen in Beziehung treten und dann schliesslich auch dieselben Symptome, wie die primären Tumoren machen können. Zu ihnen gehören namentlich die Oberkiefertumoren, welche vom Processus alveolaris, Zahnfleisch und Gaumen ausgehen, dann diejenigen, welche vom Periost der Fossa canina oder der Fissura temporalis inferior oder pterygopalatina ihren Ursprung nehmen. Die ersteren bieten keine diagnostischen Schwierigkeiten, mehr die letzteren. Sie machen frühzeitig Affektion der Trigeminus-Aeste, dann der Augenmuskelnerven, schliesslich des Optikus. Zur Affektion der Trigeminus-Aeste hat Killian einen interessanten differenzialdiagnostischen Beitrag geliefert. Er folgert also: Wird der Nerv vom Tumor ergriffen und zerstört, so muss notwendig die Sensibilität des periphären Stumpfes aufgehoben sein, während die ungestörte Nervenleitung des zentralen Stumpfes Empfindungen auslösen kann. Wir

können also mit einiger Bestimmtheit Schädigungen eines Trigeminus-Astes bei gleichzeitigem Auftreten von Neuralgien diagnostisieren, weiter die Nasenrachenraumtumoren, die noch Gegenstand meiner Abhandlung sein werden, und endlich die Tumoren der Schädelbasis und die endokraniellen Geschwülste, welche die Schädelbasis nach unten durchbrochen haben. Von den ersteren (Schädelbasistumoren) hat Flesch 1909 2 interessante Fälle publiziert, wo es in für diese Tumoren charakteristischer Weise frühzeitig durch infiltratives Wachstum zur succesiven Lähmung verschiedener Hirnnerven kam, welche die Diagnose früher ermöglichte, als der Tumor im Nasenrachenraum sichtbar wurde. Die häufig beobachtete Affektion von oberen Zervikalnerven hat heftige Anfälle von Hinterkopfschmerzen zu Folge. Von endokraniellen Tumoren hat unter anderen Delsaux berichtet (Cancer encéphaloïde, in Stirnhöhle Siebbein und Kieferhöhle eingebrochen). Wir selbst haben an der Klinik Chiari ein Endotheliom der Dura beobachtet, welches einerseits das Stirnbein durchwuchert und schliesslich beide Stirnhöhlen ausgefüllt hatte, anderseits in den Sinus longitudinalis eingebrochen war und sich metastatisch an seinem hinteren Ende abgelagert hatte: Die Radikaloperation war von einem Hirnabscess gefolgt, welcher zwar erkannt und entleert wurde, die Patientin starb aber an der endokraniellen Ausbreitung des Neoplasmas. Als zweite Gruppe der ausserhalb des Themas fallenden Tumoren habe ich kurz der malignen Tumoren der äusseren Nase zu erwähnen, die, wie schon zu Anfang angedeutet, in manchen Fällen die Nasenwand durchbrechen und in alle Nebenhöhlen einwuchern können. Bei ihnen handelt es sich dann meist um den Typus des Carcinoma basocellulare (nach Lenard und Donogany), während das verhornende Plattenepitheliom oder Kankroid sich durch enorm chronischen Verlauf (Ploeger und Lehnhoff haben von Fällen berichtet, die 28–30 Jahre gedauert hatten), und daher besondere Gutartigkeit auszeichnet, auch jeder Art von Behandlung leicht zugänglich ist. Morestin, Kraus, Jones, Katz, Polyák haben über derartige Geschwülste berichtet, welche nach und nach in die Nase und sämtliche Nebenhöhlen eingebrochen waren und dann auch in ihrem weiteren Verlauf, was die Raschheit der Rezidive und die übrigen Erscheinungen, Komplikationen etc. anbelangt, sich kaum von den primären Karzinomen der inneren Nase unterscheiden. Auch ich habe über einen in der letzten Zeit an der Klinik Chiari zur Beobachtung gekommenen ähnlichen Fall zu berichten. Fallers beobachtete ein Kankroid der äusseren Nase mit starker Verhornung, bei einer 61 jährigen Frau im Anschluss an die Entfernung einer Warze, das schliesslich in alle Nebenhöhlen eingebrochen war und auch den Gaumen ergriffen hatte. Bei der Operation wurden käsige cholesteatomartige Massen in Unmenge aus der Nase und dem Tränensack entfernt, die durch die hochgradige Verhornung des Tumors erzeugt worden war. Darier veröffentlichte zwei Fälle von Spindelzellensarkom der äusseren Nase in Form von harten Infiltraten der Haut und des Unterhautzellgewebes, welche lange Zeit Rhinosklerom vortäuschten.

Ueber Endotheliome der äusseren Nase hat Hinsberg gearbeitet, welcher unter 97 Tumoren des Gesichtes 13 Endotheliome, meist an der äusseren Nase und den Augenlidern lokalisiert, zusammenstellt, und Theodore, welcher 4 eigene und 9 Literaturfälle von diesen Tumoren an der äusseren Nase gesammelt hat. Seine Anschauungen über die histologische Natur derselben differieren etwas von denen der bei Besprechung der endonasalen Tumoren zitierten Autoren. Hervorzuheben wäre die Folgerung seiner Erfahrung, dass die Endotheliome des Gesichts im Gegensatz zu den Karzinomen dort vorkommen, wo Haut den Knochen überzieht, aber nicht an den Uebergängen von Haut und Schleimhaut.

Ferner möchte ich darauf hinweisen, dass auch histologisch benigne Neubildungen malignen Charakter annehmen können und besonders das Myxom erwähnen, welches als reine Form sehr selten in unserem Gebiete ist und sich nach den Berichten aller Autoren geradezu durch unerhörte Malignität auszeichnen. Chiari, Hajek, Virchow, Butlin, Brault, Walker Downie, in anderen Organen Simon und Ruffin haben solche Fälle gesehen. Ein Fall von Botey verlief innerhalb ganz kurzer Zeit unter den schwersten Erscheinungen, Blutungen, Zerstörung der Nebenhöhlen und des Keilbeins, rasche Rezidive, Erblindung, Einbruch in die Schädelhöhle, Meningitis. Botey behauptet geradezu, dass das reine Myxom noch viel maligner sei als das Myxosarkom.

Endlich dürfen die interessanten und auch nicht häufigen Papillome der inneren Nase nicht unerwähnt bleiben, die auch wieder Billroth als einer der ersten beschrieben hat (Zottenkrebs), und über deren benigne oder maligne Natur man lange nicht im Klaren war. Der klassische Bearbeiter der Nasenpapillome war Hopmann, der drei Typen unterscheidet: Epithelioma papillare, Papilloma molle und Papilloma durum. Bei Hanemann (1912) findet sich die Literatur-Uebersicht. Herxheimer und Brock haben in neuester Zeit die Frage der histologischen Einteilung und Pathogenese noch einmal studiert. Brock befürwortet die Einteilung in zwei Gruppen: 1. Tumoren, die ihren Sitz am Eingangsteil der Nase und in nächster Nähe der Schleimhautepidermisgrenze haben; 2. solche, die von der Tiefe der Nase ihren Ausgang nehmen aus der Gegend der mittleren Muschel und des Siebbeins und sich über die ganze Nase verbreiten können. Weitere Berichte der letzten Zeit stammen von Marbaix und Gonzalez, auch an der Klinik Chiari wurden zwei ähnliche Geschwülste beobachtet. Lewy und Herxheimer haben speziell über das Papilloma durum geschrieben; und letzterer kommt zum Schluss, dass diese Geschwulst im Gegensatz zu den weichen Papillomen zwar mikroskopisch gutartig, klinisch aber durchaus bösartig sei, was schon die beiden Billroth'schen Fälle zeigen. Der Fall von Kiesselbach-Hellmann und der von Poli, ersterer erst nach 12 Jahren, wurden tatsächlich später echte Karzinome. Auch bei mikroskopisch benignem Befund wuchern sie ähnlich den Myxomen schrankenlos, mit Zerstörungen des umgebenden Knochen weiter, eventuell bis ins Gehirn.

II. Die malignen Tumoren des Nasenrachenraums

Die Wissenschaft von den malignen Tumoren des Nasenrachenraums ist noch jung. Vor 15 Jahren wurden sie noch kaum beachtet. Mikulicz tut sie im *Heymann'schen Handbuch* bei den Rachentumoren noch recht kurz ab. Ihre Unzugänglichkeit, sowohl für die Untersuchung als auch Diagnose und Therapie und die fast absolute Aussichtslosigkeit jedes therapeutischen Bemühens verdross die Aerzte; sie gingen ihnen aus dem Wege. Ausserdem sind sie noch bedeutend seltener als die Nasentumoren; viele von den beschriebenen Fällen betreffen sekundär aus der Umgebung in den Nasenrachenraum eingewucherte Geschwülste. Die Seltenheit wird auch illustriert durch den Vergleich mit den Rachentumoren der verschiedenen Kliniken, mit denen sie namentlich früher meist zusammenpubliziert wurden. So fanden sich unter 47 Pharynxkarzinomen Czernys nur zwei Nasenrachentumoren, unter 47 Fällen Gussenbauers, publiziert von Meller, nur ein Fall von Nasenrachenkarzinom. Schumacher verzeichnet unter 136 Fällen von Pharynxkarzinomen nur zwei Nasenrachenkarzinome. Einer Statistik der Klinik Chiari von ca. 70 Nasentumoren stehen kaum 30 solche des Nasenrachenraums gegenüber. Die Hauptwerke über dieses Gebiet verdanken wir Soucy, Laval, Pauliac, Aboulker, Citelli, Trotter, Sendziak und Oppikofer, welch letzterer in dem kürzlich erschienenen dritten Heft des diesjährigen Bandes des *Fränkel-schen Archiv* auf Grund von 21 eigenen Fällen (aus der Siebenmann'schen Klinik) ein 'Standard Work' geschaffen hat, welches durch ein ungemein reichhaltiges Literaturverzeichnis erhöhte Bedeutung gewinnt. Von den Sammelstatistiken erwähne ich die von Pauliak (nur Sarkome) mit 78 Literatur- und 28 eigenen Fällen, Sendziak mit 181 Fällen, darunter 9 eigenen, und endlich die grösste von Oppikofer mit 250 Literatur- und 21 eigenen Fällen.[1]

Die Tumoren des Nasenrachenraums haben in mehrfacher Hinsicht eine Sonderstellung. Der Nasenrachenraum hat noch mehr als die Nasenhöhle vielfache Beziehungen zu seiner Umgebung (Septum, Nase, Keilbeinhöhle, Gehörorgan) und es ist bei seinen Neubildungen noch schwieriger als bei den Nasentumoren primäre von sekundären zu trennen, aber auch durch das Vorhandensein der Rachentonsille mit der Hypophysis pharyngis (Citelli) wird die Pathologie des Nasenrachenraums weiter kompliziert. Oppikofer beklagt vor allem gegenüber der grossen Zahl von Einzelbeobachtungen das Fehlen von solchen an grösserem einheitlichen Material, von denen allein aus sich sichere Anhaltspunkte für die wichtigsten Fragen dieses Gebietes gewinnen lassen; ferner den Mangel zahlreicherer Beobachtungen bis zum Ende und von Sektionsbefunden. Nach Citelli gehören *nicht* hierher die Neoplasmen der Nase und der Nebenhöhlen, der Fossa pterygopalatina, der Schädelgrube und angren-

[1] Hiezu noch neueste Beobachtungen von Ch. Mayo (2 Fälle), Menzel, Milligan, Porter, Molinié und Dupeyrac, Olkhine, Patterson, Stuart Low, Thyne und Fraser, Vorschütz, Walker Downie.

zenden Regionen, die Lymphosarkome der Zervikaldrüsen, welche nur sekundär in den Nasenrachenraum hereinwuchern, noch auch das sogenannte maligne Lymphom und die Pseudoleukämie, ebenso wenig die Fibrosarkome oder Sarkome, die erst infolge maligner Degeneration, vor allem der sogenannten harten Fibrome des Nasenrachenraums sich bilden. Solcher Art umgrenzt, seien die primären malignen Nasenrachenraumgeschwülste recht selten.

Ich kann mich mit Beziehung auf das in der ersten Abteilung bereits gesagte hier wesentlich kürzer fassen und will nur eingehender bei den Eigentümlichkeiten dieser Tumoren verweilen. Halten wir an der Einteilung der malignen Tumoren in Sarkome, Karzinome und Endotheliome fest, so sehen wir in der Sendziak'schen Statistik die Sarkome mit 88, die Karzinome mit 64 und die Endotheliome mit 29 Fällen vertreten. Zu ähnlichen Verhältniszahlen kommen auch die anderen Autoren, also im allgemeinen das Sarkom entschieden häufiger als das Karzinom. Dies liegt vor allem darin, dass hier das Lymphosarkom, in der inneren Nase kaum beobachtet, hinzukommt ; es geht meist von der Tonsilla pharyngea aus. Citelli, der besonders die Physiologie der Pharynxtonsille und der Hypophysis pharyngis studiert hat, bringt geradezu die Entstehung des Lymphosarkoms mit den Erscheinungen der Involution oder des Vorhandenseins der Pharynxtonsille in Verbindung. So waren unter den 21 Fällen Oppikofers 6 Lymphosarkome und 5 Rundzellensarkome. Citelli beobachtete unter 4 Sarkomfällen 3 Lymphosarkome. Ich selbst beobachtete seit 1907 an der Klinik Chiari unter 8 Nasenrachentumoren 5 Lymphosarkome. Doch sind auch verschiedene andere Formen von Sarkomen beobachtet worden, darunter das Rundzellensarkom am häufigsten, dann Spindelzellensarkom, Fibrosarkom (Milligan), Angiosarkom (Sendziak), teleangiektatisches Fibromyxosarkom (Botey), polymorphes Sarkom (Citelli) etc. Nager hat einen Fall von Rundzellensarkom bei einer 75 jährigen Frau auf pseudoleukämischer Grundlage berichtet, wie der typische Blutbefund gezeigt hat. Unter den Karzinomen herrscht die Form des Carcinoma simplex, von Deckepithel ausgehend, vor, seltener sind Adenokarzinome. Ziemlich häufig ist auch das Plattenepithelkarzinom. Ein (metastatisches ?) Hypernephrom beobachtete Preysing.

Auffallend häufig scheinen die Endotheliome zu sein (S. Sendziak) nach Trotter, der allerdings besonders viel Fälle (6) beobachtet hat, sogar die häufigsten Geschwülste des Nasenrachenraums. Oppikofer beobachtete 3 Fälle, Tilley 7. Umwandlung des histologischen Baues beobachtete Guthrie : Plattenepithelkarzinom bei einem 17 jährigen Mädchen, welches später den Bau eines Medullarkarzinoms zeigte, und Hopmann : Karzinom des Nasenrachenraums bei einem 18 jährigen Mann ; Probeexzision ergibt Nasenrachenfibrom. Während der Operation unter foudroyanter Blutung Exitus. Histologische Untersuchung des exstirpierten Tumors ergibt nun Karzinom. Botey : reines Myxom verwandelt sich in Myxosarkom und wieder zurück in Myxom. Blumenfeld hat in der Diskussion zu der Demonstration Hopmanns

neuerdings auf die gerade bei den Tumoren der Nase ausserordentlich häufig vorkommenden irreleitenden Schlussfolgerungen bei der Stückchendiagnose hingewiesen, welche letztere nur in Verbindung mit den klinischen Symptomen zu verwerten sei. Die Sarkome und Karzinome unterscheiden sich auch hier untereinander durch die mehr Tumor bildende Art der Sarkome und das ulzerierende destruktive Wachstum der Karzinome, doch verlief gerade in dem im allgemeinen etwas abnormen Fall Guthries das Karzinom ganz einem Sarkom ähnlich tumorbildend. Die Adenokarzinome sollen nach Citelli später als die anderen ulzerieren. Aetiologisch werden akute Entzündungen (Influenza bei Powell), mehrfach Traumen (Entfernung der Rachenmandel, Gantvoort, Clerc) angegeben. Ausgangspunkt der malignen Tumoren ist in der Regel die Rachenhinterwand und Seitenwand, selten das hintere Septumende (Labourré, Mayo, auch ein Fall vom Referenten beobachtet). Ueber die Bevorzugung von Hinter- oder Seitenwand sind die Meinungen geteilt ; die von verschiedenen Autoren gemachte Angabe, dass das Karzinom meist vom Rachendach und das Sarkom von der Seitenwand ausgeht, lassen sich nach Oppikofers Literaturstudium und eigener Erfahrung nicht als Regel aufstellen. Für die Sarkome gibt Pauliac als gewöhnlichen Ausgangspunkt die Rachentonsille oder die Schädelbasis an ; die Karzinome gehen nach Citelli vom Deckepithel, von den Drüsenschläuchen, in selteneren Fällen auch von Elementen aus, welche die Pharynxhypophyse zusammensetzen oder von anderen epithelialen Elementen des primären Hypophysenschenkels.

Was das *Geschlecht* anbelangt, so überwiegen auch hier die Männer.

Das *Alter* variiert zwischen 4 (Scheinmann, Paige) und 84 Jahren (Patterson), die kindlichen Fälle natürlich nur beim Sarkom beobachtet. Das Alter betrug weiters bei einem Fall von Senator 5 Jahre, Mager 11, Billington 13, Fein 16, doch sind auch sehr jugendliche Karzinome beobachtet worden, so von Oppikofer mit 13 und 20 Jahren, Rugani und Guthrie 17, Hopmann mit 18 Jahren. Trotter hebt das jugendliche Alter (18–35 Jahren) der Endotheliome hervor, Polyák gibt das Alter für Nasenrachensarkome im allgemeinen zwischen 20 und 40 Jahren an.

Symptome. — Bei den malignen Nasenrachentumoren ist noch mehr als bei den Nasentumoren die Latenzperiode ausgeprägt, nach Citelli länger von Karzinom als beim Sarkom. Pauliak unterscheidet eine funktionelle und organische Latenz. Die Verlegung der Nasenatmung nimmt, was die Zeit des Eintrittes anbelangt, im Gegensatz zu den Nasentumoren durchaus nicht den ersten Platz ein. Insbesondere bei Männern mit gut ausgebildetem Nasenrachenraum kann der Tumor schon eine beträchtliche Grösse erreicht haben, bevor die eine oder die andere Nasenseite sich so verlegt, dass die Patienten zum Arzt kommen, dagegen scheint es hier öfters und früher als bei den Nasentumoren zu heftigen Blutungen zu kommen ; sie stammt nach Citelli von den ulzerierten oder verletzten Gefässen des Tumors oder von geplatzten Gefässen der hyperämischen Schleimhaut der Umgebung.

Das Symptom aber, das besondere differential-diagnostische Bedeutung beansprucht, ist die *Gehörsstörung*. Sie kann entweder, wie ich schon bei den Nasentumoren erörtert habe, durch Verschluss der Tube infolge von Druck des benachbarten Tumors oder Ausfüllung derselben mit Geschwulstmasse oder Einwucherung von Neoplasma in die Paukenhöhle oder das innere Ohr, oder endlich, dieses aber nur in spätem Stadium und mehr bei den sekundären Tumoren, durch Affektion des Akustikus bedingt sein. Der Tumor, besonders das Sarkom, wuchert manchmal mit Schonung der Tubenschleimhaut bis in die Paukenhöhle in Form eines dem Knochen entlang folgenden Schlauches und kann sogar im äusseren Gehörgang erscheinen (Escat, Clerc). Die Gehörsstörung ist nach Sendziak das wichtigste Symptom, fast ausnahmslos (in Oppikofers 11 Fällen nur zweimal nicht, da der Tumor am Rachendach sass) und immer frühzeitig vorhanden. Pauliac und Oppikofer haben sich mit ihr eingehend beschäftigt. Einziehung des Trommelfells folgt, Parazentese ist *quoad* Gehör erfolglos, und kann schon da sein wo der postrhinoskopische Befund noch normal ist. Die durch den Nasenrachentumor erzeugte Otitis media ist durch ihren eigenartigen torpiden und fieberlosen Verlauf gekennzeichnet.

Ein weiteres Symptom, welches hier im Gegensatz zu den Nasentumoren hinzutritt, ist der Husten, wie die Beobachtungen von Königsberg und Lugani dartun, ein echter Reflexhusten von der Tube oder der seitlichen Rachenwand ausgelöst.

Die Schmerzen treten relativ spät auf, besonders spät beim Lymphosarkom. Sie sind zuerst auf einzelne Gebiete des Trigeminus lokalisiert oder bestehen in Hinterhaupt- oder Nackenschmerzen. Die Nachbarschaft verschiedener Hirnnerven bringt es mit sich, dass manchmal diese Neuralgien einzelne Trigeminusäste früh eintreten, wenn man noch nichts im Nasenrachenraum sieht. Ich habe hier auf einen interessanten Symptomenkomplex aufmerksam zu machen, die Trotter'sche Trias, den auch Tilley beobachtet hat. Er wurde von Trotter für die Tumoren des vorderen Tubenrandes als pathognomonisch erkannt (für die Endotheliome soll dies die typische Lokalisation sein) und besteht in Taubheit, Neuralgie im Unterkiefer und Zunge, im Verein mit Anästhesie dieser Gebiete und einseitiger Gaumensegelaffektion. Die Neuralgien sind bedingt durch die Affektion des dritten Trigeminusastes, der an dieser Stelle nahe der Innenwand des Nasenrachenraums verläuft und daher frühzeitig vom Tumor in Mitleidenschaft gezogen wird. Die einseitige Gaumensegelaffektion entsteht durch direkte Einhüllung des Levator palati durch Neoplasma, was eine Veränderung in der Ausdehnbarkeit des Muskels verursacht, so dass während der Segelhebung der Gaumen symmetrisch erscheint, da sich der Muskel noch zusammenziehen kann, während er in Ruhe asymmetrisch ist, weil der Muskel nicht nachlassen kann. Im weiteren Verlauf kann sich ein Oedem des Gaumens der erkrankten Seite entwickeln, welches dann auch eine mangelhafte Hebung des Segels bewirken kann.

Alle diese Symptome, leichte Behinderung der Nasenatmung oder auch Fehlen derselben, Blutungen, Gehörsstörung, Ohrensausen, Fremdkörpergefühl hinter dem weichen Gaumen, fehlende oder wenig konstante Schmerzen im Nacken mit Ausstrahlung in die Wirbelsäule oder in die Ohren, Augen und Gesicht, manchmal stärkere Reaktion der benachbarten Nasenschleimhaut in der Art eines akuten Schnupfens (Reizschnupfen) subsumiert Pauliac unter die Latenzperiode, deren organisches Substrat nur durch die hintere Rhinoskopie oder durch die Digitaluntersuchung festgestellt werden kann. Im weiteren Verlauf nehmen dann die Schmerzen an Intensität und Ausbreitung zu, zu den Neuralgien einzelner Trigeminusäste gesellen sich solche der anderen durch Ueberspringen des sensiblen Reizes (Oppikofer) oder durch Vordringen des Neoplasmas bis zum Ganglion Gasseri (Trotter meint, dass viele als primäre Tumoren des Ganglion Gasseri beschriebenen Fälle nichts anderes seien als bis dorthin vorgedrungene Nasenrachenraumtumoren), ferner Affektion anderer sensibler Nerven (Zervikalnerven), Affektionen der anderen Seite und diffuse Hinterkopf- und Nackenschmerzen, die dem Patienten die Nachtruhe rauben und nicht einmal durch hohe Morphiumdosen zu bekämpfen sind. Diese Zustände gehören zu den qualvollsten, die wir kennen. Völlige Nasenverstopfung mit Anosmie, Taubheit der anderen Seite, diffuse und intensive Schmerzen, starke Blutungen, Sprachstörung, Schluckbeschwerden, endlich Symptome der Infektion der Nebenhöhlen und des Mittelohrs sind die Anzeichen, dass der Tumor in die Pauliac'sche manifeste Periode eingetreten ist. Es kommt jetzt nach und nach zu den Affektionen der anderen Hirnnerven, verschiedenen Lähmungen etc., bezüglich deren ich auf die interessanten Ausführungen Oppikofers verweise. Er findet unter anderem auffallend, dass bei Vagusaffektion niemals Reizungssymptome beobachtet wurden, auch fehlt bei Lähmung der Ausfall von Erscheinungen von seiten des Herzens und Magendarmtrakts, was sich wohl nach meiner Ansicht mit der Einseitigkeit erklärt. Doppelseitige Postikusparalyse sei noch nie beobachtet, die Augenmuskeln werden relativ spät gelähmt, erst nach Durchbruch des Tumors durch die Sella und in den Sinus cavernosus, oder durch Einwachsen in die Fissura orbitalis superior und inferior ; dann kommt es auch zur Protrusio bulbi und Erscheinungen von seiten des Optikus (plötzliche Erblindung). Auch bei Oppikofer fand ich eine Bestätigung meiner Erfahrung, dass die Nerven anscheinend den malignen Neoplasmen grösseren Widerstand leisten als andere Gewebe, und kann hier nur auf den ersten Teil verweisen, wo ich die Ansicht ausgesprochen habe, dass die Ausfallerscheinungen und die Schmerzen in den einzelnen Hirnnervengebieten längere Zeit nur durch Druck oder Oedem, aber nicht durch organische Zerstörung bedingt sei. Wird die Ursache gehoben, so kann sich auch wieder die Funktion einstellen. Einen Beweis hierfür bildet ein in letzterer Zeit auf der Klinik Chiari beobachteter Fall von einem Nasenrachenkarzinom, das schon vor 6 Monaten Ophthalmoplegie und Affektion des Trigeminus und anderer Hirnnerven erzeugt hatte. Eine energische Radiumbehandlung

hat nun vor kurzem die meisten dieser Ausfallserscheinungen zum Schwinden gebracht und auch die Schmerzen behoben.

Ankylostoma, hier meist erzeugt durch die Affektion der Mm. pterygoidei, ist ein weiteres Symptom der Vorgeschrittenheit des Tumors. Endlich kommt es durch Affektion der Kopfhalsgelenke und der Halswirbelsäule zur Steifigkeit von Kopf und Hals, ja nach Oppikofer kann sogar das eigene Gewicht des Kopfes bei vertikaler Stellung genügen, um heftige Schmerzen auszulösen, besonders wenn der dem Rachendach entsprechende Teil der Schädelbasis durch die Tumormassen bis an die Dura heran vollständig durchwachsen und erweicht ist. Das bei den Siebbeintumoren so häufige Einwachsen in die Schädelhöhle wird hier selten beobachtet, infolge des Widerstandes des dicken Keilbeinkörpers und der Ausweichmöglichkeit des Tumorswachstums in die Nase, nach unten und in die seitlichen Fissuren der Gesichts- und Schädelknochen.

Die bei den Nasentumoren von manchen geleugneten im allgemeinen aber recht selten und spät auftretenden *Lymphdrüsenmetastasen* kommen hier trotz einiger widersprechender Angaben meist frühzeitig zur Beobachtung. Manchmal (auch wir haben derartige Fälle beobachtet) bilden sie das erste überhaupt am Kranken nachweisbare Symptom. Ueber die Verteilung und den Abfluss des Lymphstroms habe ich schon im ersten Teil meines Referats gesprochen. Infiziert werden vom Nasenrachenraum immer die retropharyngealen und die tiefen zervikalen Drüsen, aber nur letztere werden in der Regel beobachtet. Die ersteren verraten sich dem aufmerksamen Beobachter meist durch Druckerscheinungen auf die Hirnnerven, welche in der Reihenfolge etwas von der für die primären Tumoren typischen differieren. Charakteristisch für diese Drüsentumoren ist auch ihre Grösse. Sie sind bald in Form eines grossen, oft halb faustgrossen harten frühzeitig mit dem M. sternocleidomastoideus verwachsenen und immobilisierten Packets an beiden seitlichen Halspartien. Unter den einzelnen Arten hat besonderes das Lymphosarkom Tendenz zur frühzeitigen Bildung grosser Drüsentumoren.

Fernmetastasen sind, wenn auch selten, doch vielleicht etwas mehr als bei den Nasentumoren beobachtet. Oppikofer erwähnt einige in seinen Fällen, ähnliche Berichte stammen von Benda, Verneuil, Wassermann, Schech.

Auch die bei den Nasen- und Nebenhöhlentumoren so häufige Meningitis gehört hier zu den Seltenheiten. Von Oppikofers Fällen wurde sie in keinem Falle beobachtet. Sicher hängt dies mit der Richtung des Windstroms zusammen, der hier nur nach abwärts geht. Auch das Sensorium ist bis zuletzt ungetrübt, der Exitus letalis erfolgt hier meist durch Kachexie, selten durch Komplikationen (Verblutungen, Hirnnervenlähmung, Meningitis, Hirntumor). Diese sogar schon relativ früh einsetzende Kachexie ist zum Teil verschuldet durch die schlaflosen Nächte und die ununterbrochenen Schmerzen, zum Teil durch die bald auftretenden Schluckbeschwerden, Verschlucken und Schlucklähmung. Der hier geschilderte Verlauf variiert leider nur sehr selten und es ist angesichts dieser furchtbaren Prognose nur ein Glück, dass diese Tumoren so selten sind.

Der *Verlauf* variiert nach Oppikofer von 2 Monaten bis 7 Jahren, wobei der Durchschnitt dem ersten Extrem jedenfalls viel näher liegt. Koschier hat zwei solche Raritäten zu nennenden Fälle publiziert, von denen der erste, ein Karzinom des Nasenrachenraums bei einer 28 jährigen Frau, $2\frac{1}{2}$ Jahre nach einer jedenfalls unvollkommenen Auskratzung beschwerdefrei blieb und erst 3 Jahre nach der Palliativoperation an dem Tumor zugrunde ging. Noch merkwürdiger verlief der zweite Fall: 42 jährige Frau, Karzinom des Nasenrachenraums seit 1 Jahr, histologisch sichergestellt, Operation verweigert. Die Patientin kam nun im Laufe von *neun* Jahren wiederholt zu Koschier, welcher stets durch neuerliche Probeexzision den histologischen Befund bestätigen konnte. Erst in letzter Zeit war der Tumor sehr ausgebreitet, hatte beide Nasen ausgefüllt, das eine Auge und die Nasenbeine vorgetrieben und profuse Blutungen hervorgerufen. Aber auch in der Zeit der Veröffentlichung bestand noch keine Kachexie, keine Drüsen, nur hin und wieder etwas Kopfschmerzen, ein Verlauf, der dem des Sonnenschein'schen Falles ähnelt. Der Tumor ähnelt etwas den Mäusekarzinomen, welche keine Metastasen machen und die Umgebung weniger zerstören als sie verdrängen. Koschier tut angesichts dieses abnormen Falles den bedeutsamen Ausspruch: Jeder Operateur wäre stolz, einen solchen Fall acht Jahre nach einem operativen Eingriff noch lebend vorstellen zu können. Aehnlichen gutartigen Verlauf zeigten ein Fall von Nager und einer von Patterson.

Zum Kapitel *Diagnose* habe ich vor allem auf den ersten Teil zu verweisen und möchte auch hier in Uebereinstimmung mit den anderen Autoren, die über grössere eigene Verfahrung verfügen wie Sendziak, Citelli, Pauliac, Oppikofer etc. die Wichtigkeit einer exakten und gewissenhaften Untersuchung betonen, da die Tumoren, namentlich wenn man das Glück hat, sie im Beginn zu Gesicht zu bekommen, frappant unscheinbar aussehen und sehr leicht bei leichtfertiger Untersuchung übersehen werden können. Ich halte es nicht für überflüssig, auch hier noch einmal auf die neueren Untersuchungsinstrumente wie Salpingoskop und Pharyngoskop zu verweisen, ferner haben wir noch in den Instrumenten Yankauers und Gyergyais wertvolle Behelfe zur direkten Untersuchung, eventuell auch zur Probeexzision, namentlich an der seitlichen Nasenrachenwand. Weiters kommt hier noch die *Digitalexploration* hinzu. Obwohl man glauben sollte, dass das Ergebnis der Rhinopharyngoskopie dem der Palpation mit dem groben Finger weitaus überlegen sei, so haben doch erfahrene Rhinologen, die sicher mit der Inspektion das erreicht haben, was immer nur mit ihr zu erreichen war, sich der Digitaluntersuchung nicht begeben zu können vermeint und sie bei der Diagnostik der Nasenrachenraumtumoren stets dringend empfohlen. Die Palpation ergänzt nicht nur die Inspektion, sie kann gegebenenfalls berufen sein, sie zu ersetzen, d. h. die Diagnose zu machen, die die Inspektion nicht machen konnte, denn es ist erwiesen, dass viele maligne Nasenrachentumoren im Anfang lange Zeit unter der Schleimhautdecke in Form eines flachen kaum elevierten Infiltrats wachsen, bis sie endlich an die Oberfläche treten

und auch dem Auge Anhaltspunkte für das Vorhandensein eines Tumors geben. In diesen Fällen kann nun der tastende Finger an der scheinbar unveränderten Stelle durch Nachweis eines stärkeren Gewebspolsters über den Knochen oder einer Differenz in der Resistenz der Knochenwand und dergleichen zu einer Zeit die Diagnose machen, wo es vielleicht noch möglich ist, den Tumor operativ im Gesunden zu umgrenzen. Die Digitalexploration ist aber auch unentbehrlich zur exakten Diagnose und vor allem Indikationsstellung in allen Fällen wo der Tumor durch seine Grösse den Einblick sowohl von vorne als von hinten unmöglich macht. Sie hat namentlich, wenn schon Blutungen vorausgegangen sind, mit möglichster Vorsicht zu geschehen, eventuell nach Kokainisierung des weichen Gaumens, um stärkeres Würgen und profuse Blutung, welche manchmal dabei in bedenklicher Heftigkeit beobachtet wurde, zu vermeiden. Weiter hat bei den Nasenrachentumoren stets auch vor der Probeexzision die Blutuntersuchung zu erfolgen, um eine Leukämie oder Pseudoleukämie auszuschliessen. Die Differenzialdiagnose hat vornehmlich zwischen Neoplasma einerseits und Tuberkulose, Syphilis und gutartigen Tumoren (Angiofibrom) andererseits zu entscheiden. Tuberkulose kann manchmal gleich wie in der Nase (Chiari) grosse höckerige Tumoren machen, die den Sarkomen täuschend gleichen. Besonders die Tuberkulose der Rachentonsille lässt diese tumorartig anschwellen. Chronisch infiltrierende Lues kann Geschwüre mit wallartigem Rand und unregelmässigem, üppig granulierendem Grunde erzeugen, die mit Karzinom verwechselt werden. Endgültigen Aufschluss gibt hier natürlich nur die histologische Untersuchung, aber nur wenn sie positiv ist. Ich betone hier nochmals die Wichtigkeit mehrfacher Probeexzisionen, womöglich von verschiedenen Stellen des Tumors. Die Probeexzision ist nur kontraindiziert bei Angiofibromen, doch ist dieses Krankheitsbild so eindeutig beschrieben, dass wenigstens der Verdacht auf Angiofibrom leicht erregt ist und der vorsichtige Untersucher dann die Diagnose auf anderem Wege zu erreichen suchen wird. Wichtig ist hierbei das männliche Geschlecht, jugendliches Alter und Fehlen von Drüsenmetastasen. Was diese letzteren anbelangt, so ist es geraten, wenn eine Diagnose mit der Probeexzision aus dem Nasenrachenraum nicht zu erzielen ist, ein Lymphom oder ein Stück desselben zu exstirpieren. Nicht zu vergessen ist, dass grosse Lymphdrüsentumoren an typischer Stelle auch durch Pediculi capitis erzeugt werden können. Schwierig kann die histologische Diagnose auch bei Lymphosarkom, Pseudoleukämie, malignem Lymphom werden. Die Differenzialdiagnose zwischen Lues und Sarkom hat Pauliac genau ausgearbeitet.

Endlich kann aber die Probeexzision und alle anderen Untersuchungsmethoden versagen und die Diagnose bloss rein klinisch spekulativ auf Grund der eigenartigen Symptome von Hirnnervenaffektion eventuell mit Taubheit und Levatorerkrankung kombiniert möglich sein. Ich erinnere hier an die Fälle von Flesch, ferner an den von Mahu, wo Recurrenzlähmung das erste Symptom eines Schädelbasistumors bildete.

DISCUSSION

Dr. Luc (Paris). My only object in asking to take part in this discussion is to advocate the advantages of the use of local anæsthesia and ischæmia in our external operations for the radical cure of malignant neoplasms of the nasal cavity. Of these advantages I cannot give you better proof than by relating briefly the case of a diabetic patient of 65 years which is fully described in my report read to the *French Society of Oto-rhino-laryngology* (1911), on the application of local anaesthesia in our surgical operations.

The patient in question presented an epithelioma growing from the external wall of the right nasal fossa and extending into the frontal and maxillary sinuses. He gave no sign of suffering during the long operation, which was a combination of Jacques' frontal method, Moure's nasal method, and Caldwell-Luc's maxillary method. The cutaneous part of the operation went on thoroughly *à blanc* (bloodlessly), as on the dead body, and as regards the small quantity of blood that escaped during the intra-nasal, intra-frontal, and intra-maxillary part of the intervention, the patient helped by blowing or spitting it out. There was no sign of recurrence of the neoplasm after the lapse of more than a year.

I am conscious of having thus spared that patient the double danger of chloroform and of the penetration of blood into the air-passages, and I think that my procedure in this case may be offered to you as an example to be followed under similar circumstances.

Dr. Sargnon (Lyon). Nous avons eu jusqu'ici l'occasion d'observer neuf cas de néoplasmes malins des sinus, ayant envahi le sinus maxillaire, le sinus sphénoïdal et surtout l'ethmoïde et l'orbite. Tous ces cas ont été opérés sauf deux. La plupart de ces cas ont été publiés dans ma communication à la Soc. française de Laryngologie, mai 1911. Dans un cas, chez un homme âgé, avec suppuration ethmoïdale et exophtalmie, j'ai refusé d'intervenir, à cause de la faiblesse du malade et de son état pulmonaire. Mort trois mois après.

Nous avons observé en 1912–13 un malade atteint de cancer histologique des sinus ; il a refusé l'opération.

Nous avons observé deux cas de sarcomes du nez et des sinus ; l'un a été opéré par les voies naturelles sous cocaïne par curetage avant que la méthode naso-latérale de Moure ne soit adoptée ; le malade est mort dans le coma, trois mois après l'opération, par généralisation à la base du crâne.

Nous avons vu en 1909 son frère, âgé de 60 ans ; polypes récidivants du nez et des sinus paraissant malins, malgré des examens histologiques répétés qui disaient simplement polypes. Ablations répétées pendant deux ans par les voies naturelles : abcès orbito-interne, opération de

Moure avec nettoyage des sinus maxillaires ethmoïdaux, de la région sphénoïdale et de la région postérieure de la cloison. L'examen histologique montre alors quelques points sarcomateux. Récidive 5 mois après, du sphénoïde du toit nasal et sous-orbitaire ; réopération, récidives multiples. Mort ultérieure.

Homme, 50 ans. Pseudo-sinusite maxillaire droite supérieure ; la ponction du sinus ramène du sang. A l'examen histologique pas de cancer. Opération de Moure en 1910 qui montre un néoplasme malin envahissant le sinus maxillaire, la partie inférieure et interne de l'orbite et l'ethmoïde. L'examen histologique montre un épithélioma pavimenteux lobulaire ; deux mois après envahissement de l'œil et mort dans le coma.

En 1911, homme, 58 ans. Sinusite maxillaire avec douleurs intenses ; l'ablation par endoscopie directe par une fistule sus-alvéolaire montre de gros bourgeons dans le sinus, qui histologiquement sont cancéreux. Opération de Moure qui montre un néoplasme diffus du sinus maxillaire de l'ethmoïde de la paroi interne orbitaire et de la fosse ptérygo-maxillaire. Les douleurs atroces ont été très soulagées par l'opération ; récidive, ganglion parotidien du côté malade. Mort trois mois après.

Homme, 50 ans. Tumeur de la partie supérieure du sinus maxillaire, de la partie inférieure de l'orbite et de l'ethmoïde ; opération de Moure. A l'examen histologique sarcome globo-cellulaire et malignité locale. Opération en 1909 ; récidive orbito-ethmoïdale en 1911 ; le malade refuse l'intervention, il a fait de divers côtés des traitements variés non-opératoires ; radiothérapie ; il a actuellement une récidive intense avec cécité bilatérale.

Homme, 50 ans ; pseudo-sinusite cancéreuse envahissant le sinus maxillaire, l'ethmoïde et la partie interne de l'orbite. Opération naso-latérale par l'incision de Liston ; guérison apparente, pas de nouvelles ultérieures de ce malade. Il s'agissait d'un cancer avec des points polypoïdes à la périphérie.

En 1913 nous avons observé un homme de 50 ans atteint de cancer histologique du sinus maxillaire et de la paroi externe du nez ; il a été opéré à l'hôpital par la résection du maxillaire supérieur. Suites immédiates bonnes.

Une seule fois, et le fait est rare, nous avons observé et opéré chez un enfant (avec Molin, dans le service de Nové-Josserand) une tumeur diffuse de la partie interne de la joue et du sinus maxillaire. Incision classique de la résection du maxillaire supérieur sans ouvrir la bouche ; infiltration diffuse, jaunâtre, de nature douteuse, s'étendant depuis le sinus maxillaire jusqu'à la partie postérieure de l'ethmoïde ; curetage et ablation osseuse large ethmoïdale et maxillaire ; pas de résection totale. Récidive orbitaire trois semaines après ; la famille refuse une nouvelle opération. Mort rapide. A l'examen histologique, tumeur atypique, très probablement épithéliale, mais à caractère très malin et à développement très rapide.

En 1912 nous avons eu l'occasion, avec MM. Dor et Vignard, d'observer un très beau cas de tumeur mélanique du nerf optique avec extension aux sinus : Femme, 64 ans, tuberculose dans la famille. Depuis 4 mois tumeur rétro-orbitaire droite ; cécité brusque à droite depuis 15 jours, avec douleurs surtout fronto-temporales. L'opération n'est acceptée qu'au bout d'un mois et demi ; il y a déjà quelques petites douleurs de l'œil gauche ; opération de Moure ; la tumeur mélanique a envahi la paroi extrême du nez, la partie postérieure de l'orbite, le trou optique, la lame criblée et le sphénoïde à droite et sur la ligne médiane. Curetage prudent du néoplasme à la curette entourée de gaze, mise à nu de la dure-mère, très épaissie sur une grande étendue ; suites opératoires bonnes, cessation presque complète des douleurs ; mort deux mois après l'opération par généralisation à gauche, cécité complète et coma. L'examen histologique a montré à M. Dor qu'il s'agissait d'une tumeur mélanique de la région de la gaine du nerf optique.

Comme conclusion, nous insistons sur les points suivants : 1° L'extrême rareté des tumeurs malignes des sinus chez l'enfant, et sa fréquence relative chez l'adulte, surtout chez l'homme. 2° Cette affection simule très souvent des sinusites ; le diagnostic est difficile, cependant des douleurs intenses rebelles, et surtout des troubles sensitifs, doivent y faire songer. L'âge n'est pas toujours un élément absolu, nous venons en effet d'opérer, sous anesthésie locale d'infiltration avec anesthésie régionale du nasal et du sous-orbitaire deux, cas chez des hommes de 60 ans de lésions inflammatoires sinusiennes multiples surtout ethmoïdo-sphénoïdales qui simulaient des cancers infectés. 3° En raison de la fréquence des formations polypeuses à la périphérie du néoplasme, formations d'origine inflammatoire, en raison aussi de la transformation de polypes en néoplasmes, transformation qui ne débute pas par la périphérie ; les examens histologiques négatifs même répétés ne permettent pas d'exclure fatalement le néoplasme ; en cas de doute il vaut mieux intervenir surtout par la voie naso-latérale préconisée par Moure. 4° En présence d'un néoplasme étendu aux sinus à l'orbite, notamment à la région ethmoïdo-sphénoïdale, il faut intervenir, à moins de contre-indication absolue, même pour une opération de propreté qui consiste à mettre tous les sinus et le nez en une même et seule cavité ; le suintement, les hémorragies et les douleurs cessent à peu près complètement ; le malade a l'illusion de la guérison et la récidive est parfois très tardive ; elle se fait surtout au niveau de la région orbitaire et crânienne, en des points par conséquent où elle est le plus souvent inopérable. 5° Nous pratiquons l'opération de Moure sans inciser jusqu'à la lèvre ; avec la lampe frontale le jour est très suffisant. Le drainage à la gaze est uniquement nasal ; nous suturons totalement l'incision externe, mais pour éviter les complications cornéennes post-opératoires nous maintenons les paupières suturées pendant 4 à 5 jours. Jusque-là, nous avons fait toutes nos opérations externes nasales pour néoplasmes sous anesthésie générale, mais à l'exemple de Luc et instruit par le bon résultat de nos deux derniers cas de pansinusites

opérées sous anesthésie locale d'infiltration, avec anesthésie régionale des nerfs nasal et sous-orbitaire, nous la pratiquerons dorénavant sous anesthésie locale, combinée ou non avec une anesthésie générale très légère. Les suites opératoires ont toujours été très bonnes, nous n'avons perdu aucun malade de complications post-opératoires, même quand la dure-mère a été mise à nu.

Dr. Bond (London). The literature and the number of operations devised for these maladies appal one. I will draw only on my own experience. As to the growths in the naso-pharynx, many are inoperable, many others are best left alone. If small and with a definite outline in front they can be removed with hope of success.

Of my anterior lower nasal cases I have removed three by intra-nasal operation. One epithelioma was curetted three times, twice glands in the neck were removed, and finally the patient was cured. Another, a sarcoma on lower front of septum, seems cured.

I agree with Dr. Marschik as to the gravity of a recurring, unilateral polypus of the nose in an old person. Recently I had a case of recurring polypi, of eight years' standing, in which curetting, and finally removal of the upper jaw and other parts were carried out.

Preliminary laryngotomy has been helpful to me in operating on these cases. One has more room, there is less fear of discharge, blood, &c. getting into the lungs, and should a little get down it is more likely to be coughed up than when a tube through the mouth is used.

Professor I. Kubo (Fukuoka, Japan). Wenn die gute Prognose der Operation von der Frühdiagnose abhängig ist, so liegt das Schicksal der malignen Tumoren der Nase in den Händen der Rhinologen. Was die Operation der malignen Tumoren der Kieferhöhle anbetrifft, wäre es rationell, zuerst die Höhle von der Fossa canina aus aufzumachen und von hier aus Ausrottung zu probieren, da totale Kieferresektion oft nicht nötig ist. Wenn es nicht hilft, so operiere ich von aussen mit Kochers Hautschnitt, — meist unter Lokalanästhesie. Die Prognose der Operation ist gewöhnlich günstig, wenn die Sensibilität des weichen Gaumens auf der kranken Seite noch nicht herabgesezt oder alteriert ist. Ich habe drei Fälle von blutenden Polypen der Nasenhöhle aus der Kieferhöhle, besonders an der Nasalwand, ausgehen sehen, in denen klinische Befunde ganz wie bei malignen Tumoren waren, während man mikroskopisch das Bild eines blutenden Polypen sah. Differentialdiagnose zwischen Papillomen und Karzinomen ist oft sehr schwierig, da ein von Pathologen als Papillom beurteilter Fall doch schliesslich ein Karzinomfall sein kann. Bei einem über 60 jährigen Patienten sah ich ein hartes Papillom aus dem mittleren Nasengang, das schon seit 15 Jahren bestand und auch von anderen Aerzten mehrmals operiert wurde, ohne in eine maligne Form überzugehen. Bei der Diagnosenstellung muss man pathologisch sowie klinisch genau untersuchen.

Dr. A. JANSEN (Berlin) beschränkt sich auf einige Worte über die malignen Tumoren im Bereiche der Nase. In Unkenntnis des Italienischen weiss er nicht, ob Ferreri ebenso viel Wert auf frühzeitigen operativen Eingriff von aussen legt, wie er selbst. Da der Tumor in alle vier Höhlen früh hineinwächst, wählt er den Unterbrauschnitt, von wo man alle vier Höhlen gut übersichtlich machen kann, und der vielfach zur Radikaloperation genügt. Wenn die Kieferhöhle mehr als im hintern obern Winkel ergriffen ist, muss sie von der foss. can. eröffnet werden, von hier kann man selbst subkutan den ganzen Oberkiefer resezieren. Diagnostisch erscheint ihm die wiederholte Blutung von oben wichtig, bei geringen Veränderungen im mittleren Nasengange. Der Verdacht wächst bei leichter Protrusio bulbi und wird noch weiter unterstützt durch tiefen Schatten im Bereiche des Siebbeines. Redner verwendet Lokalanästhesie in Verbindung mit subkutanen Hyoscin-Morphium-Injektionen.

Professor SEGURA (Buenos Aires). It is my belief that success in the removal of an intranasal malignant tumour lies in early operative intervention.

The hæmorrhages are of great diagnostic value. I believe that in some inoperable cases the application of radium is of decided benefit, especially in stopping hæmorrhages. It is of importance to apply radium before an operation in order to diminish the blood supply; I have seen good results in carcinomatous cases in conjunction with Dr. Woolmer of Buenos Aires.

Mr. T. JEFFERSON FAULDER (London). (1) Reference is made in several places by Professor Ferreri to the relations between general surgeons and specialists. Thus, he alludes to 'l'opinione assai diffusa fra i chirurgi generici che essi s'intendono altrettanto bene di rinologia come gli specialisti'. To the general surgeons we might very well add general physicians and general practitioners. Certainly more use ought to be made of the special training of rhinologists particularly with reference to early diagnosis. The majority of cases of malignant disease are very far advanced when they reach us.

(2) However, I think it is our duty to deal, so far as we can, with even 'gli scarti dei chirurgi generici'. Some of those present may have seen a case which I showed here last week. The patient in question presented a remarkable view of a nearly normal larynx through a large defect in the face. This defect was caused by rodent ulcer together with surgical interference. The disease is now involving the naso-pharynx. This old man has had many forms of treatment during the forty years the disease has lasted and is fairly happy so long as something is being done for him. At the present time the growth is being controlled to some extent and the pains mitigated by X-rays.

(3) With regard to the possible transformation of one form of malignant

disease into another, I have a case to relate. I excised an upper jaw for malignant disease of the antrum, removing also the palate bone, pterygoid plate, inferior turbinal, and a considerable part of the ethmoid. The pathologists reported that the disease was epithelioma. This patient remained well for nearly four years, when he was referred to me by an ophthalmic surgeon who reported that the eye on the affected side was going blind. I found the base of the skull covered with a soft vascular diffuse growth. A large piece of the tissue was removed for examination. The pathologists reported that it was round-celled sarcoma of a most virulent type.

(4) With regard to the use of ' protesi ' in repairing the defects caused by wide extirpations of the facial skeleton, so far as my experience goes they are of great service. Indeed, before excising an upper jaw it seems a good plan to have a cast of the jaws made. From this cast a light silver wire frame can be constructed. Such a frame can be placed *in situ* at the conclusion of the operation. It enables the suturing of the external wound or wounds to be done more rapidly and accurately ; and therefore facilitates healing. It in no way prevents packing of the cavity. It improves the drainage, if improvement were needed. It makes inspection of the cavity easy and in the case of infra-orbital incisions, the well-known œdema of the lower eyelid appears later and disappears sooner. Obviously also it tends to prevent deformity of the face. The cast and the frame are a great help to the dental surgeon in making a permanent artificial apparatus.

Sir StClair Thomson (London) hoped that the work done by rhinologists would ere long render excision of the upper jaw an extinct operation. Pathologically they knew that in the majority of these cases the malignant growths started from the ethmoid or its neighbourhood. Removal of the alveolar border implied operating at the furthest point possible from the seat of disease, the bleeding was copious, the exposure very inefficient, the danger considerable, and the results in all cases disfiguring and seldom curative.

By such operations as those of Moure, Roux, Denker, or a combination of two or more of them, the ethmoid could be attacked directly, the sphenoid and infundibulum cleared out, the risk from hæmorrhage abolished by a laryngotomy, post-operative disfigurement was hardly present, and there was a good chance of permanent cure. One such case of his was still alive three years afterwards, and a second he had shown the previous day. This was a patient in whom the antrum was crowded with growth, but he had now been ten months without recurrence and there was no disfigurement.

It was not right to be hopeless before operation. Sometimes, in a promising case, the patient would show that the floor of the anterior fossa was already involved. In some other cases he had hardly hoped to have cleared away all the growth, and yet patients survived for years without

a recurrence. As in other parts of the body, hope was founded chiefly on early diagnosis and treatment by a specialist.

Mr. HERBERT TILLEY (London) said that at the Radium Institute, where they had large quantities of radium, excellent results had been attained by its use in apparently hopeless cases of malignant disease of the nasal and post-nasal regions.

The best results were observed in the sarcomatous and endotheliomatous types of disease ; in squamous epithelioma, on the other hand, no benefit was noted.

The best results were attained by inserting the radium into the middle of the growth and leaving it for twenty-four hours.

At the present moment the speaker was treating a case of endothelioma of the right side of the naso-pharynx with marked improvement so far as relief of pain and obstruction, and diminution of the facial anæsthesia were concerned ; for the first time for three months the patient had enjoyed and tasted his food and had slept well.

The speaker wished to emphasize the importance of deafness as an early symptom of endothelioma of the naso-pharynx. When examination proved that deafness was due to a collection of serous fluid in the tympanum, and when this was not cured by repeated paracentesis coupled with the usual treatment, one should be careful not to overlook the possibility of involvement of the inner end of the Eustachian tube by an infiltrating growth. Deafness in ten of his cases had been the earliest symptom of endothelioma of the naso-pharynx.

Dr. JACQUES (Nancy). A l'époque où elles se présentent à notre examen les tumeurs du naso-pharynx sont généralement trop étendues pour qu'il nous soit permis d'espérer une éradication complète. L'excision par les voies naturelles constitue pour nous une ressource, précaire à la vérité, mais, du moins, relativement inoffensive. Il s'agit le plus souvent de lymphosarcomes à évolution rapide et infiltration précoce. L'intervention n'a guère alors qu'une utilité morale.

Il semble toutefois que le recours opératoire ne soit plus l'unique solution à offrir au malade en cette triste occurrence. J'ai eu pour ma part un succès aussi remarquable qu'inattendu par l'emploi du cuivre colloïdal en injections intrafessières. Il s'agissait d'un homme de quarante ans, atteint d'un sarcome globocellulaire à évolution rapide du cavum chez qui, trois mois après une éradication aussi large que possible, avait vu sa tumeur repulluler au point de combler tout le rhino-pharynx et d'infiltrer l'espace maxillo-pharyngien et la fosse parotidienne, malgré un traitement antisyphilitique énergique préalablement pratiqué. En deux mois la tumeur régressa complètement sans aucune intervention locale et j'assistai à son élimination progressive par nécrose. Le malade, opéré par moi en septembre dernier fut soumis aux injections d'électrocuprol au début de janvier et j'ai pu le présenter entièrement guéri fin mars à la

Société de médecine de Nancy. Je le surveille de près et aucune récidive n'a encore paru.

J'ajoute que mon expérience m'a permis de vérifier l'assertion de notre collègue relative à la valeur diagnostique d'un écoulement muco-hématique persistant par l'oreille chez un opéré de cancer du nez ou du rhino-pharynx.

Replies

Il Professore Ferreri (Roma). Riassumendo la mia opinione sulla cura dei tumori maligni delle prime vie aeree, dirò che i progressi della terapia medica generale (in specie la radium-terapia, la chemo- e opoterapia) hanno ridotto molto gl' insuccessi delle eseresi radicali che per lo più non preservano da più o meno tardive recidive. La chirurgia da sola non può essere padrona che di un ben ristretto numero di tumori maligni limitati alle narici e al seno mascellare e di struttura connettivale o endoteliale. Particolarmente nell' infanzia dobbiamo essere guardinghi da gravi demolizioni e piuttosto affidarci a parziali eseresi per raggiungere il tumore, asportarlo e sottoporre il campo operativo alla radium-terapia. Nei bambini e nei giovani facemo la cloro-flebo-narcosi che evita di complicare la gravità dell' operazione con la tracheotomia preventiva. Negli adulti la via da noi preferita per l'asportazione dei neoplasmi maligni naso-faringei e delle cavità accessorie nasali è la transmascellare, anestesizzando i pazienti col metodo di Crile, di Kuhne e di Meltzer se non sono arterio-sclerotici, nefritici o neuro-patici: in questi casi dovremo ricorrere all' anestesia locale o alla narcosi per la via rettale. In generale può dirsi che i progressi della tecnica chirurgica rinologica hanno ridotto assai il numero degl' infelici che una volta venivano sottoposti alla totale asportazione del mascellare superiore, particolarmente se in tempo precoce apriscono le cavità accessorie sospette, e insisteremo nella applicazione *prolungata* del radium.

Dr. H. Marschik (Wien). Ein Teil der mir zustehenden Antworten auf verschiedene Redner ist in dem Referat enthalten, welches ich mangels an Zeit nicht vollständig durchlesen kann. Ich möchte nur auf die Affektion der Hirnnerven zurückkommen und die Erfahrungen der Wiener Klinik erwähnen, dass die Nerven, auch die Sinnesnerven, gegen die neoplasmatische Wucherung eine auffallende Widerstandskraft zu haben scheinen. Man findet die Nerven vom Tumor ganz umwachsen und doch, auch bezüglich ihrer Funktion, noch ganz intakt. Sie scheinen mehr empfindlich gegen Druck, Oedem oder toxische Schädigung zu sein. Was ferner das Resultat unserer Untersuchungen anbelangt, so ist vor allem eine grössere Anzahl von Beobachtungsserien an einheitlichem Material derselben Station, mit genauen histologischen Untersuchungen, und vor allem bei Nasenrachentumoren Beobachtung bis an das Ende notwendig. Was den Standpunkt der Behandlung anbelangt, so trachten wir, nach Möglichkeit und frühzeitig zu operieren und nachher Radium

oder Röntgen anzuwenden. Ich erinnere an zwei von mir publizierte Fälle von malignen Tumoren, der eine Carcinoma der Kieferhöhle ; bei Radikaloperation musste auf Verlangen des Patienten der orbitale Teil des Tumors zurückgelassen werden und erzeugte schnelles Rezidiv. Röntgenbehandlung brachte Heilung, bis jetzt sind es schon vier Jahre. Zweitens, eine Frau von 83 Jahren mit kleinzelligem Rundzellensarkom des Oberkiefers und Siebbeins. Da an Radikaloperation nicht zu denken war, kurettierte ich das Gros des Tumors und liess Röntgenbehandlung folgen, wodurch durch drei Jahre bis zum Tod durch Altersapoplexie ein der Heilung adäquater Zustand erreicht wurde. Bezüglich Radiumbehandlung erwähne ich einen Fall von Nasenrachencarcinoma, in dem durch Kurettage und nachfolgende Radiumbehandlung Rückgang der diversen, bereits ausgebildeten Hirnnervenlähmungen erzielt wurde.

SECTION XV

RHINOLOGY AND LARYNGOLOGY

INDEPENDENT PAPER

DIE ANWENDUNG EXPERIMENTELL-PHONETISCHER METHODEN FÜR DIE DIAGNOSTIK UND THERAPIE FUNKTIONELLER STÖRUNGEN DER STIMME

Von Professor HERMANN GUTZMANN, Berlin

Dass die experimentell-phonetischen Methoden für die Laryngologie von grosser Bedeutung sind, ist nicht nötig hier zu beweisen. Es genügt zu erwähnen, dass ihr Zusammenhang mit der Laryngologie das erste Thema war, das auf dem letzten Internationalen Laryngo-Rhinologen-Kongress (1911, Berlin) auf die Tagesordnung gesetzt wurde ; es genügt, daran zu erinnern, dass bei dieser Gelegenheit, durch eine Ausstellung von Apparaten, methodischen Anordnungen, Kurven etc., die aus allen wissenschaftlichen Zentren der ganzen Welt beschickt war, ein wahrhaft überraschendes Bild der Entwicklung jenes Zusammenhanges gegeben wurde. Es mag ferner darauf hingewiesen werden, dass in bezug auf den Zusammenhang der experimentellen Phonetik mit der allgemeinen Medizin schon von Zwaardemaker und Bouman in Budapest ein ausführliches Referat gegeben wurde und dass erst vor kurzem wieder die italienischen Kollegen Gradenigo, Biaggi, Stefanini ein Referat über dasselbe Thema auf dem Italienischen Laryngo-Otologen-Kongress als notwendig und zeitgemäss erstattet haben.

Auch für diesen Kongress bestand ursprünglich offenbar die Absicht, dieses neue wissenschaftliche Arbeitsgebiet für die Tagesordnung unserer Abteilung festzuhalten. Aus welchen Gründen man von einer allgemeinen Diskussion, d. h. Festlegung eines entsprechenden Referats Abstand genommen hat, entzieht sich meiner Kenntnis. Aber gerade dieser Umstand ist es hauptsächlich, durch den ich mich veranlasst fühlte, meinen Vortrag anzumelden. Denn ich halte es vor allem im Interesse der Laryngologie selbst für dringend notwendig, das Thema der experimentellen Phonetik auf unseren Kongressen *stets* vertreten zu sehen.

Aber noch ein anderer Grund kam hinzu.

Nachdem die experimentelle Phonetik als neue fruchtbare und nicht

nur vielen Gebieten der Medizin, sondern früher schon der Phonetik selbst, der Physiologie, der experimentellen Psychologie u.s.w. dienende Wissenschaft und Methodik neue und fast unerschöpfliche Arbeitsgebiete erschlossen hat, sind eine Anzahl von Forschern, von Linguisten, Philologen, Physiologen, Medizinern der mannigfachen Einzelgebiete, Psychologen etc. zu der Ueberzeugung gekommen, dass einer derartigen Wissenschaft, die sich wie die Medizin an das allgemein Menschliche wendet, am besten die Wege geebnet werden könnten, wenn alle die, welche von ihrer Wichtigkeit überzeugt sind, sich zu einer *internationalen Gesellschaft für experimentelle Phonetik* vereinigen und überall dort, wo es sich um die Besprechung internationaler wissenschaftlicher Arbeitsgebiete handelt, auch geschlossen zur Geltung kämen.

So haben sich die Herren Biaggi, Gradenigo, Stefanini vereinigt, um zur Gründung einer derartigen *internationalen* Vereinigung den Anstoss zu geben. Mir schien es sehr wünschenswert, Ihnen auch an dieser Stelle diese Tatsache ausdrücklich zu berichten, und ich danke unserm Herrn Präsidenten Sir StClair Thomson insbesondere dafür, dass er mir erlaubt hat, diesen Gegenstand hier vorzubringen. Sie alle, meine Herren, fordere ich zur Mitarbeit und zum Beitritt auf. Mit irgendwelchen Verpflichtungen ist dies zunächst nicht verknüpft, da die Einzelheiten den betreffenden nationalen Komitees überlassen bleiben. Uns, speziell meinem verehrten alten Freunde Gradenigo und mir, liegt hier nur daran, Ihr Interesse für diese neue Entwicklung wach zu rufen und Sie auf das Rundschreiben, das wir zirkulieren lassen, besonders hinzuweisen. Dabei darf ich wohl erwähnen, dass ein Zentralorgan für diese internationalen Bestrebungen bereits seit vielen Jahren besteht: das von mir und Dr. Panconcelli-Calzia herausgegebene *Internationale Zentralblatt für experimentelle Phonetik*: 'Vox' ('und dass im April 1914 der erste internationale Congress für experimentelle Phonetik in Hamburg stattfinden wird.' Nachtrag bei d. Korrektur).

Die Stimmstörungen, um die es sich hier handelt, gehören zu den *Phonopathien*. Die Phonopathien teilen wir entsprechend dem anerkannten Einteilungsprinzip der Lautsprachstörungen in *Dysphonien* (Störungen der inneren zentralen gesangs-musikalischen Vorgänge, entsprechend den Dysphasien der Lautsprachstörungen) und in *phonische Dysarthrien* (Störungen der Bahnen und Organe, welche die phonischen Vorstellungen auszuführen haben, entsprechend den Dysarthrien der Lautsprachstörungen). Je nachdem nun die Rufstimme, die Gesangsstimme oder die Sprechstimme betroffen ist, handelt es sich bei diesen phonischen Dysarthrien um (1) *Dysklesie* (Störung der Rufstimme), (2) *Dysarthrophonie*, hier speziell im artikulatorischen Sinne (Störung der Gesangsstimme) und um (3) *Dysphthongie* (Störung der Sprechstimme). In diesen drei Hauptgruppen kann man als Unterabteilungen unterscheiden die spastischen Formen, die asthenischen Formen (Phonasthenie), die parakinetischen Formen (z. B. die klappernde, meckernde Stimme;

die ‘ vox interrupta ’ ; die ‘ vox rauca ’) u. a. m. Als besonders eigenartige Formen erwähne ich noch die *habituellen Dyskinesien der Singstimme*, die *habituellen Dyskinesien* und *Hypokinesien der Sprechstimme*, bes. derjenigen der Kinder (*habituelle Stimmbandlähmungen*, H. Gutzmann), die *Mutationsstörungen* : ‘ Dysontogenia vocis ’ (persistierende Fistelstimme, Mutatio prolongata, Mutatio perversa etc.).

Denken wir noch an die mannigfachen Störungen der Stimme auf *hysterischer Basis*, so sehen Sie, dass das Gebiet, um dessen Diagnostik es sich hier handelt, recht gross ist, und wenn Sie sich daran erinnern, dass oft genug organische Veränderungen im Larynx als Folgeerscheinung funktioneller Störungen aufzufassen sind und dass die sichere Entscheidung über dieses Kausalverhältnis häufig auf Schwierigkeiten stösst, so geht schon hieraus hervor, dass zuverlässige Methoden, die die Diagnostik erleichtern, dem Laryngologen zur Verfügung stehen müssen. Derartige Methoden sind gerade die experimentell-phonetischen.

Die Diagnose der oben kurz angegebenen funktionellen Stimmstörungen lässt sich mit der üblichen Laryngoskopie oft genug nicht stellen. Man findet normale laryngoskopische Bilder bei den schwersten funktionellen Stimmstörungen ; gelegentlich bekanntlich aber auch auffallende Abweichungen bei gesunden Stimmen, manchmal sogar im Kehlkopf ausgezeichneter gesunder Sänger : z. B. Schiefstellung, einseitige Schwellung der Taschenlippen, Röte und Schwellung der Stimmlippen. Diese *Widersprüche zwischen laryngoskopischem Befund und Funktion der Stimme* sind wohl jedem Laryngologen bekannt.

Da der Spiegel also hier versagte, musste man nach Methoden suchen, welche eine Beurteilung der Funktion gestatteten. Eine überaus wichtige, auch zu den experimentell-phonetischen Methoden gehörende Versuchsanordnung ist die von Oertel eingeführte *Laryngostroboskopie*, zu deren Ausführung eigentlich nichts weiter gehört, als dass man auf die Achse irgendeines Motors, z. B. des bei dem gewöhnlichen ‘ Multostaten ’ vorhandenen, eine grosse Sirenenscheibe setzt, durch die der Lichtstrahl während des Laryngoskopierens und Phonierens der Tonhöhe entsprechend unterbrochen wird. In unserm Berliner Universitätsambulatorium haben wir nach vielen Versuchen diese einfachste Form der Laryngostroboskopie eingeführt. Da die Lichtunterbrechungen das laryngoskopische Bild verdunkeln, so ist allerdings dafür zu sorgen, dass die Lichtquelle eine möglichst intensive ist. Es ist bedauerlich genug, dass die Laryngostroboskopie unter den Laryngologen noch so wenig Eingang gefunden hat, ja dass sie sehr vielen sogar noch völlig unbekannt geblieben ist. Wer sich daran gewöhnt hat, die stroboskopische Scheibe fortwährend funktionsbereit neben sich zu haben und regelmässig zu benutzen, wird von dem Verfahren nicht wieder abgehen. Der Einblick in die Funktion resp. die Funktionsstörung, die Kontrolle der Funktionsbesserung unter bestimmter Therapie, der Vergleich zwischen dem stroboskopischen Befunde und dem Gehörseindruck der Stimme etc., d. h. die sichere und zuverlässige Beurteilung der Stimmfunktion, wird

in vielen Fällen nur durch das Stroboskop gewonnen. Die Laryngostroboskopie ist demnach eine der wichtigsten experimentell-phonetischen Methoden und so leicht und mit so geringen Mitteln überall zu bewerkstelligen, dass man wohl wünschen möchte, sie würde in allen laryngologischen Kliniken den Studenten auch regelrecht und regelmässig gezeigt, denn sie hat nicht nur theoretische, sondern auch eminent *praktische* Bedeutung.

Während aber die Laryngostroboskopie durch das Aufsetzen einer einfachen Pappscheibe auf den überall vorhandenen ‘Multostaten’ ausgeführt werden kann und demnach kein neues Instrumentarium bedingt, sind die *graphischen Methoden der experimentellen Phonetik* nur mit besonderen dem Laryngologen sonst kaum zur Verfügung stehenden Instrumenten ausführbar. Jedoch ist die Anwendung dieser Methoden, besonders der Pneumographie, Laryngographie, Vibrographie, Photophonographie, stroboskopischen Kinematographie, Arthrographie etc. für die Diagnostik mannigfacher funktioneller Stimmstörungen so überaus wichtig und entscheidend, dass das dazu nötige Instrumentarium wenigstens in einfacher Form auch jedem praktischen Laryngologen zur Hand sein sollte. Ich habe in einem kleinen Apparat, den ich Ihnen hier vorlege, alles dazu Nötige vereinigt. Sie sehen hier eine zierliche kleine Maschinerie, die ein fertig berusstes Papier an verschiedenen Schreibfedern vorbeiführt. Mittelst der Schreibkapseln können Sie die Atmungsbewegungen, die Stimmvibration, die Artikulationsbewegungen etc. leicht aufnehmen. Ich werde Ihnen nachher einige so gewonnene Kurven vorlegen und an ihnen den Nachweis führen, wie wichtig sowohl diagnostisch wie therapeutisch die objektive Fixierung der Funktionsstörungen der Stimme ist.

Eine, besonders therapeutisch wichtige, Art der experimentell-phonetischen Methoden stellen die sogenannten *Ausgleichsmethoden* dar: Anwendung des faradischen Stromes, des Leduc'schen Stromes, Vibration u.s.w. auf das Stimmorgan. Es ist hier nicht möglich, auf alle diese Methoden im einzelnen näher einzugehen. Ausführlich geschildert sind sie von Th. S. Flatau, der viele derselben zuerst angegeben hat, und in letzter Zeit besonders eingehend und übersichtlich von Imhofer. Auch hier wird man, um die Einführung der Methodik in die laryngologische Praxis zu erleichtern, gut tun, sich zunächst auf solche Instrumente zu beschränken, die mit dem schon vorhandenen Instrumentarium leicht in Verbindung gebracht werden können oder auch sonst schon vorhanden sind: Faradisation, Vibration und Leduc'scher unterbrochener Gleichstrom. Letzterer kann durch ein kleines Instrument erzeugt werden, das auf jede Achse eines ‘Multostaten’ leicht anbringbar ist. Neuerdings hat Flatau auch die Hochfrequenzströme zur Anwendung empfohlen. Mir scheint, mit Recht. Schon die Benutzung eines einfachen Anschlussapparats zur Erzeugung von Hochfrequenzströmen zeigt, dass z. B. bei schmerzhaften spastischen und asthenischen Zuständen der Stimme der Ausgleich oft in überraschend günstiger Weise erzielt wird.

Wendet man die verschiedenen Formen dieser Ausgleichsmethoden längere Zeit therapeutisch an, so kommt man bald dahin, *dass die Auswahl derselben für die Therapie bereits bei der Diagnostik stattfinden muss.* So ist bei den spastischen Formen, den Hyperkinesien der Stimme, die Anwendung der Vibration sehr häufig schädlich. Dagegen wirkt die Anwendung des unterbrochenen Gleichstromes nach Leduc besonders dann, wenn er *synchron unterbrochen* wird (was, wie ich früher gezeigt habe, durch meine kleine auf die Achse des Multostaten aufgeschraubte Zahnradsirene leicht kontrolliert werden kann), ausserordentlich günstig ein ; ebenso die Hochfrequenzströme. Dagegen ist die Anwendung der Vibration in ihren verschiedenen Formen bei den asthenischen Stimmstörungen, den Hypokinesien der Stimme vorteilhaft. Ebenso die Elektrizität, die Faradisation ; hier auch Anwendung des mit der Stimmhöhe synchron unterbrochenen Leduc'schen Gleichstroms. Für die Vibration ist die Verwendung der von mir angegebenen elektrisch betriebenen Stimmgabeln insofern eine eigenartige, als es sich dabei weniger darum handelt, die Stimme durch starke Vibrationsstösse zur Einhaltung der Tonhöhe zu *zwingen*, als darum, dass bei Abweichung der Tonhöhe des Patienten von der Zahl der auf seinen Kehlkopf übertragenen Vibration der elektrischen Stimmgabel *Schwebungsstösse* entstehen, die der Patient zu vermeiden trachten muss. Weicht er eine Spur nach oben oder unten von der Tonhöhe ab, so entstehen Schwebungen. Dadurch, dass er sich bemüht, die Schwebungen zu vermeiden, übt er sich darin, *durch seinen Willen die bestimmte Tonhöhe einzuhalten.* Gegenüber der sonstigen groben Vibration ist also dieses von mir vorgeschlagene Verfahren mehr auf psychischem Wege wirksam.

Ganz besonders wichtig ist die Anwendung der feinen Vibration der elektrisch betriebenen Stimmgabeln bei den Parakinesien der Stimme.

Sie sehen, dass schon die Auswahl betreffs der in dem einzelnen Falle nötigen Ausgleichsmethoden *neben der Diagnostik gleichzeitig Therapie* ist ; denn die Ausgleichsmethoden sind unsere wichstigste therapeutische Waffe für die funktionellen Stimmstörungen. Auch hier muss ich darauf verzichten, auf die Einzelheiten näher einzugehen, da sie oft genug beschrieben und bekannt sind.

Weniger bekannt aber ist es, dass die vorher erwähnten experimentell-phonetischen graphischen Methoden ebenfalls eine grosse Bedeutung für die Therapie haben. Auch bedaure ich, dass man den *Phonographen zur Registrierung der Stimme* immer noch relativ selten verwendet, obgleich schon billige Phonographen (für 50 M. = £2 10s.) vollkommen Genügendes leisten und dieser wirkliche *Spiegel der Stimme* ein ausgezeichnetes *Kontrollmittel der Therapie* ist. Schon vor 20 Jahren habe ich auf diese praktische Verwendung des Phonographen aufmerksam gemacht und durch Beispiele die Anwendbarkeit gezeigt.

Zur *objektiven Kontrolle des therapeutischen Verfahrens* wird ferner die Aufnahme der Atmungsbewegungen, der Stimmvibration und der Artikulationsbewegungen eine stets sichere Unterlage bieten. Man denke

z. B. an die Aufzeichnung der Nasendurchschläge bei den verschiedenen Formen der nasalen Stimme, die Kontrolle der Wirkung von Gaumenspaltenoperation oder Gaumenobturator oder auch die Kontrollierung der Wirkung nach Herausnahme von adenoiden Vegetationen u.s.w.

Den kleinen ' Kehltonschreiber ', den ich vorher demonstrierte, kann man auch dazu benutzen, um Abweichungen der Stimme in der Tonhöhe *dem Auge sichtbar zu machen.* Leitet man nämlich die Vibration einer elektrisch betriebenen Stimmgabel gleichzeitig mit der Vibration der Stimme auf den Kehltonschreiber, so wird er *gleichmässig* ausschlagen, *solange beide Töne gleich hoch sind,* in zuckende Bewegungen, d. h. *Schwebungen* geraten, wenn eine mehr oder weniger starke *Abweichung* stattfindet. Man kann daher *neben der akustischen Kontrolle auch die optische* und, da der Patient die Schwebungen als kleine Stösse am Kehlkopf *fühlt,* auch *die taktilen Kontrollmittel* verwenden. Ebenso wird, wie ich Ihnen nachher zeigen werde, die Kontrolle der Atmungsbewegung während der Therapie bei Laryngektomierten, welche die ' Pharynxstimme ' lernen sollen, von Wichtigkeit ; die Kontrolle lässt sich ebenfalls überaus leicht *optisch* für den Patienten darstellen, wenn die Atmungsbewegung durch eine kleine Schreibkapsel auf das Kymographion übertragen wird. Der Patient hat dann neben der *taktilen* Kontrolle seiner Hand, gleichzeitig eine *optische* Kontrolle.

Eine grosse Bedeutung — vielleicht die grösste — der experimentell-phonetischen Methoden für die *Therapie* dürfen wir darin sehen, dass sie uns *vor falscher Indikationsstellung in bezug auf operative Eingriffe bewahren können.* Das ist der Fall bei verschiedenen Formen der näselnden Stimme.

Zum Schluss gebe ich Ihnen hier einige Beispiele, in welcher Weise die Anwendung graphischer experimentell-phonetischer Methoden für die Diagnostik und Therapie funktioneller Stimmstörungen entscheidend sein kann.

(1) In diesem Fall handelt es sich um eine 27 jährige Dame, die beabsichtigte, Schauspielerin zu werden, und bei den in der Vorbereitungszeit vorgenommen dramatischen Uebungen Schwierigkeiten in der Sprache bekam. Aus verschiedenen Gründen schloss ich aus dem mir von Professor Pfaundler (München) gegebenen ausführlichen Bericht, dass es sich wohl um eine leichte Form der *angeborenen infantilen Pseudobulbärparalyse* handeln müsse, besonders da auch eine charakteristische *Schreibstörung* vorhanden war. Auf meine weitere Nachfrage ergab sich ferner, dass *in früher Jugend Schluckstörungen* bestanden hatten.

Es handelte sich um *die Entscheidung, ob unter diesen Umständen überhaupt die Absicht der Dame, Schauspielerin zu werden, durchführbar sei.* Dies konnte nur entschieden werden durch genaue graphische Untersuchung des gesamten Sprechvorganges, und Sie sehen hier das Resultat. Würde der zeitliche Ablauf in den einzelnen Artikulationsfolgen wesentlich verlangsamt gewesen sein, so müsste man von einem solchen Beruf abraten. Aber die Kurve zeigte, dass die zeitlichen Verhältnisse im Silbenablauf normale waren ; nur *die Bewegungsvorgänge selbst* waren *exzessiv gesteigert,* die ' *Amplitude* ' der Bewegung war grösser als normal. Offenbar war dies durch eine etwas übertriebene Uebung veranlasst

worden. Durch diese exzessiven Bewegungen kam es dann sehr häufig zum Versprechen (*Paralalie*) und sogar auch zu spastischen Erscheinungen.

Nachdem die Patientin gelehrt worden war, die übertriebenen Bewegungen abzustellen und sie der Norm mehr anzunähern, verschwanden innerhalb weniger Wochen die Störungen soweit, dass man für die Zukunft eine günstige Prognose stellen kann.

Es handelte sich also hier nicht nur um die Zeitmessung der Silbenfolge, sondern um die Feststellung der Bewegungsart. Diese war *durch die einfache Beobachtung nicht möglich*, wenigstens nicht von vornherein. Nachdem die Kurve erst gewonnen war und ich nun auf die Amplitude der Bewegung meine Aufmerksamkeit richtete, war natürlich auch mit dem Auge die Abweichung feststellbar.

(2) In dem zweiten Falle handelt es sich um eine *Insufficientia veli palatini mit submuköser Knochenspalte*. Die Frage war hier, ob die bestehenden adenoiden Vegetationen operiert werden sollten oder nicht, da man den Eindruck hatte, als ob eine Verstopfung des Nasenweges oder eine Verlegung der Nasenluft an dem Näseln die Schuld trage. Die einfache *Inspektion* ergab nichts Abweichendes, die *Palpation* dagegen die *submuköse Knochenspalte*. Der Klang der Stimme war wegen der mässigen adenoiden Vegetationen der eines *verstopften Näselns* (Rhinolalia clausa). Daneben hörte man aber eine leichte Beimischung von der Art des Näselns, die wir als *offenes Näseln* (Rhinolalia aperta) bezeichnen.[1] Was von beiden hier das Wesentlichere und Stärkere war, konnte *nur graphisch festgestellt werden*. Da sich zeigte, dass bei einer Reihe von Lauten, die rein oral gebildet werden müssen (Vokale, Verschlusslaute, Reibelaute), ein mehr oder weniger grosser *Nasendurchschlag* auftrat, so konnte von der Operation der Adenoiden zur Sprachverbesserung nur *dringend abgeraten werden*. Wäre hier die Herausnahme der Adenoiden erfolgt, so würde sogleich eine *ausserordentlich starke Sprachverschlechterung eingetreten sein*. Die adenoiden Vegetationen bildeten hier gleichsam eine Art natürlichen Obturators für die bestehende Insuffizienz des Gaumensegels. Anderseits machten sie keinerlei Erscheinungen weder in bezug auf das Gehör noch in bezug auf die Atmung.

(3) Wie gut man das *Tremulieren* graphisch aufnehmen kann und gleichzeitig die *Ursache des Tremolos* festzustellen imstande ist, zeigt die dritte Kurve. Sie sehen, dass nicht nur die Tonhöhe offensichtlich wechselt, sondern dass dieser Wechsel *gleichzeitig von einer spastischen Bewegung des ganzen Kehlkopfs verursacht ist*. Diese Bewegung war so gering, dass sie nur bei der ersten Untersuchung entging. Bei der graphischen Darstellung trat sie jedoch stark zutage. Ausserdem war die Beziehung zwischen der spastischen Bewegung des Kehlkopfs und der Tonhöhenschwankung im Tremolo sofort objektiv nachzuweisen.

(4) Hier handelte es sich um einen ausserordentlich seltenen *Fall von plötzlich entstandenem verstopften Näseln*, und zwar *auf hysterischer Basis*. Hier hatte allerdings schon die Untersuchung von Nase und Nasenrachenraum der 21 jährigen Frau ergeben, dass organische Hemmnisse nicht vorhanden waren. *Es zeigte sich, dass ausserhalb des eigentlichen Sprechvorganges der Nasenweg frei war*, dagegen mit dem Beginn jedes Sprechversuches das Gaumensegel sich krampfhaft in die Höhe zog und nun den Zugang zur Nase vollständig abschloss, solange der Sprechversuch dauerte. Aus den hier vorliegenden Kurven gewinnt

[1] Beide zusammen ergeben das eigentümliche Timbre, das ich als Rhinolalia *mixta* bezeichnet habe, und das gerade besonders häufig für die vorliegende Frage der operativen Indikation in Betracht kommt.

man ein recht klares Bild dieser hysterischen Störung, die wie gesagt überaus selten ist. Wenigstens habe ich trotz eines reichlichen Materials bisher noch niemals ähnliches gesehen und ebensowenig Herr Prof. Killian, der mir diese Patientin aus seiner Poliklinik zur Untersuchung überwies.

(5) Auf der fünften Kurve sehen Sie, in welcher Weise die *Atmungsbewegung bei Laryngektomierten* sichtbar gemacht wird und zu welchem Zwecke dies geschieht. Die Hauptvorbedingung für die Erwerbung der Pharynxstimme oder Pseudostimme bei Laryngektomierten besteht darin, *dass die bisherige Verknüpfung der Atmungsbewegung mit der Stimmproduktion vollkommen aufhört.* Die Atmung darf nicht während des Sprechvorganges gemacht werden, weil sonst durch die Kanüle resp. die Trachealöffnung ein scharf reibendes Geräusch entsteht, das nicht nur die Hörbarkeit der gesprochenen Konsonanten fast völlig aufhebt, sondern auch für den Patienten überaus ermüdend ist. Bei der Entwicklung der Pharynxstimme muss demnach die gewöhnliche Sprechatmung zunächst vollkommen ausgeschaltet werden. Sie muss von der bisherigen Koordination getrennt, dissoziiert werden. Welche Mühe dies dem Laryngektomierten anfangs macht, sehen Sie aus der Kurve, ebenso wie hier der Laryngektomierte aus der während des Uebens entstandenen Kurve ersah, ob er das von ihm Verlangte richtig machte oder falsch.

(6) Endlich zeige ich Ihnen noch eine Kurve, aus der Sie sehen können, dass *beim Stottern die Störung nicht nur während des eigentlichen Sprechvorganges eintritt,* sondern dass man eine spastische Abweichung auch konstatieren kann, wenn man die Sprechausatmung allein aufnimmt oder irgendeinen Vokal auf eine bestimmte Tonhöhe lang anhaltend geben lässt. Dies ist offenbar noch keine Sprache, sondern erst ein Teil des Sprechvorganges, es ist gleichsam eine aus der sonstigen Koordination herausgenommene Einzelbewegung. Nach den bekannten psychischen Theorien zur Erklärung des Stotterns, die eine ' Koordinationsneurose ' ablehnen und alles Heil in der Psychoanalyse sehen, dürfte dies nur in dem Moment auftreten, wo irgendwelche psychischen Hemmungen beim *Sprechen* auftauchen. Dass das nicht immer der Fall ist, sehen Sie an dieser Kurve. Hier zeigt sich auch während des langen auf möglichst gleichbleibender Tonhöhe gemachten Vokals ein deutliches, spastisches Schwanken ganz unregelmässiger Art (nicht etwa mit dem Pulse zusammenhängend), ein Schwanken, das man mit dem Ohr zunächst kaum wahrnahm. Erst nach Feststellung der Kurve und Ausrechnung der Tonhöhe konnte man es bei besonders darauf hingerichteter Aufmerksamkeit auch mit dem Ohr feststellen.[1]

Zum Schluss möchte ich daher auf diesen Umstand besonders hinweisen. Es zeigt sich oft genug, *dass die experimentell-phonetische Graphik uns geradezu schärfer beobachten lehrt* : Die objektive Darstellung von Erscheinungen, die wir zuerst nicht wahrnehmen konnten oder wegen ihrer Kleinheit übersahen, schärft unsere Beobachtung, indem sie uns auf die Dinge hinweist, die wir sehen oder hören sollen.

[1] Aehnliche Beispiele gegen die Auffassung der Psychoanalytiker könnte. ich Ihnen in grosser Zahl vorlegen.

DISCUSSION

Mr. C. MacMahon (London). I should like to allude to two matters bearing upon the subject-matter of Professor Gutzmann's most interesting paper, and in the few remarks which I have the honour of being allowed to make, I would desire to say very briefly what is in my mind. It seems to me that whatever methods of diagnosis are employed, it is important to remember that some forms of organic mischief in the throat owe their origin to functional causes and that some forms of functional troubles owe their origin to organic causes. I think these facts are of great importance when the curative treatment is considered. Professor Gutzmann has pointed out that, in cases of voice and throat trouble, vocal treatment is a necessary adjunct to medical treatment, and especially is this so in cases of chronic pharyngitis and laryngitis. These ailments are, of course, organic in nature, but when one bears in mind that they are often brought about by improper voice production, it is obvious that they then owe their origin to functional causes. The original cause of the mischief is, to a large extent, the erroneous constriction of the throat muscles above the larynx during phonation. These muscles act wrongly in controlling the air coming from the lungs, and the mucous membrane of the throat consequently suffers very severely and, if this practice is continued, the vocal cords also become affected and serious trouble is established. This action of the throat muscles is radically wrong from a functional point of view, but these muscles can be rapidly thrown out of gear in the act of phonation, especially for the speaking voice, by developing the orbicular muscle of the mouth, by making the lips the first voluntary control of the vibrating air column, by lowering the pitch of the voice, by teaching the correct positions of the main vowel sounds whereby overtones and harmonics are established which amplify the general resonance of the voice enormously; and by obtaining, by well-known movements, an adequate and properly balanced ascent of the air from the lungs. When all these conditions are established, the functional cause of the trouble is removed, and with the aid of medical treatment recovery is very quickly brought about.

The converse is also true: severe laryngitis is often followed by aphonia which is purely functional, and which exists long after all trace of inflammation has left the vocal cords. This trouble can, I submit, be guarded against by proper breathing exercises, by muscular exercises of the throat and by correctly shaping for the main vowel sounds without voice being attempted, so that when speech is again permitted the throat will be free from all constriction and the conditions that suggest speech without phonation will not be present. In other words, functional aphonia will not occur.

In conclusion, I should like to say how greatly I appreciate the privilege of being present when Professor Gutzmann read his paper and of hearing and seeing one whose work is so well known.

Prof. GRADENIGO (Turin). Je désire de remercier vivement M. le professeur Gutzmann pour avoir recommandé notre nouvelle Association internationale de Phonétique expérimentelle. Nous espérons d'avoir beaucoup d'adhérents à cette Association, et la première réunion aura lieu probablement à Milan dans le printemps de 1914.

SECTION XV

RHINOLOGY AND LARYNGOLOGY

INDEPENDENT PAPER

WEITERE STUDIEN ÜBER DIE SOLITÄREN CHOANAL-POLYPEN AUS DEN NASENNEBENHÖHLEN

Von Professor Dr. INO KUBO, Fukuoka, Japan

Seitdem ich im Jahre 1908 durch die vor der Polypenoperation vorgenommene Eröffnung der Kieferhöhle Killians Auffassung über den Ursprung der solitären Choanalpolypen einwandfrei bewiesen und zugleich über die Radikaloperation derselben geschrieben hatte, sind schon 5 Jahre verflossen.

Viele Berichte wurden seither veröffentlicht. Aus der Literatur konnte ich exklusiv meiner eigenen Fälle, 18 Fälle von Ohno, Ito, Katsuya, Nakamura, Azai, Yoshii, Tadokoro, u.s.w. in Japan seit 1909 und ca. über 70 Fälle von Killian, Kelly, Thurm, Lake, Lermoyez, Viollet, Nepveu, Oppenheim, Berini, Lang, Hoffmann, Katz, u.s.w. im Auslande seit 1907 sammeln ; genaue Mitteilungen fehlten leider. Es scheint mir, dass die Ansichten über den Ursprung sowie die Operation noch auseinander gehen.

Ich möchte Ihnen heute auf Grund meiner Erfahrungen von genau untersuchten 36 Fällen während der letzten 6 Jahre etwas mitteilen.

Es ist manchmal recht schwierig, den wahren Ursprungsort in der Kieferhöhle zu bestimmen, selbst wenn man die Höhle eröffnet, da bald der dünne Stiel leicht zerreisst, bald die Höhle von Zysten oder Polypen ganz gefüllt ist, und bald die angeschwollene Schleimhaut besonders auf der medialen Wand durch den Zug des Polypen in die Nasenhöhle prolabiert. Bei einer 36 jährigen Patientin konnte ich bei der Radikaloperation keinen Zusammenhang zwischen dem Polypen und der Kieferhöhle finden und glaubte, dass dieser Fall kein Antralpolyp sei. Aber als die Patientin wieder ins Bett ging, kam das grosse vom Stiel losgerissene Stück in den Schlund und sie verschluckte es. Wie Killian bei Diskussion in Budapest vorgeworfen hat, ist Langes antroskopische Untersuchung von einem Loch an der Fossa canina unvollkommen. Die Feststellung der wahren Ursprungsstelle durch einfache Sondierung ist selbstverständlich nicht hinreichend.

Nach meiner Ansicht scheint es rationell zu sein, die solitären Choanal- oder Retronasalpolypen in 3 Gruppen einzuteilen, je nach dem Ursprungsort, da eine jede eine besondere klinische Bedeutung hat; 1. Antrochoanalpolypen: solche aus der Kieferhöhle; 2. Sphenochoanalpolypen: solche aus der Keilbeinhöhle, und Pseudochoanalpolypen: solche aus anderen Orten der Nasenhöhle. Unter typischen Choanalpolypen versteht man Antro- und Sphenochoanalpolypen. Unter 36 Fällen fand ich 27 Antro- oder Maxillochoanalpolypen, 3 Sphenochoanalpolypen und 6 Choanalpolypen unsicheren Ursprungs. Einige Exemplare, die ich vom fernen Osten über das Meer getragen habe, zeige ich heute den Anwesenden.

Die solitären Choanalpolypen kommen gewöhnlich einseitig, wie Killian betonte, aber ich fand Antrochoanalpolypen doppelseitig bei 2 Kranken. In diesen Fällen war der Polyp auf einer Seite bedeutend kleiner. Kleinere Sphenochoanalpolypen, die ich heute nicht mitteile, sind gewöhnlich doppelseitig.

Die Choanalpolypen betreffen meist jüngere Individuen, wie Brown Kelly darauf hingewiesen hat. Ich fand 15 Fälle im 10.–20. Lebensjahre, 10 im 21.–30. Lebensjahre, und 10 im 36.–58. Lebensjahre. Sehr auffallend ist es, dass sie oft vor der Pubertätszeit vorkommen. Ohnos Fall betraf ein 7jähriges Mädchen, das schon im 3ten Lebensjahre Polypenoperation bekam.

Sie rezidivieren sehr oft, wie die Autoren hie und da bemerken; im Jahre 1907 entfernte ich z. B. einen 47 cm langen, linksseitigen Antrochoanalpolypen bei einem 55 jährigen Manne mit kalter Schlinge und musste im Jahre 1909 wieder wegen des Rezidivs von Fossa canina aus radikal operieren.

Die Antrochoanalpolypen sind meist von Sinusitis chronica begleitet— 25 mal unter 36 Fällen (69 Prozent). Die betreffenden Kieferhöhlen sind meist mit Tochterpolypen oder Zysten gefüllt — 19 mal bei 26 radikaloperierten Fällen (73 Prozent). Daraus versteht man, dass die Choanalpolypen als Folge der abgelaufenen oder noch vorhandenen Sinusitis zustande kommen.

Die Ursprungsstelle der Antrochoanalpolypen befindet sich meist auf der *hinteren* (10 mal), dann *unteren* (8 mal), medialen (7 mal), lateralen (6 mal), oberen (3 mal) und vorderen (1 mal) Wand. In 2 Fällen war sie unsicher. Akzessorische Mündung der Kieferhöhle ist dabei meist gross, wegen der mechanischen Druckausübung des Polypenstiels.

Was die Sphenochoanalpolypen (Kubo) anbetrifft, konnte ich nachher aus der Literatur einige höchst wahrscheinliche Fälle finden, z. B. solche von Massei (1 Fall), Moure (2 Fälle) u.s.w. Sie sind meist kurz gestielt, kommen aus der Keilbeinhöhle und hängen über den unteren Ostiumrand. Diagnose gegenüber den typischen Nasenrachenfibromen ist manchmal sehr schwierig.

Was die Operation angeht, so bleibe ich noch heute auf dem früheren Standpunkte, dass die Polypen radikal mit dem Mutterboden zusammen

entfernt werden müssen; — solche aus der Kieferhöhle samt der Kieferhöhlenschleimhaut und solche aus der Keilbeinhöhle ebenfalls mit der Keilbeinhöhlenschleimhaut zusammen, da sie gewöhnlich mit Sinusitis kombiniert und von Zysten oder Polypen begleitet sind und leicht rezidivieren. Es scheint mir merkwürdig, dass die neuen Publikationen noch Langes Haken als die einzige Waffe für die Operation zu betrachten scheinen. Selbst bei der Operation der gewöhnlichen Nasenpolypen müssen manchmal die Ethmoidalzellen oder andere Nebenhöhlen ausgeräumt werden, sonst ist das Rezidiv nicht gesichert. Die solitären Choanalpolypen sind meist Antral- oder Sphenoidalpolypen und gewöhnlich mit Sinusentzündung kombiniert und von Zysten oder Polypen begleitet. Es scheint natürlich noch solche anderen Ursprungs zu geben, aber sie sind sehr selten, wenn die Untersuchung sorgfältig unternommen wird.

Die uralte Methode, das Angeln und Abreissen des Polypenstiels, ist selbstverständlich sehr einfach, aber sie ist eine blinde und primitive. Das Abreissen am Stiel ist sogar zeitweise sehr gefährlich. Ein illustriertes Beispiel teile ich Ihnen mit. Als ich im Jahre 1908 bei einem 25 jährigen kräftigen Manne einen grossen linksseitigen Antrochoanalpolypen mit kalter Schlinge vom Stiel aus entfernte, kam der Patient mitternachts wieder zu mir, voll Blut auf totbleichem Gesicht. Blutung befand sich im Sinus maxillaris und liess sich durch Nasentampon nicht stillen. Am nächsten Tage wurde der Patient nach meiner Angabe radikal operiert. In diesem Falle wurde die Höhlenschleimhaut beim Abreissen von der Grundlage abgehoben und teilweise zerrissen. Seither operiere ich Antrochoanalpolypen als Regel radikal nach meiner Methode. Die Spezialisten in Japan operieren gewöhnlich nach mir und ebenso im Auslande, wie Lake, Katz, Brown Kelly, Killian, etc. Die Eröffnung der Kieferhöhle von Fossa canina aus ist heut zu Tage als eine kleine Operation zu betrachten. Aber selbstverständlich muss eine genaue Untersuchung der Ursprungsstelle vorangehen.

DISCUSSION

Dr. Brown Kelly (Glasgow). I believe that choanal polypi, or naso-antral polypi, as I prefer to call them, are comparatively common. When there is a single polypus in the posterior part of the middle meatus, on snaring which a flow of translucent watery fluid takes place, one should always think of a naso-antral polypus.

I might point out that in these cases transillumination is often brighter on the affected side than on the healthy side.

I have opened the maxillary sinus in a number of these cases, and in almost all I have found it filled with a cyst. After loosening this from the antral wall the whole growth is best removed by catching it with a bent snare in the naso-pharynx and pulling it out through the mouth. In all

my cases there has been an ostium accessorium, through which the growth has come from the antrum. Some years ago I examined the antra of 100 cadavers, and I was then surprised to find that when the mucous membrane was cystic an ostium accessorium, as a rule, was present. I therefore think that the naso-antral polypus begins as a cyst of the antral lining membrane, and when the cyst fills the cavity a part of it prolapses through the accessory opening and develops into what appears to be a polypus.

I would also like to mention that I have had a case of the analogous condition in the sphenoidal sinus—a naso-sphenoidal polypus.

Dr. MARSCHIK (Vienna). In spite of the experience of Professor Kubo I would advise that the old simple method of endo-nasal, or better, peroral extraction of the polyps be tried once or twice. This method is very simple and easy, owing to the thin and friable pedicle of the polyp. The radical operation should be reserved for cases of recurrence. In Vienna we have not observed a hæmorrhage such as referred to by Professor Kubo.

Dr. D. MCKENZIE (London) had drawn attention some years ago to the occasional occurrence of gangrene in the choanal segment of the hour-glass-like antro-choanal polyp. It was evidently due to constriction of the vessels and cessation of the circulation arising from ' kinking ' or doubling over of the polyp at its isthmus, aided doubtless by the pressure upon the sharp edge of the osseous opening.

REPLY

Professor KUBO (Japan). Ich danke meinen verehrten Kollegen für die interessante Diskussion. Ich bleibe doch auf dem Standpunkte, dass man bei solitären Choanalpolypen die Ursprungsstelle klar vor Augen bringen und den Mutterboden gründlich ausräumen muss.

SECTION XV

RHINOLOGY AND LARYNGOLOGY

INDEPENDENT PAPER

SUR UNE MÉTHODE TRÈS UTILE POUR GUÉRIR L'APHONIE HYSTÉRIQUE

Par M. le Professeur CITELLI, de Catane

L'aphonie hystérique sans doute est une de ces maladies du larynx où les nombreuses et utiles méthodes thérapeutiques que nous avons à présent à notre disposition peuvent dans certains cas échouer complètement. Nous ne pouvons être sûrs de guérir chaque malade atteint d'aphonie hystérique qui se présente à nous, parce qu'aussi les moyens plus efficaces contre cette affection (comme le massage vibratoire électrique, la régulation de la respiration, les exercices vocaux, la pression manuelle sur le larynx, la faradisation et aussi la méthode de Seifert, c'est-à-dire la phonation pendant l'extension forcée du cou) peuvent, dans quelques cas, rester sans effet. Pour cette raison chaque nouveau procédé utile pour cette maladie doit être accueilli avec satisfaction, car la possibilité d'avoir des échecs peu agréables dans la pratique diminue.

Ma méthode, bien différente de la pression manuelle ordinaire sur le larynx ou sur les grandes cornes de l'os hyoïde, consiste dans la pression forte, douloureuse et soudaine (complètement imprévue par le malade) faite avec les deux premiers doigts de la main droite sur les grandes cornes de l'hyoïde ou de la cartilage thyroïde.

Quand j'ai diagnostiqué une aphonie hystérique, sans que le patient puisse penser à ce que je fais, je fixe avec la main gauche la nuque du malade et, avec l'index et le pouce de la main droite, je saisis les parties latérales de l'os hyoïde ou de la thyroïde, j'y exerce rapidement une pression forte, passablement douloureuse, et, pendant que le patient, presque effrayé, cherche à crier pour me dire de suspendre ma pression, je le tempête de demandes faites à haute voix, rapidement, l'une après l'autre, surtout pour lui demander comment il se porte, s'il va mieux, ce qu'il avertit, etc. Alors le malade, par l'émotion et sans avoir le temps de penser (c'est-à-dire sans ressentir plus l'influence de son état spécial de conscience, pour lequel il ne peut ou ne sait plus phoner) répond avec sa voix normale. Alors, il est guéri, spécialement si on continue pendant quelque temps à l'interroger pour le faire continuer à parler, cela pour lui

faire démentir complètement son état ancien de conscience, qui le rendait aphone.

Dans ma méthode, donc, la pression sur l'hyoïde ou la thyroïde, pas légère et prolongée comme dans l'ordinaire pression sur le larynx, n'a pas le but, comme dans ce cas, de soutenir les cordes vocales pour les aider à donner la phonation normale ; mais spécialement il agit par un autre mécanisme, comme nous l'avons vu ci-dessus.

J'ai traité jusqu'à présent avec cette méthode quatre malades atteints d'aphonie hystérique laquelle, dans un cas (une vieille demoiselle), durait pendant deux ans, et j'ai obtenu à la première séance et rapidement toujours la guérison. J'espère donc qu'elle pourra rendre des grands services dans la pratique : pour cela j'ai cru utile la faire connaître à mes confrères.

[Tuesday Afternoon, August 12]

SECTION XV

RHINOLOGY AND LARYNGOLOGY

INDEPENDENT PAPER

CONTRIBUTIONS A LA LARYNGOSTOMIE

Par M. le Dr. SARGNON, de Lyon

Dans cette communication, nous voulons mettre au point les travaux depuis nos dernières communications à la Société française de Laryngologie, mai 1912, et au Congrès espagnol de Bilbao, août 1912.

Depuis cette époque, il a paru d'assez nombreux travaux à l'étranger et en France. Signalons notamment, en Belgique, un cas nouveau dû à van der Calseyde, et présenté à la Société belge le 16 février 1913. Il s'agit d'un cas de lupus sténosant, guéri par cette méthode avec dilatation au moyen d'un tube en verre en forme de L.

Tout récemment Broeckaert (*Presse belge* No. 3, 1913 : 'Quelques interventions pour tumeur de la trachée'), signale un cas de guérison chez un enfant par trachéo-laryngostomie pour papillomes rebelles du larynx et de la trachée.

En Russie, nous avons eu connaissance de plusieurs cas présentés en 1911, le 16 décembre, à la Soc. Oto-Laryng. d'Odessa, et qui nous avaient échappé. A cette société Pensky fit une communication (au sujet de la laryngostomie) ; Hecheline ('Sur trois cas de laryngostomie', analyse in Chauveau, janvier-février 1913, p. 229), publie trois cas : l'un de guérison pour une sténose cicatricielle chez un enfant rétréci à la suite d'une trachéotomie pour corps étranger, et deux insuccès pour des périchondrites après fièvre typhoïde et croup dans l'enfance avec trachéotomie.

Heryng, dans son remarquable 'Traité de Laryngologie', 1912, à propos de la laryngostomie, signale son dilatateur caoutchouté avec épiglotte artificielle, et réclame la priorité pour la dilatation caoutchoutée, avec engainement de la canule, procédé décrit et imaginé en France par Fournier, de Marseille, et publié dans la Revue de Moure, 1908 ('Sténose laryngée chez un enfant tubarde-canularde, laryngostomie par Fournier et Sargnon' ; voir aussi contribution à l'étude de la dilatation caoutchoutée dans la laryngostomie par Fournier, Société française de Laryngologie, mai 1909). Ce procédé mérite donc bien, à notre avis, de porter les deux noms de Fournier-Heryng.

Tout récemment Ivanow, de Moscou, a publié en 1912 (*Zeitschrift für Lar.*, 1912 ; extrait de la clinique Basanova ; ' L'extirpation de l'aryténoïde dans la sténose du larynx '), un cas très remarquable d'opération d'une sténose pour paralysie bilatérale des abducteurs par la laryngofissure avec résection aryténoïdienne et dilatation consécutive. Cette pratique, tirée dans une certaine mesure de l'art vétérinaire, nous semble avoir été faite pour la première fois par cet auteur ; nous l'avons suivie.

En Hollande, Posthumus Meyjes, d'Amsterdam (20e Congrès de la Soc. néerlandaise de Laryngologie, nov. 1911, analyse in Chauveau, janvier-février, 1913), publie un cas de laryngostomie : Enfant, 4 ans, diphtérie, trachéotomie, canulard par bourgeonnements, laryngostomie, port quelques semaines d'un tube en caoutchouc en T de Killian ; fermeture rapide de la plaie. Par contre, dans un cas de sténose trachéale, chez un adulte canulard depuis dix ans, van Stockum (Soc. hollandaise de Chirurgie, avril 1912, analyse in Chauveau, septembre-octobre 1912, p. 551), guérit rapidement son malade par la résection trachéale, la suture des moignons, et la trachéotomie plus basse.

C'est surtout en Italie et en France que se sont faits le plus grand nombre de laryngostomies. Signalons notamment un article de Della-Vedova (*La Pratica ot.-rin.-lar.*, octobre 1912 ; Congrès de la Soc. italienne de Lar. de Venise, septembre 1912 : ' Quelques renseignements utiles sur un cas de sténose chronique du larynx '), qui, à propos d'un cas de sténose chondrale et muqueuse spécifique, signale l'insuccès de la laryngo-fissure, de la dilatation interne, de la dilatation caoutchoutée avec laryngostomie, la possibilité du tubage prolongé combiné à la laryngostomie ; il conclue à l'utilité de la dilatation interne, et réserve la laryngostomie surtout aux cas graves chez les jeunes atteints de cicatrices endo-muqueuses ; il insiste sur la possibilité de combiner la laryngostomie et la dilatation interne.

Au Congrès de Venise de la Soc. italienne de Laryng., sept. 1912, analyse in Chauveau, janvier-février 1913, p. 175, Genta, de Gènes, apporte une contribution très intéressante, quoique négative, à la laryngostomie dans le cancer (' Sur le résultat négatif de la Laryngostomie dans deux cas de tumeur maligne du larynx ') ; il s'agissait d'épithélioma circonscrit de la corde vocale droite sans infiltration diffuse apparente ; la laryngostomie suivie d'excision de la tumeur et d'interventions successives sur le larynx, ne réussit pas à prévenir la récidive dans les deux cas.

Bilancioni, de Rome, au Congrès de la Soc. italienne de Chirurgie, 7 et 10 novembre 1912 (' Sur la technique de la Laryngostomie ' ; analyse in Chauveau, janvier-février 1913, p. 243), insiste à nouveau sur les questions de priorité qui semblaient pourtant bien établies et bien précisées au Congrès de Budapest de 1909 pour les deux écoles italienne et française ; rappelons, en effet, que dans la discussion le professeur Massei nous a remercié d'avoir rendu justice à l'école italienne. Bilancioni décrit la technique suivie à Rome par Ferreri et conseille de rééduquer

chez ses malades la respiration nasale, car chez eux le type respiratoire est très modifié.

En France, signalons la thèse de Monlaur (Paris, 1912, chez Oberthür, Rennes ; analyse in Chauveau, décembre 1912, p. 922 ; 'De la chondro-périchondrite tuberculeuse du larynx') où il étudie entre autre le traitement des sténoses. Dans sa thèse (Paris 1912–13, 'De la laryngostomie dans le traitement des papillomes diffus chez l'enfant'), thèse inspirée par Sébileau, Fontaine étudie l'historique de cette question, et déclare que si Ruggi est incontestablement le premier à avoir appliqué la laryngostomie dans le traitement des papillomes rebelles 'c'est un devoir de justice d'associer à son nom ceux de Beco et Sargnon, qui, dès la première heure, essayèrent d'attirer l'attention des laryngologistes sur ce nouveau mode de traitement.' A signaler entre autres l'opinion de Sébileau et de l'auteur qui après la guérison du papillome font la plastique au bout de trois mois. L'auteur rapporte dans sa thèse 22 observations dues à Ruggi, Beco, Sargnon et Vignard, Calamida, Wildenberg, Ferreri, Viannay, Jauquet, Gradenigo, Moure, Guisez, Baumgarten, Sébileau et Halphen (3 cas) ; le seul cas de mort est celui publié par Sargnon et Vignard, où la petite malade a quitté l'hôpital emmenée par ses parents pour question budgétaire. Elle est restée canulée et est morte faute de soin de la canule ; cet incident est donc totalement en dehors de la méthode. Fontaine conclut qu'en cas d'échec par les méthodes endo-laryngées endoscopiques ou autres, il faut pratiquer la laryngostomie. Tout récemment (*Annales des Maladies de l'Oreille*, 4e livraison, 1913, p. 342 : 'Quatre cas de papillomes laryngés diffus récidivant de l'enfance, traités par la laryngostomie'). Halphen et Fontaine, outre les trois cas déjà publiés dans la thèse de Fontaine, signalent un 4e succès. Personnellement nous trouvons la formule opératoire de Sébileau, Halphen et Fontaine peut-être un peu absolue, car la simple mise au repos de l'organe par la trachéotomie a donné à de nombreux auteurs et à nous-mêmes de beaux succès. Ce n'est qu'en cas d'insuccès, après plusieurs mois de mise au repos, que nous pratiquons la laryngostomie.

Signalons la communication de Pautet, à la Société française de Laryngologie, mai 1913, et le bon résultat obtenu par la laryngostomie pour une chondrite tuberculeuse. L'importante communication de Molinié au même congrès ('Modifications au pansement de la Laryngostomie') a été publiée à nouveau dans le *Larynx*, mai-juin 1912, et le *Recueil de Laryng.* de Nepveu, décembre 1912). Personnellement nous avons repris la question à la Société française de Laryngologie, mai 1912 ('Contribution à la Laryngostomie'), et au Congrès espagnol de Bilbao, août 1912, où la question fut suivie de discussion (*Arch. de Laryng.* de Botey, sept.-décembre 1912).

Nous avons aussi avec Vignard présenté des cas de laryngostomie à la Soc. des Sciences méd. de Lyon (2 juillet 1912, in *Lyon Méd.*, 17 novembre 1912, et 15 janvier 1913). Signalons encore au Congrès de Laryngologie, mai 1913, la communication de Moure et Richard ('Considérations cliniques sur quelques cas de sténose laryngo-trachéale').

Technique

Depuis deux ans nous avons peu modifié notre technique. Signalons cependant :

1° L'utilité de la suture du larynx à la peau avant d'exciser l'obstacle, car les fils servent de tracteurs.

2° La suppression de la dilatation caoutchoutée, et l'emploi exclusif de la gaze, les dix premiers jours à la période de sphacèle ; ce sphacèle est aussi très diminué par l'emploi de la pommade au rouge neutre d'aniline à 4 pour cent.

3° Il faut, dans la mesure du possible, chercher à raccourcir la longue durée des pansements et de la dilatation. Grâce à l'emploi de la pommade rouge, et surtout aux modifications apportées à la dilatation, on peut arriver à réduire sensiblement la durée du traitement, surtout pour les sténoses cicatricielles chez l'enfant. C'est ainsi que dans nos derniers cas, pour des soudures cricoïdiennes tubulaires, trois mois de dilatation ont suffi. Par contre, si, outre la soudure, il y a aplatissement antéro-postérieur, il faut créer à la longue avec le caoutchouc un nouveau larynx dans la cicatrice aplatie. Le minimum de temps que nous ayions mis est de 6 à 7 mois en pareil cas ; au début il nous fallait un an.

Si nous pouvons fixer approximativement une limite de temps pour la cure des sténoses cicatricielles chez l'enfant, il n'en est malheureusement plus de même pour les sténoses chondrales. Nous sommes tout à fait d'accord sur ce point avec les auteurs qui s'en sont occupés, notamment Ivanow, Marschik, etc. La difficulté provient alors des hypertrophies considérables des parties molles qu'il faut extirper au cours de l'opération, et souvent aussi dans la suite, soit à la pince coupante, soit à l'anse froide, soit par des cautérisations au galvano, au nitrate d'argent, à l'acide chromique. Parfois aussi il y a lieu dans ces cas d'extraire en une série de séances des bourgeons développés au niveau du coude ou de la portion descendante de la canule. Il en résulte parfois des réactions inflammatoires, qui arrêtent momentanément la dilatation, des bourgeons qu'il faut enlever, des abcès avec fistule qu'il faut ouvrir. Dans deux cas déjà signalés au Congrès de Bilbao nous avons eu à extraire des séquestres du cricoïde et du thyroïde, l'un chez un tuberculeux (Pautet et Sargnon) l'autre chez un syphilitique. Au Congrès de 1913 de la Société française, Moure a signalé un séquestre du cricoïde tombé dans la trachée.

Signalons aussi la transformation heureusement rare de la chondrite ou des papillomes rebelles chez l'adulte en néoplasmes malins, Moure, Broeckaert, Marschik, nous-mêmes, avons observé dans cet ordre d'idées des cas de néoplasmes anormaux, méconnus avant l'opération, ou dégénérés au cours de la dilatation. Nous discuterons plus longuement cette question à-propos des complications.

4° Malgré l'opinion de nombre d'auteurs, notamment de Ferreri, de Viannay, de Sébileau et de ses élèves, Halphen et Fontaine, nous sommes

d'avis de garder plusieurs mois les malades en surveillance plus ou moins immédiate avant de les suturer, et dans les cas graves de faire une suture pas tout à fait complète ; c'est peut-être un excès de prudence. De plus quand la dilatation est supprimée, nous croyons utile chez le canulard très spasmodique, surtout chez le tout petit enfant, ou de maintenir l'orifice béant avec un tube de caoutchouc en T, ce qui n'est pas toujours facile chez le petit, car le tube s'oblitère facilement, ou mieux de mettre la nuit une toute petite canule de sûreté. D'ailleurs, une surveillance médicale très sérieuse est de rigueur les premiers jours du décanulement. Pour avoir cru à un résultat très complet chez une fillette de cinq ans atteinte de soudure avec aplatissement avec réfection du larynx en sept mois de dilatation, et n'avoir pas pris cette précaution, nous avons perdu cette malade à l'hôpital par spasme brusque, et aussi, il faut bien le dire, par insuffisance de la garde-malade trop novice. Il suffisait simplement d'écarter à temps les lèvres de la stomie pour empêcher l'enfant de mourir.

5° Dilatation

La question du mode de dilatation, suivant les cas, est primordiale. Dans des cas, exceptionnels d'ailleurs, l'idée a été émise de maintenir l'écartement des lèvres sans faire de dilatation, tandis que dans l'immense majorité des cas la dilatation est nécessaire. Elle se pratique de façons differentes suivant les circonstances. Pour l'immense majorité des malades, le port de la canule est nécessaire ; très rarement on peut faire de la dilatation tout en supprimant la canule.

Nous adoptons donc comme grandes divisions :

A. Maintien de la Stomie sans Dilatation Endo-Laryngée.
B. Dilatation Endo-Laryngée.
  1° *Avec maintien de la canule.*
    (*a*) Dilatation douce (gaze coton).
    (*b*) Caoutchouc.
    (*c*) Autres procédés de dilatation.
  2° *Sans maintien de la canule.*

A. Maintien de la Stomie par Dilatation des Bords, sans Dilatation Endo-Laryngée

A la Société française de Laryngologie, mai 1912, après la communication de Molinié, Bourguet a relaté un cas de soudure syphilitique des cordes où il a fait la laryngostomie sans aucune dilatation caoutchoutée avec écartement seul des lèvres de la stomie. Le malade a bien guéri. C'est là une application très ingénieuse et très pratique de la laryngostomie avec simplification énorme des pansements ultérieurs. Mais une pareille conduite n'est applicable qu'aux lésions membranoïdes. Il suffit alors de maintenir l'écartement trachéo-laryngé pour obtenir la guérison ; en pareille circonstance une laryngotomie interne, une laryngo-fissure avec dilatation consécutive par le caoutchouc ou le tubage, peuvent

donner aussi de bons résultats. Signalons aussi la possibilité d'obtenir de bons résultats par la nouvelle canule dilatatrice qui, dans le service du professeur Sébileau, a déjà donné des succès.

Mais la laryngostomie a cette grande supériorité de permettre la surveillance directe et facile de la lésion. C'est donc là une application extrêmement intéressante de la laryngostomie.

B. Dilatation Endo-Laryngée

1° *Avec maintien de la canule*

(*a*) *Dilatation douce.* — Elle se pratique le plus souvent avec un rouleau de gaze coupée en biseau, à l'extrémité inférieure, et enduite, soit de vaseline simple, soit de pommade à l'argyrol ; nous utilisons la pommade au rouge neutre d'aniline. Nous avons employé ce mode de dilatation, préconisé par l'école italienne, pour les lésions papillomateuses, les sténoses cicatricielles chondrales trop enflammées, d'une façon, soit continue, soit intermittente. L'école italienne, avec Ruggi, Canepele, a utilisé la gaze entourée de gutta, nous avons essayé ce procédé qui nous a donné toute satisfaction dans les sténoses enflammées. Nous enduisons la gutta de pommade rouge. La gaze seule, si elle a l'avantage d'être douce, a aussi l'inconvénient de pénétrer dans les tissus, et dans les cas très enflammés d'amener un peu de bourgeonnement ; elle marque sa place dans les parties molles. Entourée de gutta, elle n'adhère plus aux tissus ; il y a moins de bourgeonnement et de suintement hémorragique lors des pansements ; l'épidermisation est bien plus rapide. En un mois nous avons obtenu de bons résultats d'épidermisation dans nos derniers cas.

Cette épidermisation est surtout facilitée par la diminution de la fréquence des pansements. En effet, la gaze mise seule s'imbibe très vite des liquides de la bouche et des suintements de la plaie ; il faut la renouveler tous les jours les premiers temps. Avec la gaze entourée de gutta, il n'y a pas de fermentation, ou tout au moins elle est très tardive. Les pansements peuvent facilement se faire tous les 2, 3 ou 4 jours, suivant l'époque de la dilatation, excepté, bien entendu, pendant la période de sphacèle.

Ferreri utilise constamment le coton comprimé, qui augmente de volume au contact de la plaie et amène une grosse dilatation. Nous l'avons rarement employé, et préférons la gaze entourée de gutta.

Bien entendu nous réservons ces procédés de dilatation douce aux cas cicatriciels qui ne sont pas très serrés (membrane), ou pour ceux qui, quoique très serrés, sont momentanément enflammés, ou encore quand la dilatation est absolument secondaire, comme dans les papillomes. Pour les sténoses très serrées, nous restons absolument fidèles à la méthode caoutchoutée, que nous avons d'ailleurs notablement modifiée ces derniers temps.

(*b*) *Dilatation caoutchoutée.* — Brünings, et après lui Killian, ont

utilisé en maintes circonstances comme dilatation, notamment pour le nez, des lanières de caoutchouc éponge qui sont très douces. Killian a employé ce procédé comme dilatation du larynx. Nous l'avons utilisé aussi, mais momentanément.

On obtient une dilatation rapide, mais, outre l'impossibilité de la stérilisation absolue, nous reprochons au caoutchouc éponge de fermenter vite, et surtout de faire mascérer les tissus. Il faut donc changer les lanières tous les jours, ou tous les deux jours. Peut-être pourrait-on atténuer cet inconvénient en enveloppant la lanière de gutta, mais alors la dilatation serait moindre.

Dans les sténoses très serrées le tube biseauté de caoutchouc, que nous avons décrit avec Killian, conserve toute son importance, sauf les premiers jours après la laryngostomie, et, quand il survient, une poussée inflammatoire. Il est fermé en haut pendant les premiers temps de la dilatation, soit avec de la gaze seule, soit mieux avec de la gutta.

Parfois la gaze obturatrice s'échappe dans la bouche, inconvénient qui n'existe pas avec la gutta qui est attachée avec un fil. Dans les cas difficiles très serrés, nous utilisons le procédé de Fournier, canule engaînée dans le caoutchouc. Il donne une fixité absolue ; il est habituellement bien supporté. On peut, soit l'utiliser ouvert, ce qui, avec une canule fenêtrée permet la respiration buccale, soit l'employer l'extrémité supérieure étant obturée par de la gutta. Par ce procédé les gros pansements sont espacés de trois à quatre jours. Dans certains cas exceptionnels de dilatation très dure nous avons utilisé momentanément le drain bourré de gaze de haut en bas pour lui donner une rigidité extrême. Ce procédé rend des services pour créer un trajet artificiel, mais au bout de quelques jours il doit être abandonné, car il est très irritant et fait bourgeonner. Nous l'avons utilisé, soit avec le drain biseauté libre, soit avec le drain engaîné de Fournier.

Le procédé de Cheval et Labarre (plusieurs caoutchoucs superposés et attachés à la canule) a permis à Egger, dans un de nos cas, de venir à bout d'une soudure grave avec aplatissement. Les auteurs de ce procédé emploient le caoutchouc attaché à la canule, mais non engainé dans cette canule ; il y aurait, croyons-nous, avantage à l'utiliser ainsi.

Dans les cas pas très serrés, et aussi dans les cas inflammatoires, surtout à la période de sphacèle du début, nous avons utilisé dès 1906 le caoutchouc entouré de gaze. Melzi et Cagnola ont employé semblable méthode, qui est bonne, très douce, mais a l'inconvénient de s'imprégner dans les tissus, de faire bourgeonner, et saignoter parfois, surtout lors de l'ablation du pansement. Nous avons observé aussi chez l'adulte des chatouillements très pénibles du côté de l'orifice supérieur du larynx, amenant des quintes de toux, et des envies de vomir. Tous ces inconvénients ont disparu, en entourant le caoutchouc et la gaze d'une couche de papier à la gutta très mince. C'est le procédé que nous utilisons actuellement, et que nous recommandons tout particulièrement. Il donne une bonne dilatation, facilite l'épidermisation, et n'amène ni

bourgeonnement, ni suintement hémorragique ; le pansement ne fermente pas, et peut rester facilement trois ou quatre jours en place. Les pansements intermédiaires sont superficiels. Naturellement la gutta est enduite de pommade rouge, ou de vaseline. C'est là actuellement pour nous le procédé de choix dans la dilatation des sténoses cicatricielles et chondrales. Si l'on utilise le caoutchouc engainé dans la canule, on peut garnir, plus ou moins, de gaze seule ou mieux de gaze et de gutta, la partie libre du caoutchouc. On aura ainsi les avantages de la fixation du caoutchouc dilatateur, combinés à la douceur épidermisatrice du pansement à la gutta.

(*c*) *Autres dilatateurs.* — On peut, à l'exemple de Hope, de Chiari et Marschik, utiliser des dilatateurs en ébonites, munis d'une ailette pour maintenir la stomie. Ces deux derniers auteurs avaient employé auparavant des tubes métalliques biseautés, soit fermés, soit ouverts, suivant l'époque de la dilatation ; ils n'utilisent d'ailleurs plus ce procédé. Melzi et Cagnola, dans les cas très serrés, ont préconisé des dilatateurs durs en bois, ou même en os. On pourrait même à la rigueur se servir de dilatateurs métalliques de Thost, ou d'Uchermann.

2° *Sans maintien de la canule*

Heryng depuis plusieurs années a utilisé un tube de caoutchouc avec épiglotte artificielle. Cette disposition très ingénieuse a été montrée par Siems à la Société française de Laryngologie, mai 1912. Un fil mobilisant la partie haute du drain ferme et obstrue le tube pendant l'alimentation. Le même dispositif peut aussi s'utiliser avec la canule. C'est surtout Molinié qui, à la Société française de Laryngologie, mai 1912 (*Le Larynx*, mai-juin 1912), a cherché à supprimer de très bonne heure la canule en utilisant un drain à ailette de caoutchouc souple, drain qui peut s'utiliser aussi avec la canule. Quand le malade est porteur du drain, sans la canule, il y joint une soupape antérieure, véritable porte dans le drain, qu'un simple fil permet d'ouvrir ou de fermer à volonté. Parfois même cet auteur adapte un fil qui, simultanément, ferme l'extrémité supérieure bivalve du drain et ouvre la soupape de sûreté. Les figures parues dans le journal *Le Larynx* montrent mieux le fait que toute description.

A la Société française de Laryngologie, Moure a montré le drain de caoutchouc, qu'il utilise sans canule, drain qui est muni d'une double ailette pour maintenir plus facilement la stomie dans toute sa hauteur ; l'ailette est double pour qu'elle puisse se replier l'une contre l'autre, et faciliter ainsi l'introduction du drain.

Mode d'attache du fil. — Le mode d'attache du fil a été très perfectionné ces derniers temps, et pour nous il a une grosse importance. Avec l'ancien fil médian, dont les deux chefs sont attachés aux deux ailettes de la canule, plus ou moins lâchement d'ailleurs, on observait parfois des inconvénients, le déplacement du drain, exceptionnellement en bas, généralement en haut du côté du pharynx, la section des bords de la plaie

par le fil qui retarde d'autant l'épidermisation. Actuellement nous utilisons toujours le fil médian, mais nous l'entortillons entre le drain et la canule pour éviter la coupure des bords de la stomie, et nous l'attachons après avoir fait un double tour fixateur entre les deux plaques externes de notre canule. On a ainsi une fixation parfaite du drain qui, maintenu en avant par le pansement, ne peut plus se déplacer. Quand nous utilisons le caoutchouc seul, obturé en haut par de la gutta, nous fixons cette gutta à l'aide d'un fil supérieur ; si on obture l'extrémité supérieure du drain avec de la gaze, il faut passer un fil de sûreté à travers ce bouchon de gaze.

Nous avons adopté dans les cas difficiles, et actuellement dans presque tous les cas, le fil d'extraction préconisé par Roure en 1911. Ce fil, placé un peu au-dessous du fil de fixation à la canule, n'est pas attaché à la canule ; l'extraction est ainsi bien simplifiée, et jamais hémorragique.

Indications. — Elles découlent de la maladie causale et des circonstances diverses comme l'âge, l'état général, l'état respiratoire.

(*a*) *Indications causales.* — Elles sont cicatricielles, endo-muqueuses, ou bien affectent le type de chondrite et de périchondrite.

Pour les sténoses cicatricielles, l'indication opératoire est des plus nettes. Les cas bénins, où la respiration est peu compromise, sont justifiables de la dilatation interne, ou des opérations endo-largyngées par voie directe ou indirecte.

Les sténoses serrées et par conséquent les soudures tubulaires, ou avec aplatissement, sont justifiables de l'opération externe, variable suivant les auteurs. Boulay et Pieniazek utilisent la laryngo-fissure avec dilatation caoutchoutée ; Killian utilise parfois la dilatation avec l'éponge de caoutchouc. Schmiegelow, (*Arch. de Fraenkel*, 1911, p. 512) utilise sa méthode de dilatation translaryngée. Pour nous, comme d'ailleurs pour l'école italienne et l'école belge, la laryngostomie est en pareil cas l'opération de choix surtout chez les tubards canulards.

La dilatation des sténoses chondrales réussit parfois par voie interne ; mais si la sténose est un peu serrée, l'opération externe, notamment la laryngostomie, s'impose. La dilatation interne a échoué deux fois entre nos mains, et les malades ont dû être laryngostomisés.

Cependant, nous devons reconnaître que dans la chondrite syphilitique, à cause du bourgeonnement intense, des épaississements pachydermiques, des gommes, des fistules, des séquestres, qui existent avant la laryngostomie ou apparaissent au cours de la dilatation, le traitement des sténoses spécifiques est des plus difficiles. Dans un de nos derniers cas, nous avons même observé une transformation néoplastique maligne, dont la genèse est la suivante :

Homme de 53 ans, spécificité très probable, peut-être héréditaire, sténose laryngée sous-glottique grave, trachéotomie, amélioration par le 606, bourgeonnements sus et médiocanulaires, intenses, insuccès de la dilatation caoutchoutée interne pendant un mois, et des ablations de bourgeons ; laryngostomie qui montre un épaississement intense des parties molles, avec cartilage ossifié ; extirpation immédiate et au cours

de la dilatation des obstacles ; dilatation caoutchoutée seule, ou avec de la gaze, pendant sept mois, au cours de laquelle il y eut élimination de deux séquestres du thyroïde avec chondrite à droite et fistule cutanée survenue à la suite d'une angine grave fébrile. Décanulement pendant un mois, récidive des bourgeons sus-péricanulaires, nouvelle dilatation, ouverture chirurgicale des fistules ; treize mois après le début de la laryngostomie, transformation néoplastique avec adénite à droite ; généralisation progressive, malgré les rayons X et les injections de cuprase ; mort au bout de cinq mois.

A quoi est due cette transformation néoplastique ? Est-elle le fait, comme cela se voit assez souvent dans les syphilis graves, d'une dégénérescence maligne, ou bien a-t-elle été influencée par la dilatation ? Il est à remarquer que l'analyse de bourgeons péricanulaires en dehors de la zone de dilatation, par conséquent, était simplement inflammatoire au début, et plus tard l'examen histologique a dit ' cancer ectodermique '. A la même époque l'examen d'un fragment pris au niveau du thyroïde dégénéré a donné la même formule ; il semble donc que la nature spécifique grave de l'infection primaire soit surtout en cause, puisque le port prolongé de la canule a suffi pour amener la transformation néoplastique des bourgeons médiocanulaires, primitivement inflammatoires. L'étude de ce cas, que je crois unique jusqu'à présent, comme complication imprévue de la laryngostomie, sans que cette dernière puisse être incriminée, nous permet donc de conclure que chez les canulards âgés atteints de sténose serrée syphilitique, il vaut peut-être mieux renoncer à une dilatation interne prolongée, et, en cas de laryngostomie, chercher un résultat minimum et dilater peu longtemps son malade.

Rappelons seulement les trois cas de sclérome guéri par la laryngostomie dont deux sont dus à Canepelé, et un à Szleifstein. Nous n'en connaissons pas d'autres nouveaux, car la plupart des cas de sclérome sont actuellement traités par les rayons X.

La tuberculose, qui semblait au début de la méthode être une contre-indication formelle de la laryngostomie, a été l'objet de quelques tentatives dans cet ordre d'idées. Les tuberculoses ordinaires, plus ou moins ulcéreuses, plus ou moins subfébriles ont donné des échecs à Ruggi et à nous-mêmes dans un cas. Mais la laryngostomie a donné des résultats dans les formes cicatricielles et notamment lupiques.

Signalons cependant un insuccès entre les mains de Chiari et Marschik, mais la dilatation caoutchoutée a été continuée trop peu de temps. Nous-mêmes, pour une soudure lupique avons obtenu par la gaze et la pommade rouge, la formation d'un canal, mais le malade, très amélioré, ne peut pas encore se passer de sa canule.

Par contre, dans un cas opéré par Pautet et que nous avons vu ultérieurement avec lui, il y eut un bon résultat.

Signalons aussi comme succès un cas de Hölscher et un cas de Gavello (membranes cicatricielles) ; tous ces cas ont été déjà signalés dans notre rapport au Congrès de Bilbao en 1912.

Tout récemment, à la Société belge de Laryngologie (mars et juillet 1913), van der Calseyde a publié un bon résultat de dilatation avec une canule de verre en T, et Moure un cas à la Soc. franç. de Laryng. (mai 1913).

Il y a là une indication intéressante dans certaines formes fibreuses de bacillose qui seraient à préciser et permettraient les divers traitements directs récents. Naturellement ces laryngostomies ne sont pas à comparer avec les malades opérés par Gluck avec résection partielle ou transplantation de lambeaux cutanés, opérations qui peuvent amener la guérison de la lésion, mais pas toujours le rétablissement de la phonation et de la respiration buccale.

La question de la laryngostomie dans les papillomes a été très discutée, car le pronostic même des papillomes suffocants chez l'enfant est souvent variable. Cependant nous sommes bien d'accord avec l'école italienne et l'école belge, notamment avec Beco, pour conseiller et utiliser la laryngostomie dans les papillomes suffocants surtout chez l'enfant, après échec des ablations endoscopiques, et après insuccès de la mise au repos prolongé par la trachéotomie.

Rappelons à ce propos la thèse récente de Fontaine qui a rassemblé la plupart des cas connus et dont les résultats sont rapportés dans l'historique. Dans un de nos cas, très récidivant, cependant, avec généralisation progressive péricanulaire, la laryngostomie a échoué, c'est le seul cas que nous connaissions.

Par contre, chez un petit malade, trachéotomisé par le professeur Mermod, et qui a fait ultérieurement une généralisation papillomateuse suffocante de sa trachée même thoracique, nous avons pu, en deux ans d'extirpations endoscopiques avec emploi de la magnésie calcinée à l'intérieur et à l'extérieur, guérir la sténose trachéale ; des ablations répétées endoscopiques directes, hautes des papillomes glottiques n'ont pas empêché la récidive. La laryngostomie avec deux mois de dilatation à la gaze saupoudrée de magnésie calcinée nous a amené la guérison, mais la voix reste aphone. Si, chez nos laryngostomisés pour sténoses on peut promettre le retour plus ou moins complet de la voix, il faut faire des réserves en cas de papillomes.

Au début de la méthode la laryngostomie dans les cancers limités semblait devoir donner des résultats. Cette méthode a été essayée dans des cas soit de cancers nets, soit de formes anormales prises pour d'autres lésions, soit dans des cas de transformation néoplastique au cours de la dilatation.

Dans le premier ordre d'idée nous connaissons les deux cas récents de Genta signalés plus haut dans l'historique et qui n'ont pas donné un bon résultat. Il s'agissait de laryngostomie combinée aux ablations de parties molles.

Dans le cas de Botey traité par la laryngostomie, avec laryngectomie partielle du cartilage, il s'agissait d'une tumeur localisée et dégénérée ; le résultat a été bon.

Pour les cancers méconnus ou plutôt de diagnostic impossible au début signalons le cas déjà ancien de Broeckaert et le cas plus récent de Marschik. Dans ce dernier la lésion simulait absolument une chondrite et fut laryngostomisée. Le diagnostic du cancer fut fait ultérieurement, mais la lésion était trop étendue et trop basse pour une extirpation totale. Comme cas de transformation d'une lésion bénigne en apparence en une tumeur maligne signalons l'observation de Moure ou une laryngostomie fut faite pour des papillomes récidivants chez un adulte. La lésion devint néoplastique ; Moure fit une laryngectomie, mais il survint une récidive basse.

Dans notre cas de transformation de syphilis en cancer signalé plus haut, la laryngectomie ultérieure était impossible à cause des lésions basses rétrosternales.

En résumé, à part le cas un peu spécial de Botey, la laryngostomie n'est pas indiquée actuellement dans le cancer, sauf pour les formes rares plus ou moins pédiculées de tumeur bénigne dégénérée ou la laryngostomie complète la laryngectomie partielle.

Nous connaissons aussi deux cas ou la laryngostomie a donné de bons résultats pour la cure des fistules œsophago-trachéales : l'un est dû à Blanc et Jarsaillon, d'Oran, et l'autre à White.

Ajoutons enfin que la laryngostomie avec dilatation consécutive a été utilisée pour une sténose par adduction des cordes chez un canulard ; dans ce cas, opéré en 1912 par Ivanoff, cet auteur a fait la laryngo-fissure avec résection des deux aryténoïdes par incision médiane interaryténoïdienne, puis de la dilatation pendant trois mois. Tout récemment, chez un militaire canulard, trachéotomisé pour un abcès du larynx, et qui présentait de la sténose des cordes par arthrite des deux aryténoïdes. Toubert et nous-même avons pratiqué la laryngostomie avec résection des deux aryténoïdes par incision transversale postérieure, avec curetage des ventricules à la façon des vétérinaires, pour obtenir de la soudure externe des cordes. Ivanoff a opéré son malade sous anesthésie générale ; le nôtre a été opéré sous anesthésie locale d'infiltration et régionale, mais pour pratiquer la résection des aryténoïdes nous avons dû donner un peu d'anesthésie générale. Ce sont là des indications exceptionnelles, mais qui pourront probablement donner de bons résultats.

Comme indications ou contre-indications de la laryngostomie, indépendamment du diagnostic causal, il y a lieu de considérer certaines conditions ; c'est ainsi qu'un cachectique ne peut pas supporter une longue dilatation interne, à plus forte raison une longue dilatation externe post-opératoire. La fièvre est également une contre-indication. Cependant, chez une syphilitique atteinte d'asthme fébrile, nous avons pu guérir la sténose par une dilatation de trois mois à la gaze, malgré la fièvre, et un petit abcès chondral momentané. L'état des voies respiratoires est très à considérer avant de pratiquer la laryngostomie, car la broncho-pneumonie, quoique rare, est une des complications des plus fréquentes.

Chez le tout jeune enfant avant d'opérer il faut songer aussi à la possibilité d'une hypertrophie du thymus, complication qui nous a occasionné deux décès.

Résultats

Notre statistique personnelle porte actuellement sur 28 cas avec 5 morts, deux insuccès, une amélioration, 19 guérisons, et un malade opéré tout récemment. Ces 5 morts sont dues, l'une à une broncho-pneumonie post-opératoire, le malade n'ayant pas été opéré assez à froid. Le 2e cas était guéri et décanulé ; cette fillette est morte à l'age de 12 ans après sa suture, faite sous anesthésie générale ; la mort a été amenée par suffocation sous-canulaire par gros thymus ; c'était une malade guérie, et qui ne respirait que par son larynx seul avant la suture ; l'hypertrophie brusque du thymus doit vraisemblablement être mise sur le compte de l'anesthésie générale. Ce cas de mort est donc tout à fait indépendant de la méthode.

Dans le 3e cas où tout semblait bien marcher, chez un tout jeune il y eut suffocation sous-canulaire et mort au 16e jour par hypertrophie du thymus ; là aussi l'opération de la laryngostomie avait été faite sous anesthésie générale.

Dans le 4e cas il s'agit de papillomes laryngés rebelles à la trachéotomie et à la laryngostomie ; la malade reprise par ses parents malgré nous, fut laissée sans soins canulaires, et mourut de suffocation, probablement canulaire, près de deux mois après son départ de l'hôpital.

Le 5e cas, signalé plus haut, est dû à un spasme survenu la nuit chez une petite malade très nerveuse, décanulée, et bien dilatée, pour une soudure totale avec aplatissement. La mort est due à la surveillance insuffisante d'une jeune garde-malade à l'hôpital. Ces deux derniers cas sont nettement indépendants de la méthode.

Depuis trois ans, à part ce dernier cas, qui est le résultat d'un hasard très malheureux, nous n'avons pas perdu un seul malade. Il est vrai que nous les pansons nous-mêmes, détail très important, et que actuellement nous pratiquons, soit la laryngostomie, soit les plastiques, uniquement sous anesthésie locale. Comme insuccès, signalons le cas de ce syphilitique qui a dégénéré en cancer et une laryngostomie pour tuberculose ulcérée du larynx ; comme amélioration rappelons ce cas de soudure lupique, dont nous avons pu refaire complètement le canal, sans pouvoir enlever encore la canule ; il suffira très probablement d'une nouvelle dilatation pour en venir à bout. Tous les autres cas sont décanulés, et une bonne partie sont suturés, car nous ne pratiquons pas la suture précoce, et il y a des cas où nous attendons plusieurs années pour la faire.

Malgré la gravité extrême de la plupart des cas que nous avons eus à laryngostomiser, car nous dilatons par voie interne les cas bénins, et réservons la laryngostomie seulement pour les sténoses sévères, et surtout pour les soudures, notre statistique ne s'éloigne pas sensiblement de la statistique générale que nous avons rapportée au Congrès de Berlin de

1911 (*Bull. de Laryng.*, Castex et Guisez, 1er janv. 1912), statistique qui porte sur plus de 150 cas et qui donne 75,80 pour cent de guérisons, 6,30 pour cent d'améliorations, 9,21 pour cent d'insuccès, et 8,51 pour cent de morts, imputables à la méthode.

DISCUSSION

Dr. H. Marschik (Wien). Die Kanulards sind in der Regel nur durch Schwellung der Kehlkopfschleimhaut infolge des Fremdkörperreizes in diesem Zustand erhalten, sowie Präsident StClair Thomson schon erwähnt hat, und durch einfache Versetzung der Kanüle zu heilen, mit Vermeidung der Laryngostomie, welche bei Kindern wegen der gefährdeten Entwicklung des Kehlkopfs durch die Narbe mit der äusseren Haut überhaupt nach Möglichkeit zu vermeiden wäre. Die Unterscheidung zwischen echter Narbenstenose und diesen Fremdkörperschwellungen geschieht durch direkte Laryngoskopie.

Mr. E. B. Waggett (London) wished to commend a very simple method devised by William Hill. After laryngo-fissure two plates of thin metal shaped somewhat like wound-retractors are introduced into the larynx. The limbs of these retractors which lie within the larynx are curved so as to maintain a cylindrical lumen. Not only is the method very efficient, but the ease with which the plates can be removed and replaced renders it both clean and painless.

Professor von Eicken (Giessen) macht auf die ausgezeichneten Erfahrungen aufmerksam, die in seiner Klinik mit einer sogenannten Bolzenkanüle seines Assistenten Dr. Brüggemann bei drei kleinen Kindern im Alter von vier bis sieben Jahren gemacht wurden, bei denen eine fast impermeable Stenose in der Höhe des Kehlkopfes bestand. Alle andern Methoden — Tracheotomia inferior mit örtlicher Dilatation der Stenose, translaryngeale Fixation nach Schmiegelow, Thost'sche Behandlung — hatten zu keinem Ziel geführt. Die Bolzenkanüle ist von Brüggemann im letzten Heft der *Archives internationales* publiziert worden.

Sir StClair Thomson (London) said that the paper was of great interest to British laryngologists: firstly because the cases of laryngeal stenosis appeared to be less frequent in this country than abroad, and secondly because laryngostomy was apt to be tedious and disappointing.

Reply

Dr. Sargnon (Lyon). Nous sommes tout à fait d'accord avec le docteur Marschik pour dire que la trachéotomie trop haute est une cause de non-décanulement, qui doit être traitée par une trachéotomie plus

basse, comme l'ont fait déjà depuis dix ans Castaneda (de Saint-Sébastien), Moure, Schmiegelow. M. Waggett utilise pour la dilatation un tube métallique à ailette ; Molinié de Marseille en emploie un analogue (Soc. franç. de Lar., 1912) en caoutchouc. Le professeur v. Eicken utilise la dilatation interne, comme Thost, Uchermann, Sébileau etc. ; c'est une excellente méthode, mais peu utilisable dans les sténoses très serrées et les soudures, justifiable dans la méthode externe. Les sténoses très serrées et les soudures du tubard canulard, et les sténoses très serrées en général, nécessitent l'opération externe ; les autres cas appartiennent à la dilatation interne.

SECTION XV

RHINOLOGY AND LARYNGOLOGY

ON THE DESIRABILITY OF A MORE EXTENDED AND DISCRIMINATIVE NOMENCLATURE FOR THE NERVES DISTRIBUTED TO THE NOSE

By L. HEMINGTON PEGLER, M.D., Senior Surgeon, Metropolitan Ear, Nose, and Throat Hospital, London

Mr. President and Gentlemen,—The expedience and utility of a more extended and uniform nomenclature for the nasal nerves was impressed upon me when engaged in collecting materials, examining specimens, and comparing standard authorities in various languages, for the preparation of a map scheme of the trigeminal nerve.

Pictorial presentations, as seen in our atlases and anatomical works, leave little to be desired, but the nomenclature, at least from the clinical standpoint, seems somehow to be out of keeping with the artist's work, and seeing that rhinology now occupies a recognized and essential relationship to surgery and medicine, it is to be expected that those who make a special study of this department will sooner or later demand a practical and uniform terminology.

I approach the subject with a full sense of its difficulty, and if the suggestions I am about to make only lead the way to something better than the present state of things I shall be gratified ; in the meantime they are embodied in my map scheme of the fifth nerve and are open to criticism.

It may here be stated that the delicate filaments distributed to the Schneiderian membrane are not at all easy to demonstrate, nor are they usually dissected by the student ; some, too, are inconstant or variable, so that one feels a little doubtful which of the branches should receive a distinctive title and which not. Lastly, one shrinks from interfering with an old nomenclature, even though imperfect. If asked, what of the Basle Anatomical Nomenclature ? I reply that this has served a useful purpose in many ways, but that as regards the nasal nerves it has rendered no help ; the 'rami' are overloaded with adjectives indicating relative position, but seldom destination,[1] and the general result as regards rhinological progress is in my view distinctly retrogressive.

[1] e. g. 'Rami posteriores superiores nasales mediales,' for branches supplying the posterior superior part of the septum.

Coming to the actual subject, I propose to deal with it in the following order :

First, the nomenclature and subdivision of the nasal nerve, the third main branch of the ophthalmic trunk.

Secondly, some considerations regarding the naming of nerves to the following districts of the nose: (1) The cutaneous area; (2) the septum; (3) the outer wall—comprising the turbinal bodies and meatuses; (4) the roof and floor of the cavum nasi.

The nasal branch of the ophthalmic trunk, formerly known only as the 'nasal nerve', receives now in practically all text-books as synonym, the name 'naso-ciliary', which had been already adopted by Henle in 1868, but was reintroduced more recently in the Basle Anatomical Nomenclature. In the latter publication the name is employed, however, only for the orbital part, that is, up to the point at which the nerve gives off the infra-trochlear, for here the main nasal or naso-ciliary nerve, is regarded by the authors as dividing into two, namely, the infra-trochlear and the anterior ethmoidal, corresponding to the French 'nasale externe' and 'nasal interne'.

This manner of describing the nerve is adopted in the newest edition of Cunningham's *Anatomy*, 1913, and by all who follow the B.N.A. nomenclature; in other text-books commonly read by English students, e. g. those of Quain and Gray, the term 'naso-ciliary' is used as a synonym for 'nasal', in regard to the entire nerve.

We have here, therefore, some disagreement to commence with, and if we follow this point further we shall find that the nasal nerve has long been regarded by some anatomists as splitting up into branches at its point of exit from the cranial cavity to gain the cavum nasi (nasal fossa), while by others it is considered to continue as the nasal or naso-ciliary nerve, giving off lateral and medial branches as it descends in the nasal groove.

I therefore suggest that the old familiar title of 'nasal nerve' be retained as the most simple and rational for descriptive purposes and for the convenience of the clinician, the main stem being regarded as divisible into the following four sections clearly defined by natural demarcations; see Fig. 1.

1. *An orbital section,* extending from immediately behind the sphenoidal fissure to the orbital aperture of the anterior ethmoidal canal. This part corresponds very nearly[1] to the naso-ciliary nerve, and its situation in the orbit suggests this title. The nerve has been operated upon in this section for the relief of nasal neuroses.

2. *A cranio-ethmoidal section,* extending from the anterior ethmoidal canal to the anterior nasal canal.[2] This stage corresponds, save for the

[1] The difference being the interval between the infra-trochlear branch and the point of entrance into the ethmoidal opening.

[2] The term 'anterior nasal canal' is here suggested to signify the fronto-ethmoidal passage through which the nasal nerve issues from the cranial cavity to the cavum nasi; it is lateral to the ethmoidal fissure often mentioned in text-books as

tiny intermediate piece just indicated, to the anterior ethmoidal nerve of the new nomenclature, and the name suggested for it indicates its situation in the cranium with relation to the ethmoid bone upon which it rests.

3. *An intra-nasal section*, extending from the nasal aspect of the anterior nasal canal to the aperture of escape of the nerve between the nasal bone and the superior lateral cartilage, corresponding to what is termed in many of our text-books the external branch of the naso-ciliary. This is actually, however, the continuation of the cranio-ethmoidal section of the nerve now lying in the sulcus on the nasal bone, described time out of mind in osteologies as the groove for the nasal nerve ; as already indicated, it is

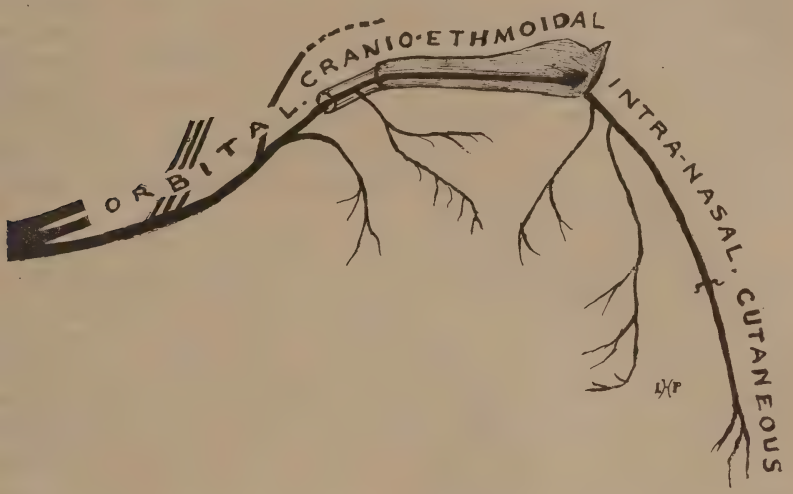


Fig. 1. Diagrammatic representation of the four stages of the nasal nerve.

comprised within the part entitled anterior ethmoidal nerve in the new nomenclature (B.N.A.) and by those authors who adopt this system. The designation ' external branch ' of the nasal nerve has been employed for it in Gray's *Anatomy* ever since the first edition in 1858 ; there are, however, yet other ways of naming this section, and the title ' external branch ' is applied appropriately by Quain's editors to the lateral offset arising from the parent stem as it enters the nasal groove. There seems to be required, therefore, some designation for this stage that will convey the fact of the nerve now in the nasal fossa or cavum having thus become actually a *nasal* nerve in accordance with its original title; the name 'intra-nasal section ' has therefore been chosen with that end in view, and I must submit that I have already found it of considerable service.

The difference between most of our standard text-books and the

giving the nerve exit (consult Quain, vol. iii, part 2, p. 16). This small canal is best seen by inverting a half section of the macerated skull and passing a bristle from below through the cribriform plate in direct continuation of the groove in the nasal bone.

Basle nomenclature is, then, that the branches given off below the cribriform plate are described as terminal branches of the nasal or naso-ciliary nerve in the former, and as branches of the anterior ethmoidal in the B.N.A.

4. The fourth and last is *the cutaneous section*, extending from the escape of the main stem through the osteo-cartilaginous junction, to its termination as a cutaneous filament at or near the point of the nose.

This continuation of the nasal nerve on the face is still the ' external nasal ramus ' of the B.N.A. and of Cunningham's *Manual* ; it is the ' anterior or superficial ' branch of Quain's *Elements*, as well as of Gerrish's *Anatomy* (1899), but in the latter the term ' anterior or superficial ' branch has already been applied to the nerve higher up, i. e. at the commencement of the intra-nasal stage. The simple designation 'cutaneous' for this superficial section has been selected as at least expressive of its destination—the skin ; no rhinologist will fail to realize, on inquiry, how confusing is the present state of the nomenclature of the nasal nerve, especially its distal part ; in suggesting any change, therefore, simplicity is the chief object to be attained.

I have already stated that the B.N.A. authorities consider the naso-ciliary nerve to divide anteriorly in the orbit into two parts, one constituting the infra-trochlear, and the other the anterior ethmoidal nerve, but this latter designation, agreeing with that of the canal and companion artery, has not been much adopted ; perhaps its popularity would have been more universal had the small ' posterior ethmoidal ' branch been more constant (as is the posterior ethmoidal artery) ; it is recorded in Poirier and Charpy, however, that Delber found this nerve only twice in six subjects.

The cutaneous nerves of the nose (Fig. 2). There is not much comment to be made upon these nerves from a terminological point of view, but they admit of simplification by arrangement into three groups dependent upon their relative position, which may be designated as follows :

1. *The superior lateral cutaneous nasal nerves* from the infra-trochlear, and an occasional twig from supra-trochlear (ophthalmic division), supplying the root and upper part of the side of the nose.

2. *The inferior lateral cutaneous and vestibular nasal nerves* : the terminal external and internal nasal branches of the infra-orbital (maxillary division), supplying lower part of side of nose and skin of ala (vestibular aspect).

3. *The dorsal or anterior cutaneous nasal nerves* : the terminal stem and branches of the cutaneous section of the nasal nerve (ophthalmic division), supplying the lower anterior aspect of nose as far as the point.

The trigeminal nerves of the septum (Fig. 3) easily fall into two groups separated from each other by the olfactory region : *an anterior*, contributed by the ophthalmic division ; *a posterior*, by the maxillary division.

The anterior group, occupying the area of the ophthalmic division, lies well in front of the olfactory region and the naso-palatine sulcus, and

extends over nearly the whole of the anterior aspect of the septum. It comprises the medial branches of the intra-nasal section of the nasal nerve which are distinguishable as the *anterior septal nerves.*

The posterior group occupying the area of the maxillary division includes :

(*a*) A few trigeminal fibres from the vidian nerve, which pass in relation to the spheno-palatine ganglion, but avoid the foramen of that name, and supply the extreme upper and posterior part of the septum in the region of the rostrum. These are part of the *supreme posterior nasal nerves,* to be further alluded to immediately.

(*b*) The medial set of the posterior superior nasal nerves from the maxillary trunk passing through the spheno-palatine ganglion and foramen ; these are distributed to the main upper and posterior area of the septum and above and below the naso-palatine groove along the course of which the naso-palatine nerve itself distributes fibrils. All these together constitute the *postero-superior septal nerves.*

The trigeminal nerves to the outer wall of the cavum nasi (Figs. 4 and 5) are also divisible into an anterior or ophthalmic, and a posterior or maxillary group, avoiding the olfactory area.

The anterior group, which is made up by the filaments derived from the lateral branch of the intra-nasal section of the nasal nerve, called by French writers ' naso lobair ', is distributed to a small anterior part of the turbinate bodies and of the intervening spaces or meatuses, and adjoining anterior wall. These are the *anterior turbinal and meatal nerves. The posterior group* includes a superior and inferior set, and is, I believe, clinically very important, owing to their exposure to pressure on the convex wall of the turbinal bodies. The superior set, viz. those to the superior turbinal body and meatus, and middle turbinate are members of the lateral set of the postero-superior nasal nerves ; they are the *postero-superior turbinal and meatal nerves,* and comprise a *superior turbinal nerve,* and a *posterior middle turbinal nerve.*

The inferior set are the nerves to the inferior turbinal body and to the middle and inferior meatuses, they are the nasal branches of the anterior palatine, and this nerve sometimes sends a branch to the middle turbinate also. They constitute the *postero-inferior turbinal and meatal nerves,* and include specifically a *posterior inferior turbinal nerve.*

The nerves to the roof and floor of the cavum nasi (Fig. 5).

The anterior slope of the roof is innervated by filaments of the intra-nasal section of the nasal nerve.

The posterior slope of the roof, at the level of the rostrum of the sphenoid, is supplied by a few filaments of the lateral posterior superior nasal nerves or of the nasal branches of the vidian, which pass posteriorly in the muco-periosteum avoiding the spheno-palatine foramen ; former authorities assigned their origin to the vidian only. As they are neither precisely septal, pharyngeal, nor lateral nasal, the name suggested for them, in company with the few filaments directed to the septal aspect of the roof,

is *supreme posterior nasal nerves* ; owing to their position in the inspiratory breathway, they are probably of considerable importance clinically.

The *floor* of the cavum, and neighbouring wall of the inferior meatus receive a nasal branch of the anterior superior alveolar ; its fibres of distribution are clinically interesting through referring sensory impressions to the canine and incisor teeth to which the main or dental branch of the nerve is continued. These nerves constitute the *antero-inferior meatal nerves*.

If these simple extensions of the nomenclature of the nasal nerves are allowed, I trust that as matters advance and our knowledge of their clinical pathology progresses, we shall indicate unequivocally, at desire, the nerve to any area or spot in the nasal cavities that is morbidly affected. The suggestions I have made are not very numerous, and the one I hold to be of greatest importance is that for a specific title for the principal branch to each turbinal body or concha, and to the areas of the septum ; I also plead for greater uniformity in descriptions of the nasal branch of the ophthalmic nerve.

Dr. Pegler demonstrated this paper by means of his 'Map scheme of the fifth nerve and its root origin',[1] also the mechanism of the 'mis-reference' of painful sensations excited in the turbinal and septal, to the supra-orbital and occipital regions.

DISCUSSION

Sir StClair Thomson (London) congratulated Dr. Pegler on his paper, and the Section on the fact that the two laboratory papers of the meeting —this one from the anatomical laboratory, and that of Messrs. Harmer and Logan Turner from the bacteriological laboratory—had been offered by the British school.

They had sometimes regretted that the tendency in England was so purely clinical, but these papers showed that good research work was being done outside the clinic.

Dr. John Cameron (Middlesex Hospital) stated that Dr. Pegler had done well in directing attention to the confusion which existed in the description of the nasal nerve, a structure which was admittedly of great importance to the rhinologist. Another point which Dr. Pegler had helped to clear up was the description of the minute twigs distributed over the sphenoidal rostrum and vomer, usually designated the superior nasal branches of Meckel's ganglion and vidian nerve, for which he suggested the name 'supreme posterior nasal'.

[1] Baillière, Tindall and Cox ; London, 1913.

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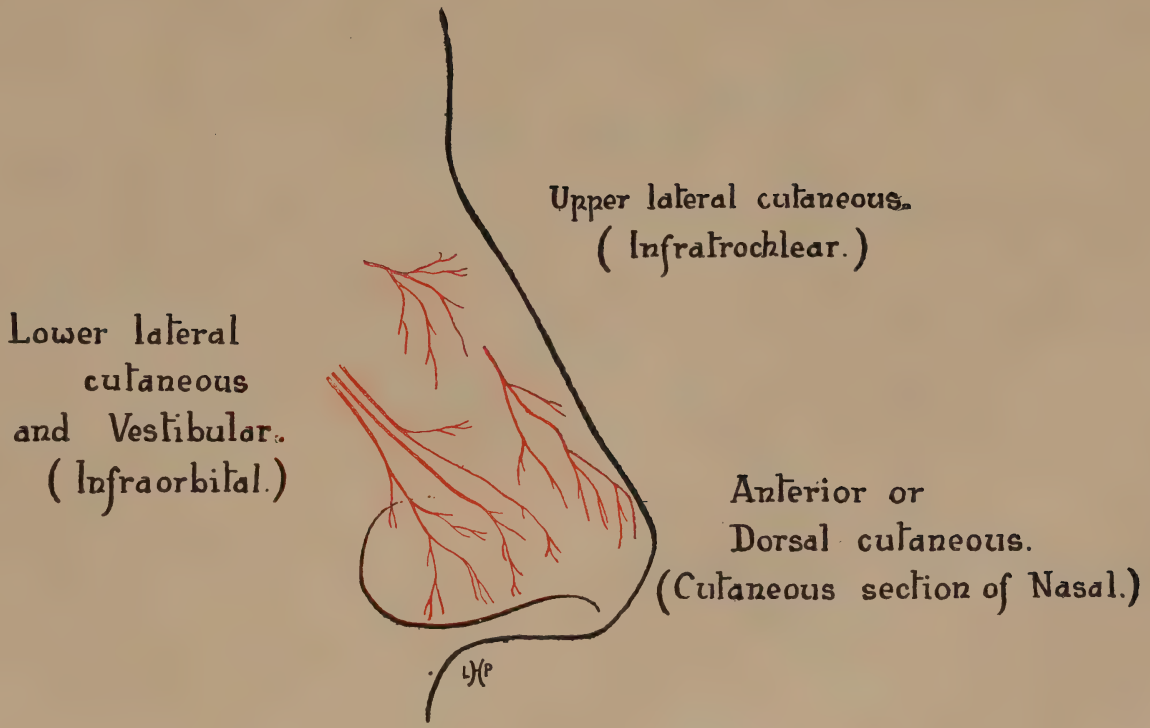


FIG. 2. The cutaneous nerves of the nose.

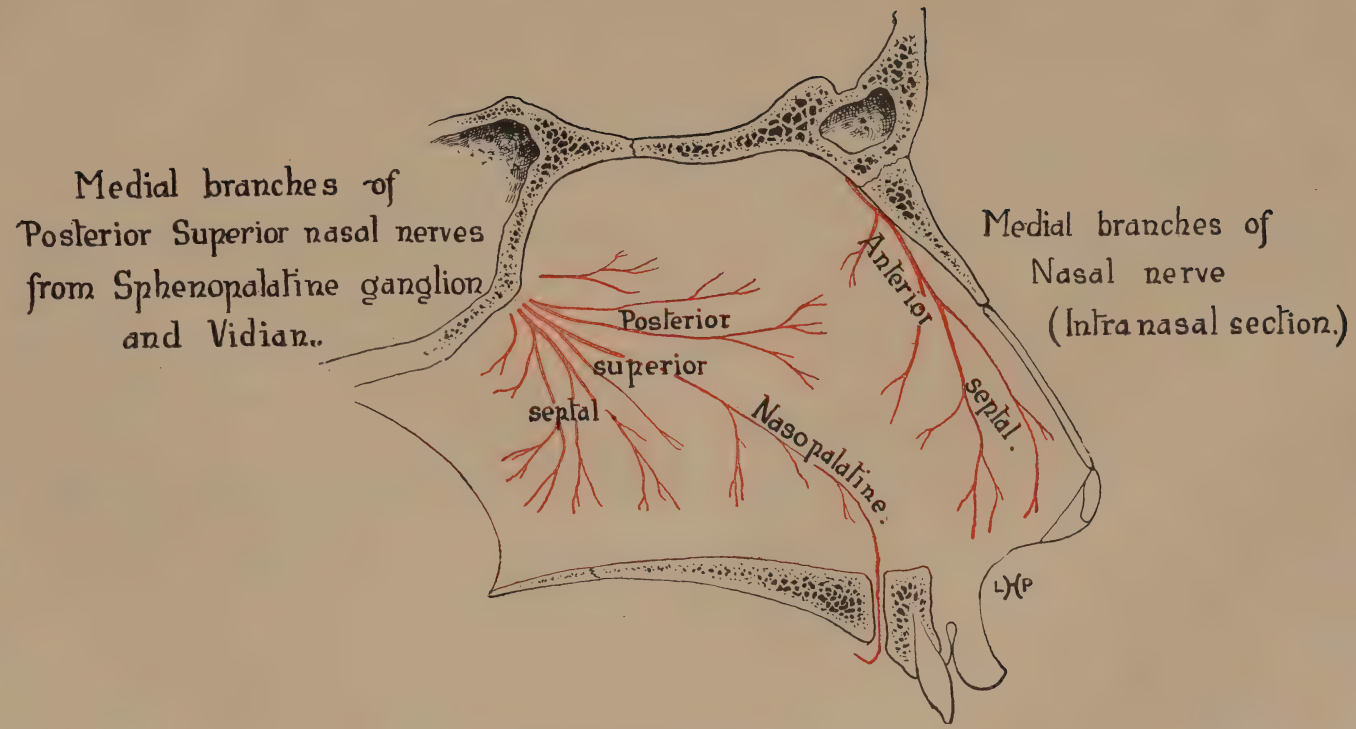


FIG. 3. The nerves of the septal wall.

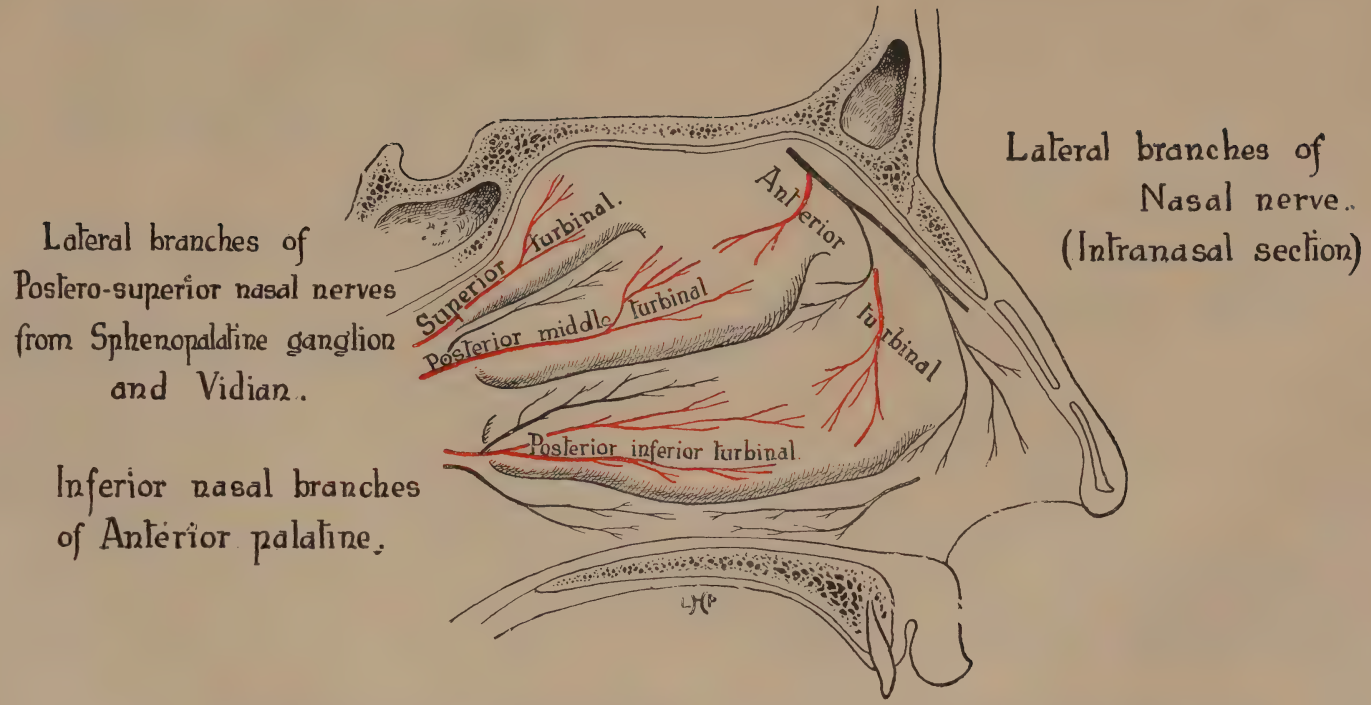


FIG. 4. The nerves of the lateral nasal wall (*diagrammatic*). The turbinal branches are printed red, the meatal black.

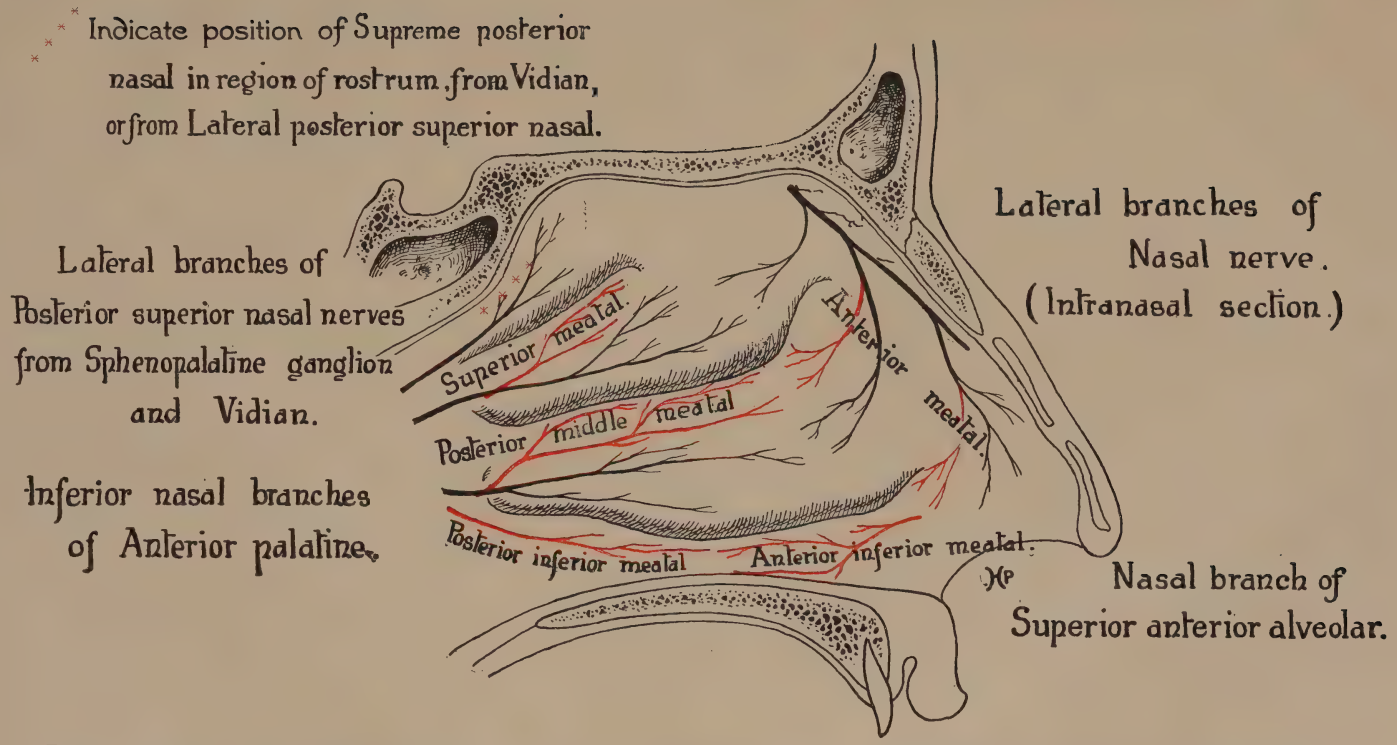


FIG. 5. The nerves of the lateral nasal wall, roof, and floor (*diagrammatic*). The meatal nerves are printed red, the turbinal black.

SECTION XV

RHINO-LARYNGOLOGY

INDEPENDENT PAPER

THE DEVELOPMENT OF THE OLFACTORY NERVES IN VERTEBRATES

By JOHN CAMERON, M.D., D.Sc., F.R.S.E., Lecturer on Anatomy, Middlesex Hospital Medical School; and WILLIAM MILLIGAN, M.D., M.S., Lecturer on Diseases of the Ear, University of Manchester, Aurist and Laryngologist to the Royal Infirmary, Manchester.

Contents

1. Introductory 451
2. The Olfactory Syncytium in Fishes 452
3. The Olfactory Syncytium in Amphibia 456
4. The Olfactory Syncytium in Birds and Mammals 457
5. The α- β- and γ-Neuroblasts of the Olfactory Syncytium and Nerve . 460
6. The Prefibrillar and the Fibrillar Phases of the Olfactory Nerve-axons. 461
7. The 'Vulnerable Points' of the Olfactory Nerve 462
8. The Mode of Continuity of the Olfactory Sense-epithelium with the Olfactory Bulb 463
9. Summary 464

1. Introductory

A few years ago the authors published the results of a research on the development of the auditory nerve, which seemed to afford ample evidence of the multicellular origin of nerve-fibres, as opposed to the monocellular theory which holds that an *axon* is the outgrowth of a single nerve-cell. The end organ or otic vesicle and the hind-brain were shown to be in intimate structural continuity from very early stages by means of a syncytium (the so-called auditory ganglion of embryological text-books), so that the exact mode of origin of the nerve-fibres appeared to be capable of interpretation with moderate ease. Since then we have been devoting our attention to the development of another nerve which afforded somewhat similar facilities for study. The results of the present investigation confirm in almost every particular our observations on the auditory nerve. We have studied types representing all the great vertebrate classes, and it may be stated at the outset, that in all the specimens examined one could recognize in the early stages, particularly in lower vertebrate types, the existence of a 'protoplasmic bridge'

connecting the olfactory epithelium with the wall of the fore-brain. These structures were thus found to constitute one continuous syncytial mass, akin to the auditory syncytium. It may be mentioned here that this bond of union between the end organ and the brain corresponds to the delicate bridge of protoplasm connecting the spinal cord with the myotome, described by Graham Kerr [8] in Lepidosiren embryos.

Methods. The embryos studied were representative of fishes, amphibians, birds, and mammals. They were fixed in Bles's fluid and cut in transverse section. The sections were mounted serially and stained on



FIG. 1. A microphotograph of the olfactory epithelium. The current view of the origin of the olfactory nerve fibres is, that they are outgrowths from their epithelial cells. Our conclusions are antagonistic to this view. Note how clearly the olfactory hairs are shown. ×600.

the slides. We again utilized the iron-alum-hæmatoxylin method of Heidenhain, slightly modified, as the chief colouring agent. This stain in our experience demonstrates the primitive nerve-fibrils in the most favourable degree. We would, therefore, strongly recommend this mode of staining to those who wish to study the histogenesis of nerve-fibres.

2. THE OLFACTORY SYNCYTIUM IN FISHES

The embryos of two Teleost fishes (*Agonus cataphractus* and *Cyclopterus lumpus*) were utilized to represent this class of vertebrates. In both these genera the early stages of the histogenesis of the olfactory nerves are exactly the same. The most striking feature in both cases is the presence of a ' protoplasmic bridge ' directly uniting the olfactory pit to

the fore-brain (Figs. 2 and 3). When this bridge is examined by higher powers of the microscope it is found to present the usual features of a syncytium, namely, an ill-defined mass of protoplasm with nuclei embedded in it, there being thus no evidence whatever of the existence of boundaries mapping out the various cell-elements. Indeed, the appearance presented by this mass is in all respects similar to that of the 'auditory syncytium' previously described by the authors[1, 2]. Proceeding on the same principle in reference to the olfactory nerves we propose to call this 'bridge' the olfactory syncytium. It is thus remarkable to note that

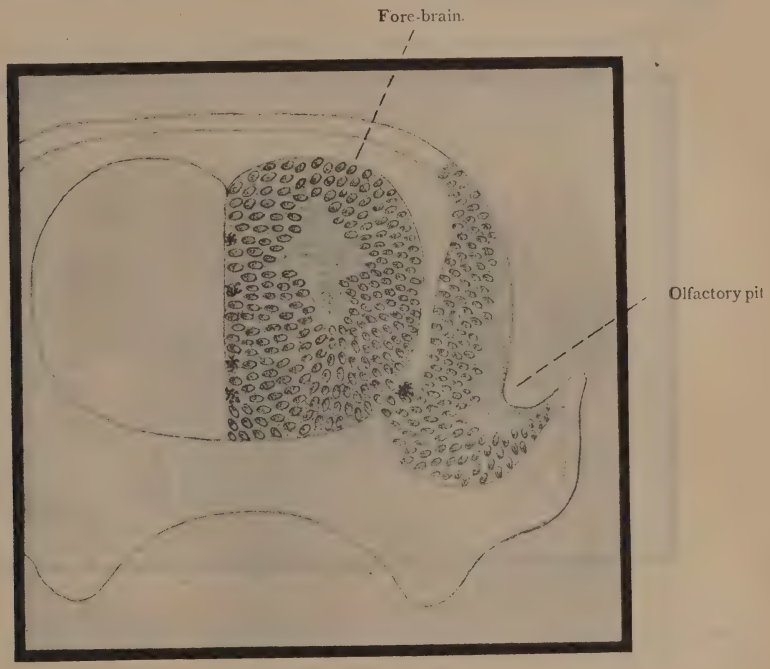


FIG. 2. Protoplasmic bridge uniting the cell elements of the olfactory pit to the fore-brain. Embryo of *Agonus cataphractus* two days after hatching. ×425.

in the case of both these sense organs during the early phases of development the mode of structural continuity between the end organ and the brain is exactly the same. We would, however, go a step further and include not only the 'protoplasmic bridge', but also the walls of the nasal sac and the fore-brain, with which it is in continuity, as the olfactory syncytium, seeing that throughout the whole mass one can readily recognize the syncytial structure. It is interesting to note how closely these observations accord with the results obtained by Elliot Smith[10] in the olfactory nerves of Lepidosiren. He describes the existence of a protoplasmic connexion between the nasal sac and the brain. Thus he states, 'not only is the nasal sac in contact with the cerebral

hemisphere at this time, but there is organic continuity between the two structures. A protoplasmic bridge extends from the nasal sac to the cerebral hemisphere, and in the neighbourhood of this connecting band I have been unable to define the boundaries of the cells either in the brain or the olfactory organ : in other words, the protoplasm of the one is in uninterrupted continuity with the other.' Holm [7] some years ago directed attention to a similar condition existing in Torpedo embryos.

This stage corresponds to our achromatic or prefibrillar phase of development of the auditory nerve, that is to say, the site of the future nerve-fibres is represented by this bridge of practically structureless

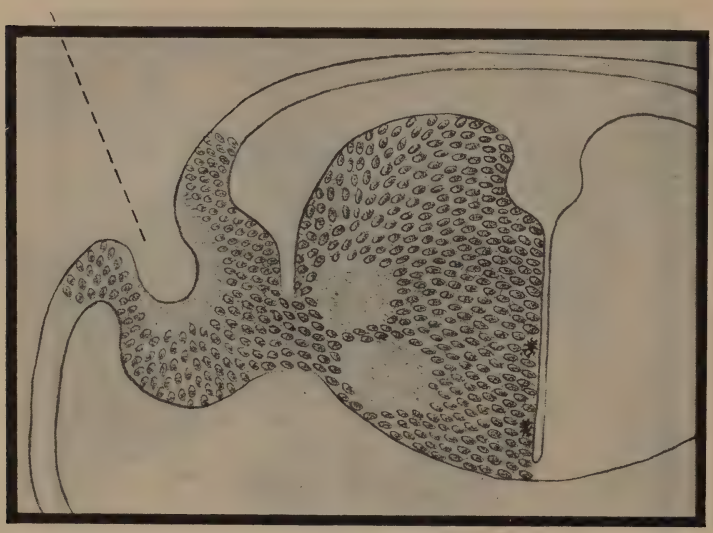


FIG. 3. Protoplasmic bridge uniting the cell elements of the olfactory pit to the fore-brain. Embryo of *Cyclopterus lumpus* at hatching. ×500.

undifferentiated protoplasm. This is the stage which upholders of the unicellular theory of nerve genesis have failed to recognize or appreciate. It certainly represents a crucial point in the controversy regarding the mode of origin of nerve-fibres. As Elliot Smith [10] clearly shows, the structure of the olfactory nerve possesses several primitive characters, and therefore presents the most favourable object for the study of the nature and mode of origin of nerve-fibres.

There can thus be no real doubt regarding the existence of this pre-fibrillar or syncytial phase, which is as prominent in the olfactory as in the auditory nerve. It is followed in a few days by the stage of fibrillation which in the case of *Agonus cataphractus* could be readily recognized ten days after hatching (Fig. 4). A process exactly similar to that which occurs in the case of the auditory nerve takes place here. That is to say, the previously undifferentiated ' protoplasmic bridge ' becomes fibrillated

throughout its whole extent *simultaneously*, a fact which seems to us to clearly prove that the axons are formed *in situ* as localized longitudinal modifications of this homogeneous material. This rather sudden alteration in the staining reactions of the ' protoplasmic bridge ' must imply a change in its chemical composition. It therefore seems to us that if one could by some means ascertain the exact nature of the chemistry of this transformation, then a good deal of the difficulty regarding nerve genesis would be cleared up.

These fibrils become aggregated into bundles to form the axons of the olfactory nerves, and it becomes obvious that, from this point of view,

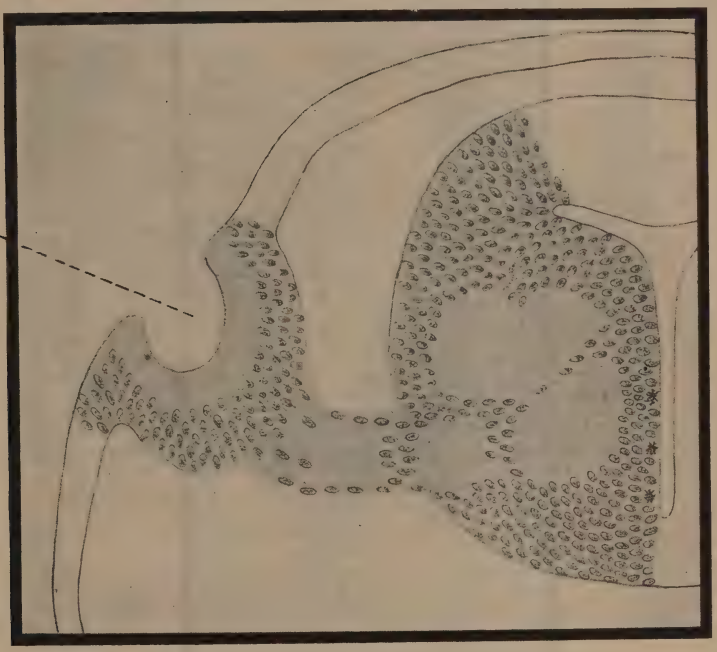


FIG. 4. Stage of fibrillation of the olfactory syncytium in an embryo of *Cyclopterus lumpus*, ten days after hatching. ×450.

the unit of nerve structure is not the axis-cylinder but the fibrilla—a fact which we have already pointed out in the case of the auditory nerve.

It is interesting to note what happens to the nuclei of the ' protoplasmic bridge ' during this period. The behaviour of these nuclei is again an exact repetition of what we described in the case of the auditory nerve. Thus the primitive neuroblasts, which we designated the α-type, develop later into the β- and γ-types. Those of the γ-type become the most highly evolved, and are represented in the adult by the olfactory epithelium and the mitral cell-elements of the olfactory bulb. Those of the β-type are subsidiary to the γ-type, as in the auditory nerve, and persist as the nuclei of the neurilemma sheaths of the olfactory nerve. It will be recognized

from this description that the neuro-fibrillation must pass continuously throughout the syncytium from the nasal sac into the olfactory bulb. The direct anatomical continuity which existed between these structures in the early developmental phases is thus maintained in the later stages, and quite confirms our results in the auditory nerve.

3. The Olfactory Syncytium in Amphibia

The relationship of the cell-elements of the olfactory pit to the fore-brain in Amphibia is very similar to what has just been described for Teleosts.

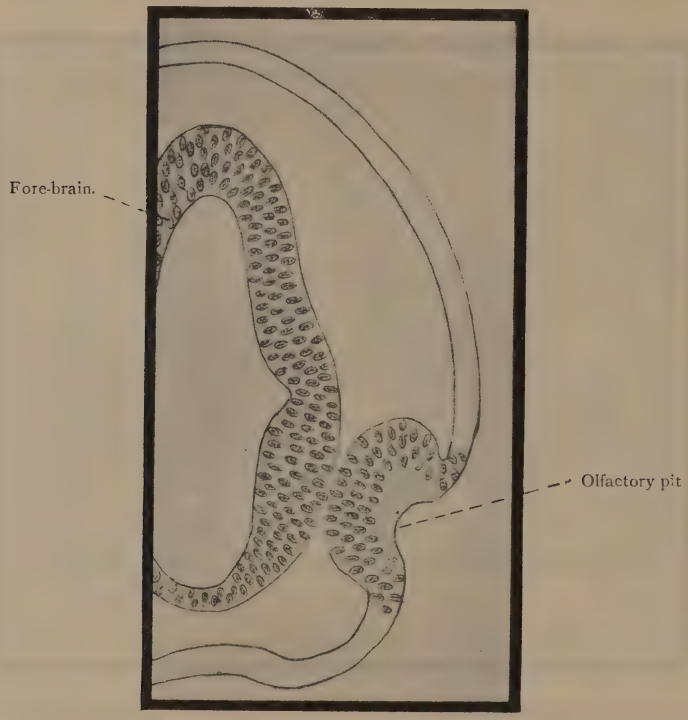


Fig. 5. Direct protoplasmic continuity between cell elements of olfactory pit and the fore-brain in a 6·5 mm. frog embryo. ×450.

In a 5-mm. frog embryo, for example, the nasal pit is in close contact with the wall of the fore-brain, and over a certain area the existence of the protoplasmic bridge of continuity can be demonstrated (Fig. 5).

The stage of fibrillation likewise admits of convenient study in this vertebrate class, for the neuro-fibrillæ are prominently displayed in frog embryos by the iron-alum-hæmatoxylin stain. So much so that it seems an easy matter to trace them from the nasal sac along the syncytium into the olfactory bulb (Fig. 6).

The process of evolution of the β- and γ-types of neuroblast from the primitive α-types is also readily demonstrated in Amphibia, owing to

the large size of the nuclei. The progressive increase in the size of the nuclei of the γ-type is strikingly characteristic, and must denote the existence of great metabolic activity in these. They can thus be easily identified in the walls of the nasal sac and the olfactory bulb, where they are to be the forerunners of the olfactory epithelium and the mitral ' cells ' respectively. The β-type, on the other hand, can

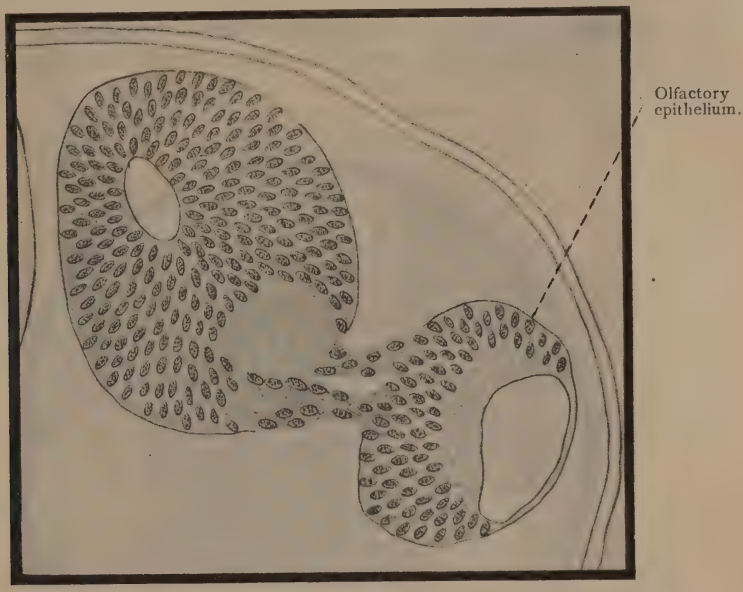


FIG. 6. Stage of fibrillation of the olfactory syncytium in an embryo frog, 10 mm. long. ×400.

be seen to best advantage in the substance of the rapidly fibrillating protoplasmic bridge, where they are obviously destined to form the neurilemma cell-elements.

4. The Olfactory Syncytium in Birds and Mammals

These two vertebrate classes will be considered together, since we have found that the early developmental stages of the olfactory nerves display a close similarity in both. For example, it was found that in a 48-hour chick and in a 6-mm. cat embryo the nasal sac and the fore-brain were separated from one another by a very decided interval occupied by mesenchyme (Figs. 7 and 9). We could find no direct protoplasmic continuity between these during the earliest stages in birds and mammals. If, however, the mesenchyme tissue intervening between the olfactory pit and the fore-brain be closely studied by higher powers of the microscope in a chick about the 48th hour of incubation, it will be found that its nuclei begin to exhibit active karyokinesis about this period. Fig. 7, which is from a 48-hour chick, shows the earliest signs of this proliferation.

The nuclear division in this region of the chick embryo is so free that after the lapse of only 12 hours—that is, at about the 60th hour—a nucleated ' protoplasmic bridge ' directly uniting the olfactory pit to the fore-brain has been established (Fig. 8). The mode of division of these nuclei is well worth study, for it will be noted that the daughter-nuclei always separate from one another in the direction of the line of the future olfactory nerve. This is exactly the same plane of division that we described in the auditory syncytium [1, 2] and also in the spinal nerves [6].

Another striking fact about the newly formed protoplasmic bridge is the abundance of the clear protoplasm in which the nuclei are embedded,

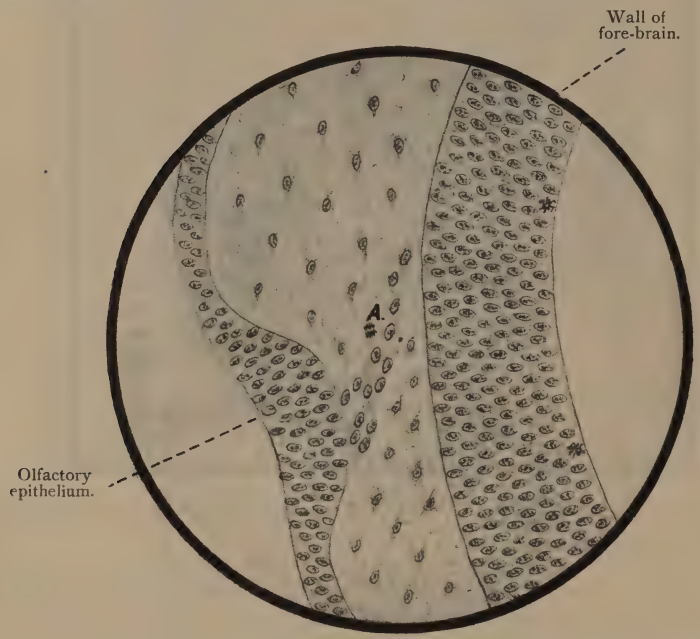


FIG. 7. Embryo chick at 48th hour. Note the division of the mesenchyme nuclei at A to form the olfactory syncytium. ×450.

which contrasts strongly with the scanty cytoplasmic investments of the mesenchyme cell-elements in the vicinity. This, again, was found by us to be a characteristic of the developing auditory [1, 2] and spinal nerves [6], and we may make the same suggestion here as to the origin of this material, which apparently seems to be a derivative of the metabolic activity of these nuclei. Of this fact we are becoming more and more convinced.

Another suggestive point brought out by a study of chick embryos was that during the formation of this ' protoplasmic bridge ' by the mesenchyme, the fore-brain and the olfactory pit were becoming rapidly separated from one another. Thus it was found that between the 48th and 60th hours of incubation the point of attachment of the syncytial bridge to the fore-brain had grown forward no less a distance than 130 μ from the olfactory

pit in these 12 hours. This will afford some idea of the karyokinetic and metabolic activity displayed by the nuclei of the mesenchyme in building up the syncytial bridge during this period. This fact appears to us to have a very important bearing upon the question of nerve genesis, for it seems impossible to imagine how even the most rapidly growing axons from the olfactory cells could ever keep pace with this swift separation of the brain from the end-organ.

The formation of the olfactory syncytium was studied in rat and cat embryos as representing mammals, and here, just as in the chick, the



Fig. 8. From a 60th-hour chick embryo, to show a further stage in the formation of the olfactory syncytium. ×450.

syncytial bridge was found to be formed by a proliferation of mesenchyme cell-elements occupying the interval between the nasal sac and the fore-brain (Fig. 9).

It seems to us quite remarkable that the mesoblast should be solely responsible for the formation of the 'protoplasmic bridge' connecting the olfactory pit to the fore-brain in birds and mammals. This discovery will come as a great surprise to those who have always been accustomed to regard nerve tissue as being essentially epiblastic in origin. One of us [6] has already shown that mesoblast tissue takes a considerable part in the histogenesis of the spinal nerves, so that the present results are confirmatory of these earlier observations.

Fibrillation of the olfactory syncytium in birds and mammals begins immediately after the completion of the bridge, that is to say, at about

the 60th hour of incubation in the case of the chick. The phenomena displayed during its progress are similar in every respect to those already described in fishes and amphibians; whilst the differentiation of the β- and γ-neuroblasts may likewise be readily observed in the two highest vertebrate classes.

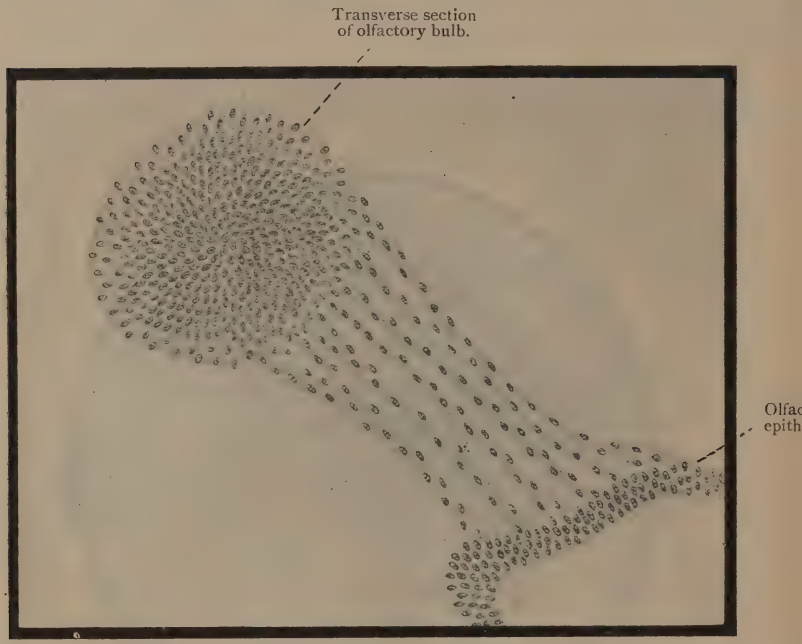


FIG. 9. Note the olfactory syncytium (derived from mesenchyme) connecting the olfactory bulb to the olfactory epithelium. Fibrillation is just commencing. 6 mm. cat embryo. $\times 300$.

5. THE α- β- AND γ-NEUROBLASTS OF THE OLFACTORY SYNCYTIUM AND NERVE

These researches have brought into prominence an interesting contrast between the lowest and the highest vertebrate classes in regard to the relationship of the nasal sac to the fore-brain. Thus in the two lowest classes the olfactory pit and the fore-brain are directly fused to one another over a certain area, and are thus brought into intimate structural continuity from the very outset. In the two highest vertebrate classes, on the other hand, the intervening mesoblast is utilized to form this protoplasmic bridge of continuity, owing to the fact that in the early developmental stages the olfactory pit and the fore-brain are more widely separated in avian and mammalian embryos.

It is significant to note that the α- β- and γ-types of neuroblast can be demonstrated in the olfactory syncytium throughout vertebrates. The existence of these three types was pointed out by one of us seven years

ago[5] and we likewise proved that they existed in the auditory syncytium[2], so that the present results confirm these earlier observations. In the early developmental stages of the olfactory syncytium the primitive α-type is the only representative just as in the case of the auditory nerve, and their nuclei are found to exhibit similar metabolic activity. The chief index of the latter is to be found in the products of their metabolism. Thus in the early stages the cytoplasmic investment of these nuclei is very scanty. Very quickly, however, they become surrounded by a clear homogeneous protoplasm, which blends to form one continuous nucleated mass uniting the cell-elements of the olfactory pit to those of the fore-brain.

A second index of the activity of the nuclei of the olfactory syncytium is furnished by the very free mitosis which they display. This has been previously shown to be one of the earliest signs of the development of the spinal[6] and auditory[2] nerves; and it is a striking coincidence that it should also be prominently exhibited in the case of the olfactory syncytium. Moreover, the plane of division always occurs at right angles to the line of the future nerve, as in the previous instances.

The further life-history of the α-neuroblasts of which the olfactory syncytium is composed, will be found to possess certain features of interest. Thus, as development proceeds, one can recognize a gradual evolution of the β- and γ-neuroblasts from the primitive α-type.

The evolution of the γ-type will be readily observed in two situations, namely in the fore-brain and in the wall of the nasal sac. Those in the latter situation become the sense-epithelium. Their nuclei are spherical, and the cytoplasmic investment exhibits fine fibrillæ which are continuous with those in the distal portion of the olfactory nerve.

The γ-neuroblasts which develop in the central end of the olfactory syncytium become the mitral 'cells' of the olfactory bulb. Their cytoplasm is characteristically abundant, and also possesses fine fibrillæ continuous with those of the syncytium.

The β-neuroblasts represent by far the greater proportion of those originally constituting the olfactory syncytium, and become distributed along the whole course of the olfactory nerves. Their nuclei, which are small and oval, are applied along the line of the nerve-fibres. One has thus little difficulty in recognizing that the β-neuroblasts become the 'cells' of the nerve sheath, and thus assume a position subsidiary to those of the γ-type, both developmentally and functionally.

6. The Prefibrillar and the Fibrillar Phases of the Olfactory Nerve-axons

We have previously shown that the life-history of the axons of the auditory nerve possessed a nascent or prefibrillar and a mature or fibrillar phase[2]. The material which was laid down along the path of the future nerve was found to be comparatively homogeneous and undifferentiated,

and thus merited the title of achromatic or prefibrillar. The mature or fibrillar phase of the auditory nerve was shown to be produced by a peculiar chemical alteration of the above material, whereby it became affected in a definite manner by staining agents. Treatment by the latter exhibited the existence of a fine longitudinal fibrillation which manifested itself *simultaneously* along the whole length of the nerve tract.

We find that a corresponding transformation is displayed by the olfactory syncytium. Thus the previously undifferentiated cytoplasm of the latter becomes fibrillated simultaneously at all parts of its course. Moreover, this change takes place at the same time that the fibrillæ are appearing in the cytoplasm of the γ-neuroblasts. More important still, the primitive fibrillæ may be traced along the whole olfactory nerve tract, particularly in lower vertebrates (e. g. amphibians); whilst their continuity with the neuro-fibrillæ of the fore-brain and with those of the sense-epithelium can be established. This simultaneous appearance of the fibrillæ shows that they are not developed independently of the syncytium as central processes from the olfactory epithelium, which is the usually accepted view of their mode of formation. These primitive fibrillæ are exceedingly fine. The future axon seems to be at first represented by a single one of these, the remaining constituents being laid down alongside, as development proceeds. The unit of nerve structure is, from this standpoint, not the axis cylinder but the *fibrilla*. We have certainly been unable to detect an outgrowth of fully developed axons from the olfactory epithelium going to form the olfactory nerve.

The cytoplasm of the olfactory syncytium, as in the case of the auditory, does not become wholly transformed into fibrils. The latter become congregated into groups to form axis cylinders, the undifferentiated portion of the protoplasm surrounding these persisting as the neurilemma sheath. The nuclei of the β-neuroblasts are, of course, the precursors of the nuclei of the neurilemma. None of the syncytial material is transformed into medullary sheath, and, therefore, the olfactory nerves are of the grey or non-medullated type.

7. The 'Vulnerable Points' of the Olfactory Nerves

Those portions of the olfactory syncytium which are situated next to the fore-brain and the wall of the nasal sac exhibit rather significant changes during development. In these regions the nuclei are very scanty and the protoplasm does not on that account appear to increase in amount, seeing that it is in some measure a derivative of nuclear metabolism. The result is that as the syncytial bridge gradually lengthens owing to the continued growth of neighbouring parts, these portions become attenuated, and quite free from β-neuroblasts. Indeed, by the time fibrillation occurs the calibre at these points has become decidedly less than that of the remainder of the syncytium. It is obvious, therefore, that since there are no β-neuroblasts left in these segments of the nerve

tract, there can be no development of a neurilemma sheath. This explains why the latter terminates just as the olfactory nerve-fibres enter the bulb and the sense-epithelium. Owing to this deficiency in the covering of the fibres it is obvious that these segments must constitute unprotected spots in the nerve trunk. We described exactly similar spots at the central and peripheral ends of the auditory nerve [2]. The researches of Orr and Rows [9] on the lymphogenic origin of toxic infection of the central nervous system have an important bearing upon this point. These observers have demonstrated that, both in the spinal and cranial nerves in general paralysis and other nervous affections, the degeneration commences in the sensory roots just before they enter the central nervous system, that is, exactly at the spot where they lose their neurilemma sheath. Orr and Rows have likewise found that after the implantation of celluloid capsules containing toxins in various parts of the body, the resulting degeneration of the sensory fibres invariably started at this situation. They have accordingly termed this the 'vulnerable point', and there can be no doubt from what we have described, that it is so, considered from a developmental standpoint as well.

8. The Mode of Continuity of the Olfactory Sense-Epithelium with the Olfactory Bulb

We have already pointed out that the olfactory syncytium, the sense-epithelium and the γ-neuroblasts of the olfactory bulb become fibrillated simultaneouly. The resulting neuro-fibrillæ thus form one continuous bond of union between the sense-epithelium and the brain. This conclusion is antagonistic to the usually accepted view of continuity of the peripheral olfactory tract. The histology of the olfactory nerve has been studied chiefly by means of the Golgi impregnation method, and the usual description is that the central fibres of the olfactory nerve end in arborizations around the mitral 'cells' of the olfactory bulb. Our experiments in the application of this method to embryonic tissues have been followed by somewhat equivocal results. The iron-alum-hæmatoxylin method is, in our estimation, much more satisfactory and more immune from ambiguity in the interpretation of the appearances presented by the tissues. Certainly, in the embryos of vertebrates below the rank of primates, this mode of staining demonstrates not a contiguity by synapse, but a direct structural continuity of the peripheral olfactory tract. Of course it is possible that the conditions which hold during embryonic life may become profoundly modified by the time full maturity has been attained; but our investigations on more mature tissues by the iron-alum-hæmatoxylin method have so far convinced us that this is not so. We have, therefore, just as in the case of the auditory nerve, decided to abandon the idea of a synapse in favour of a direct structural continuity. Thus the intimate connexion which the olfactory end-organ bears to the brain during the early stages through the medium of the olfactory syncytium *never really becomes severed*.

9. Summary

1. The olfactory end-organ is brought into direct structural continuity with the fore-brain in the early stages by means of a nucleated tract of cytoplasm to which the authors have applied the term *olfactory syncytium*. The latter corresponds to the auditory syncytium previously described by us.

2. Three types of neuroblasts may be identified in the olfactory syncytium. The term α-neuroblast has been adopted for those existing during the early stages.

3. The β- and γ-neuroblasts are further elaborations of the α-type, and represent distinct phases in the ontogeny of the nerve-cell.

4. The γ-neuroblasts make their appearance in two situations, namely, in the olfactory bulb and in the wall of the olfactory pit, where they become the mitral cell-elements and the olfactory epithelium respectively.

5. The β-neuroblasts become the cell-elements of the neurilemma sheath of the olfactory nerves.

6. The protoplasm of the olfactory syncytium is undifferentiated during the early stages. This represents the nascent or prefibrillar phase of the olfactory nerve-axons.

7. This material becomes fibrillated longitudinally in a definite manner to form one continuous tract of neuro-fibrillæ uniting the sense-epithelium with the mitral cell-elements of the olfactory bulb. This represents the mature or fibrillar phase of the olfactory nerve-axons.

8. The latter are thus not unicellular but syncytial in origin.

9. Each axon is probably represented at first by a single fibrilla. The unit of nerve structure is therefore not the axon but the fibrilla.

10. Those portions of the olfactory syncytium next to the olfactory bulb and the sense-epithelium become deprived of β-neuroblasts, and as a result there is no development of a neurilemma sheath at these points. The latter obviously represent 'vulnerable points' at which toxins may readily find an entrance.

11. The intimate association which the end-organ bears to the fore-brain during the early stages through the medium of the olfactory syncytium never really becomes severed. We have, therefore, decided to abandon the idea of contiguity by synapse in favour of a direct structural continuity of the peripheral olfactory tract.

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SECTION XV

RHINOLOGY AND LARYNGOLOGY

DEMONSTRATIONS

ORGANIZED BY E. D. DAVIS, F.R.C.S.

Thursday, August 7

Dr. R. LEWISOHN (New York) demonstrated an angular gastroscope and explained its application.

Dr. JULIUS LONDON (New York) showed a gastroscope and demonstrated its use on a model of a stomach. The instrument was a modification of the cystoscope devised by him.

Mr. T. JEFFERSON FAULDER (London) showed a remarkable case of rodent ulcer of the cheek, in which a direct view of the larynx was easily obtained through the large cavity of the ulcer.

Dr. WILLIAM HILL (London) gave a demonstration on living subjects, under local anæsthesia, of some modifications in the technique of laryngoscopy, trachea-bronchoscopy, œsophagoscopy, and gastroscopy.

I. Hill's method of combined direct and indirect vision œsophago-gastroscopy. The outer direct tube was first passed into the stomach (of a man with gastritis) by the aid of Brünings' handle lamp. The indirect optical apparatus was then inserted through this outer tube and the stomach inflated before the optical window was introduced. All present inspected the pyloric antrum, and saw the engorged veins of the lesser curvature. The instrument was in the stomach twenty-five minutes.

II. Œsophagoscopic inspection of former endotheliomatous stricture, 30 cm. from the teeth, in a woman who had had five applications of radium at intervals during the last three and a half years. At the end of the demonstration the exhibitor's radium apparatus was passed into the stricture with the aid of his special slotted œsophagoscopic funnel.

III. Tracheo-bronchoscopy on the same woman. The exhibitor's slotted laryngoscopic tube was first passed into the laryngeal vestibule and a bronchoscopic tube (Killian's pattern modified) was then inserted through it.

IV. Demonstration by Mr. Hope on another patient, an old man, of Hill's new method of passing through the slotted laryngoscope a gum elastic tracheal catheter with a rubber shoulder and umbrella three inches from its distal extremity for the intratracheal administration of chloroform and also to prevent blood entering the air-passages during

sanguinary operations in the nose, throat, mouth, and jaws. An easy, efficient, and inexpensive substitute for Kuhn's complicated apparatus.

V. A complete set of Hill's various endoscopic tubes and accessory apparatus was also shown.

Friday, August 8

Professor KILLIAN (Berlin) demonstrated the most recent improvements in his method of suspension laryngoscopy. Two patients (supplied by Dr. Hill) were prepared by Mr. Hope and Mr. Warren, but the final toilet was made by Professor Killian himself, the upper alveolar regions being rendered less sensitive by painting with a solution containing 10 per cent. cocaine and 25 per cent. rectified spirit. Neither of the patients had previously been examined by this method, and one was suspended forty-two minutes and the other thirty-five minutes. More than 100 members of the Section obtained a perfect direct view of the interior of the larynx, including the anterior commissure.

Dr. J. GAREL (Lyons) demonstrated his new improved apparatus for stereoscopic photography of the larynx in the living subject.

Mr. E. B. WAGGETT (London) demonstrated a patient to show the result of the removal of a post-cricoid epithelioma. The operation had been performed six months previously; laryngectomy had been unnecessary. The patient could swallow and speak with ease.

Demonstrations of cases were given at a number of the London Hospitals, namely: St. Bartholomew's; The Central London Nose, Throat, and Ear Hospital; Guy's Hospital; and the Metropolitan Nose, Throat, and Ear Hospital.

Monday, August 11

Professor KUBO (Japan). Demonstration der seiner Angabe nach konstruierten Instrumente.

1. Hakennadel zur Gaumennaht, mit neuer Methode der Nahtanlegung.

2. Septometer und regulierbares Septummesser für submuköse Septumresektion.

3. Leicht zerlegbares Nasenspekulum ohne Schraube (2 Formen).

4. Leicht zerlegbare Fremdkörperzange für Endoskopie.

5. Tropfflaschenfasser.

(Die Instrumente sind bei Fischer oder Pfau in Berlin zu beziehen.)

Dr. v. TÓVÖLGYI (Budapest) showed various nasal instruments.

Dr. MARSCHIK (Vienna) showed a small selection of instruments.

Dr. A. GYERGYAI (Kolozsvár) gave a demonstration of his direct method of examination and manipulation of the Eustachian tube on a living subject.

Dr. P. WATSON-WILLIAMS (Bristol) demonstrated on a patient his syringe for exploration of the accessory sinuses of the nose.

He also showed, on a model, the stages of his radical operation on the frontal sinus.

Sir StClair Thomson (London) showed a patient on whom he had performed Moure's operation for malignant disease of the ethmoid and maxillary antrum with very satisfactory results. The operation scar was almost invisible.

Tuesday, August 12

A demonstration of cases was given at the Throat Hospital, Golden Square.

Dr. M. Halle (Berlin) gave a lantern demonstration of operations for external deformities of the nose.

SECTION XVI

Otology

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CONTENTS

SECTION XVI: OTOLOGY

Proceedings of Wednesday Afternoon, August 6

PAGE

PRESIDENTIAL ADDRESS.
By Mr. A. CHEATLE 471

DISCUSSION No. 1

THE SURGICAL ANATOMY OF THE MASTOID.
Report by Professor Dr. J. MOURET (*see* Part I, pp. 205–89) . 473
Discussion by Professor MYGIND 473
Reply by Professor MOURET. 473

SUR LE DRAINAGE PHARYNGIEN DES SUPPURATIONS CRÂNIENNES D'ORIGINE OTIQUE.
Paper by Professor Dr. P. JACQUES 475
Discussion by Professor MOURE 481
Reply by Professor JACQUES 481

SECONDARY SUTURE AFTER SIMPLE RESECTION OF THE MASTOID PROCESS.
Paper by Professor Dr. H. MYGIND 483
Discussion by
Professor MOURE, Professor ALEXANDER,
Professor KUBO, Professor FERRERI,
Dr. BACON, Professor DENKER 489–91
Dr. JACK,
Reply by Professor MYGIND 491

DE L'OSTÉOMYÉLITE DU ROCHER.
Paper by Dr. L. BAR 493

Proceedings of Thursday Morning, August 7

DISCUSSION No. 2

THE PATHOLOGY OF ACQUIRED DEAFMUTISM.
Report by Professor Dr. H. MYGIND (*see* Part I, pp. 291–307).
Report by Professor Dr. A. DENKER 499
Discussion by
Dr. CASTEX, Professor ALEXANDER,
Professor UCHERMANN, Dr. HORNE,
Professor BRÜHL, Dr. SPIRA 521–3
Professor KÜMMEL,
Replies by Professor DENKER and Professor MYGIND . . 523–4
Demonstration by Professor SIEBENMANN 524

Proceedings of Thursday Afternoon, August 7

ZUR 'THERAPIE' DER 'SUBJEKTIVEN OHRGERÄUSCHE'.
Paper by Professor W. KÜMMEL 525
Discussion by
Dr. HALÁSZ, Mr. SCOTT,
Dr. SPIRA, Mr. JENKINS,
Dr. BAR, Professor ALEXANDER 533–4
Reply by Professor KÜMMEL 534

CONTENTS

PAGE

NOTE CRITICHE SULLA CURA CHIRURGICA DELLE SUPPURAZIONI CRONICHE AURICOLARI.
Paper by Professor G. Ferreri 535
Discussion by
Mr. Cheatle, Mr. West,
Dr. Dench, Professor Alexander,
Professor Mygind, Mr. Jenkins . . 552–4
Dr. Jansen,

DE QUELQUES REMARQUES SUR LES PANSEMENTS DES ÉVIDÉS.
Paper by Professor E. V. Segura 555

THE MECHANISM OF LABYRINTHINE NYSTAGMUS AND ITS MODIFICATION BY LESIONS IN THE CEREBELLUM AND CEREBRUM.
Paper by J. G. Wilson and F. H. Pike 563
Discussion by Dr. Bárány and Professor Alexander . 569–70
Reply by Dr. Wilson 570

THE CLINICAL DEVELOPMENT OF MY SYMPTOM-COMPLEX.
Paper by Dr. R. Bárány 571

LOCAL ANÆSTHESIA OF THE DRUM-HEAD.
Paper by N. Rh. Blegvad 591

Proceedings of Friday Morning, August 8

DISCUSSION No. 3

(Jointly with Section XV, Rhinology and Laryngology, Discussion No. 2)

THE METHODS AND RESULTS OF TREATMENT OF DISEASES OF THE THROAT, NOSE, AND EAR BY SALVARSAN AND OTHER ARSENICAL COMPOUNDS.
Report by Dr. A. Castex (*see* Part I, pp. 35–58).
Report by Professor Dr. P. Gerber (*see* Part I, pp. 59–110).
Discussion by
Dr. H. L. Whale, Professor Brühl,
Professor Alexander, Sir Robert Woods,
Dr. Maurice, Professor Finder,
Professor de Stella, Dr. Dundas Grant,
Dr. A. Lieven, Dr. Dan McKenzie,
Dr. H. Marschik, Dr. Kœnig . . . 129–35
Professor E. V. Segura,
Replies by Dr. Castex and Professor Gerber 135

Proceedings of Friday Afternoon, August 8

OTOSCLEROSE.
Paper by Professor Dr. G. Brühl 593
Discussion by Dr. O'Malley 596
Reply by Professor Brühl 596

PATHOLOGICAL CHANGES IN OTOSCLEROSIS.
Lantern Demonstration by Dr. A. A. Gray 597

ZUR PROGRESSIVEN FORTSCHREITENDEN SCHWERHÖRIGKEIT.
Paper by Dr. H. Neumann 599
Discussion by Professor Brühl 607

OTOSCLEROSIS.
Paper by Mr. G. J. Jenkins 609
Discussion by Dr. Bárány 617–18
Reply by Mr. Jenkins 618

PAGE

NOUVEAU MODE DE TRAITEMENT DE L'ANKYLOSE DES OSSELETS.
Paper by Dr. J. Molinie 619
Discussion by Dr. Maurice 628

Proceedings of Saturday Morning, August 9

DISCUSSION No. 4

DIE ANATOMIE UND KLINIK DER NICHT EITR GEN LABYRINTHERKRANKUNGEN.
Report by Professor Dr. G. Alexander 629
Report by Professor Dr. K. von Eicken 667
Discussion by
Professor Kümmel, Dr. Bárány,
Mr. Scott, Dr. Grant . . 683–6
Replies by Professor Alexander and Professor von Eicken . 686
HISTOLOGICAL SPECIMEN OF INFLUENZAL LABYRINTHITIS.
Demonstration by Priv. Docent Dr. F. R. Nager . . . 687
Reply by Dr. Nager.
MICROSCOPIC SPECIMENS ILLUSTRATING SOME OF THE NON-SUPPURATIVE DISEASES OF THE LABYRINTH.
Demonstration by Dr. J. S. Fraser 689
TWO CASES OF LOSS OF CALORIC VESTIBULAR REACTION, WITH OPERATIVE FINDINGS.
Paper by E. B. Dench, M.D. 693
PRÉVENTION DES AFFECTIONS SUPPURÉES DU LABYRINTHE.
Paper by Dr. Trétrôp 699
BEITRÄGE ZUR LEHRE VON DEN ACUSTICUS-ERKRANKUNGEN BEI LUES.
Paper by Dr. E. Vály 703
Discussion by Professor Mygind 712
L'ACOUMÉTRIE MILLIMÉTRIQUE ET SES AVANTAGES EN PRATIQUE COURANTE.
Paper by Dr. Trétrôp 713

Proceedings of Monday Morning, August 11

DISCUSSION No. 5

(Jointly with Section XV, Rhinology and Laryngology, Discussion No. 4)

SPECIAL TREATMENT OF THE THROAT, NOSE, AND EAR DURING THE ACTIVE STAGES OF CERTAIN INFECTIOUS FEVERS.
Report by E. W. Goodall, M.D. (*see* Part I, pp. 135–47).
Report by Dr. V. Delsaux 717
Additional Remarks by Dr. Goodall 729
Discussion by
Mr. A. Cheatle, Dr. Watson-Williams,
Professor Jacques, Mr. H. Tilley,
Dr. Dundas Grant, Mr. S. Scott,
Dr. Bronner, Dr. Trétrôp,
Professor Pick, Professor Mygind,
Dr. McKenzie, Dr. Maurice,
Dr. Kœnig, Professor Segura,
Mr. Harmer, Dr. Lautmann . 731–8
Reply by Dr. Delsaux 738
Resolution 738

CONTENTS

PAGE

Proceedings of Monday Afternoon, August 11

VARIOUS MICROSCOPIC PREPARATIONS.
Demonstration by Professor W. KÜMMEL 739
Discussion by Dr. WILSON 741

MICRO- AND MACRO-SCOPIC PREPARATIONS.
Demonstration by Professor G. ALEXANDER 743

VARIOUS HISTOLOGICAL SPECIMENS EXEMPLIFYING PATHOLOGICAL CONDITIONS OF THE LABYRINTH.
Demonstration by Dr. J. S. FRASER 745

EXTRA MACULÆ FOUND IN THE AMPULLÆ OF SOME BIRDS.
Demonstration by Professor U. PRITCHARD 749

RECONSTRUCTION MODEL OF THE HUMAN LABYRINTH.
Demonstration by Dr. J. K. MILNE DICKIE and Dr. J. S. FRASER 751

MICROSCOPIC SPECIMENS OF HUMAN LABYRINTH.
Demonstration by Mr. S. SCOTT 753

NOTE SUR LA TECHNIQUE DES PANSEMENTS MASTOÏDIENS.
Paper by Dr. M. MIGNON 755

Proceedings of Tuesday Morning, August 12

DISCUSSION No. 6

CLIMATIC AND OCCUPATIONAL INFLUENCES IN DISEASES OF THE EAR.
Report by Dr. C. J. BLAKE (*see* Part I, pp. 309–18).
Report by Professor G. GRADENIGO (*see* Part I, pp. 319–35).
Discussion by
Mr. CHEATLE, Dr. CHILD,
Dr. BLEGVAD, Dr. KŒNIG,
Dr. DAN MCKENZIE, Dr. MAURICE . . 759–63
Professor VON EICKEN,
Reply by Dr. BLEGVAD 763

Proceedings of Tuesday Afternoon, August 12

DEUX CAS DE MÉNINGITE OTOGÈNE SUPPURÉE SUIVIS DE GUÉRISON.
Paper by Dr. G. COULET 765
Discussion by
Mr. WEST, Mr. S. SCOTT,
Professor MYGIND, Dr. SYME,
Dr. J. S. FRASER, Dr. KŒNIG,
Dr. JANSEN, Professor ALEXANDER,
Dr. DAN MCKENZIE, Dr. JANSEN . . 768–71
Reply by Dr. COULET 772

NEW METHOD FOR THE DIRECT DILATATION OF THE EUSTACHIAN TUBE.
Paper by Dr. A. V. GYERGYAI 773
Discussion by Professor ALEXANDER and Dr. MAURICE . . 779

SECTION XVI

OTOLOGY

PRESIDENTIAL ADDRESS

By Mr. ARTHUR CHEATLE, F.R.C.S.

On behalf of my British colleagues I have the honour to give a most hearty welcome to our guests.

Much water has passed under the bridges since this Congress last met in London; to be precise it was in 1881 and therefore thirty-two years ago.

On looking at the Transactions of that Congress we see that Sir William Dalby was president of the Otological Section, and Dr. Urban Pritchard with Dr. Laidlaw Purves were the secretaries. I am happy to say that all three are alive and well to-day.

Among the visitors who took part in the work of the section we see such names as Fournier, Gellé, Guye, Knapp, Loewenberg, Lucae, Ménière, Politzer, Sapolini, Turnbull, and Voltolini. Alas! most of these have passed away, but the great Politzer is still with us in spirit, if not in the body.

If the veterans of those days were living they would doubtless ask what progress has been made during the last thirty-two years? I venture to think they would be satisfied with the answer, for among the more important advances made we could point to the following:

The temporal bone has been thoroughly investigated and described as to its variations and possibilities. The prevention of ear disease has been forwarded by the examination and treatment of school children.

Much light has been thrown on syphilitic and tuberculous processes affecting the hearing tract. Otosclerosis has been separated as a definite pathological and clinical entity from catarrhal conditions of the middle ear, although the treatment still eludes us.

All the operations for infection of the middle-ear tract and the complications thereof have been evolved, such surgical measures having been rendered possible by the work of Lister. The work on the labyrinth alone would be a splendid record, and I think that the veterans of 1881 would be startled if they knew what has been and is being done in this direction.

Your British colleagues sincerely hope that you will thoroughly enjoy the work of the Section and the entertainments they have been delighted to prepare for you.

Professor Alexander has kindly brought with him a letter from Professor Politzer, in which he wishes success to our meetings; I shall send him our most hearty thanks for his good wishes and our regrets that he is unable to be with us.

SECTION XVI

OTOLOGY

DISCUSSION No. 1

THE SURGICAL ANATOMY OF THE MASTOID

(For Report by Professor Dr. J. MOURET, see Part I, pp. 205–89)

DISCUSSION

Professor MYGIND (Copenhagen). There is only one question I should like to ask Professor Mouret: in which cases does he make use of the operation in which the mastoid antrum is opened through the auditory meatus? for it is of course impossible to know beforehand whether there is a fœtal antrum present or not.

REPLY

Professeur MOURET (Montpellier). En principe, il est difficile de pouvoir prévoir à l'avance si l'on se trouve en présence d'un antre petit et haut placé. J'ai bien donné quelques signes dans une communication faite il y a quelques mois, mais ils n'ont rien d'absolu et, en principe, il convient toujours de se comporter comme si l'on devait se trouver en présence d'un antre haut placé et d'un sinus procidens et faire la trépanation trans-spinoméatique, qui conduit toujours dans l'antre facilement et en toute sûreté.

SECTION XVI

OTOLOGY

INDEPENDENT PAPER

SUR LE DRAINAGE PHARYNGIEN DES SUPPURATIONS CRÂNIENNES D'ORIGINE OTIQUE

Par M. le Dr. P. JACQUES, Agrégé, Directeur de la Clinique d'Otologie de l'Université de Nancy

Les affinités pathologiques de l'oreille et de la gorge sont trop connues pour qu'il faille s'étonner de voir certaines phlegmasies de l'appareil auditif aboutir à des collections rétro-pharyngées. Le fait a été depuis longtemps signalé. Toutefois, en dehors d'hypothèses pathogéniques isolées visant des cas particuliers, je ne sache pas que cette remarquable complication de l'otorrhée ait donné lieu à une étude d'ensemble et qu'il en soit sorti certaines indications profitables au pronostic et au traitement.

Mon expérience me porte à croire que la survenance, au cours d'accidents suppuratifs otopathiques, d'une fusée rétro-pharyngienne, doit être considérée non pas comme une complication aggravante, non pas comme un épiphénomène d'importance secondaire, mais comme un accident critique, dont une thérapeutique avisée peut et doit tirer un parti décisif. Cette opinion est basée sur l'examen de trois faits personnels que je crois utile de résumer ici et dont on trouvera le détail dans la récente thèse de mon élève Guillemin.[1]

Obs. Ire. — Une fillette de 3 ans fait, au cours d'une otite moyenne aiguë paracentésée à temps, de l'ostéite du plancher de la caisse ; puis une hémorragie grave par le conduit. Conjurée par un tamponnement, l'hémorragie se reproduit deux jours plus tard. Bientôt apparaît du côté du pharynx une tuméfaction, dont l'ouverture donne issue à un flot de sang veineux, et nécessite, en même temps qu'un bourrage de la cavité à la gaze, une trachéotomie. Nouvelle hémorragie, quarante-huit heures après, par l'oreille, le nez, la bouche et la canule. L'enfant guérit néanmoins après élimination d'un séquestre du plancher tympanique. La convalescence, traînante, fut marquée par une paralysie du pharynx et de la langue, accompagnée d'atrophie, qui céda en quelques mois et résulta sans doute d'altérations inflammatoires des nerfs du trou déchiré postérieur et de l'hypoglosse, au cours de leur trajet dans la gaine des vaisseaux. Soupçon non vérifié de tuberculose.

[1] *Contribution à la pathogénie et au traitement des collections pharyngiennes d'origine otique*, Thèse de Nancy, juillet 1913.

Obs. II. — Un cultivateur de 50 ans entre dans mon service pour mastoïdite gauche datant de plusieurs semaines. Ouverture de l'antre et évidement de l'apophyse, qui est trouvée infiltrée de pus dans toute son étendue, sauf à la pointe. Soulagement subjectif sans tarissement de l'otorrhée. Puis réapparition de douleurs pariétales persistantes. Évidement pétromastoïdien, qui ne permet de découvrir immédiatement aucun trajet vers l'endocrâne.

La température baisse sans atteindre la normale, puis se relève entre 38° et 39°. Le patient prend l'habitus cérébral ; le teint devient gris. La douleur pariétale persiste et s'étend vers la tempe. Quelques jours plus tard apparaît un empâtement œdémateux au-dessus de la racine zygomatique. L'incision rétroauriculaire, prolongée vers la tempe, met à découvert une fistule de l'écaille, dont l'élargissement conduit sur un foyer fongueux extradural, n'opposant à la sonde aucune résistance dans la direction du lobe sphénoïdal : écoulement de liquide céphalo-rachidien, sans flux purulent.

En dépit de ce débridement la température demeure élevée, les phénomènes cérébraux tendant à s'accentuer ; je résèque une bonne partie de l'écaille temporale, depuis la fistule antérieure jusqu'au toit de l'antre, en ménageant simplement une travée osseuse destinée à contenir la hernie menaçante de l'encéphale. Évacuation d'une poche par le toit antral trépané.

La température demeure élevée, mais la douleur a cédé en avant et tend à se reporter vers la nuque, en même temps qu'apparaît un torticolis symptomatique et un empâtement profond sous-occipital. L'incision et la rugination de la base montrent que le pus sourd du crâne en dedans de la raînure digastrique. Je poursuis le décollement jusqu'au trou stylomastoïdien et au condyle occipital, en arrière du trou déchiré.

Peu de jours après cette dernière intervention, en même temps que s'abaissait la température, une certaine dysphagie attirait mon attention vers le pharynx, où il me fut aisé de deviner l'existence d'une collection latérale, soulevant la gouttière gauche en arrière du pilier postérieur amygdalien. L'incision immédiate au galvano fournit en grande abondance du pus à caractères identiques à ceux de la plaie rétroauriculaire.

Dès lors tous les phénomènes commencent à s'amender : la suppuration se réduit singulièrement par la perte de substance opératoire ; le teint s'éclaircit, l'appétit renaît, la température tend vers la normale. L'ouverture pharyngienne dut être renouvelée et maintenue par une divulsion quotidienne ; mais, grâce à cette précaution, tous les accidents si graves qui, pendant plusieurs mois, avaient mis en question l'existence du malade, cédèrent définitivement. La vaste plaie opératoire se répara vivement et quelques semaines plus tard notre homme pouvait quitter l'hôpital, ne conservant de sa grave affection qu'une certaine raideur du cou et quelques difficultés dans la déglutition.

Obs. III. — Mlle M., 46 ans, depuis longtemps atteinte de rhinite purulent chronique, fait en septembre 1911, au cours d'une grippe, une sinusite maxillaire aiguë et une otite perforative droite. La sinusite cède au traitement habituel ; mais l'otite ne tarde pas à se compliquer de mastoïdite.

Trépanation antromastoïdienne : corticale très épaissie recouvrant un foyer diffus d'ostéite périantrale. Atténuation des douleurs sans abaissement notable de la température. Infection des téguments mastoïdiens, suppuration abondante. Trois semaines plus tard, accès vertigineux intense avec vomissements à type cérébral, sans altération de la motilité,

de l'intelligence ni de la sensibilité ; sans fièvre. Les phénomènes alarmants cèdent après 24 heures ; mais l'écoulement purulent va en s'accentuant, en même temps que s'accusent les signes de résorption septique. Il existe de toute évidence une collection extradurale, vraisemblablement périsinusienne, qui se draine imparfaitement par une voie détournée.

Un large évidement pétromastoïdien montre l'os sain partout, sauf au niveau du plancher de l'antre, où le stylet pénètre de 1 cm. environ vers le coude inférieur du sinus. Par ce trajet, élargi à la curette, reflue dans les jours suivants du pus en abondance, sans que paraissent s'amender sensiblement les phénomènes d'infection générale. La jugulaire au cou n'offre aucun signe de phlébite. Le sinus latéral, décortiqué, n'est thrombosé nulle part. Entre temps se manifeste un torticolis de plus en plus accentué ; la nuque, douloureuse à la pression, témoigne d'une tendance du foyer suppuratif osseux à migrer au-dessous de l'occipital. Peu après surviennent des troubles dysphagiques et l'inspection révèle l'existence d'une voussure rénitente de la gouttière pharyngée droite, dont l'incision évacue du pus en très notable quantité.

Dé ce jour commence une nouvelle période, caractérisée par une atténuation progressive de l'écoulement mastoïdien et des phénomènes d'infection. Le teint s'éclaircit de jour en jour, l'état général se relève et j'éprouve l'impression nette que la crise est franchie et que la guérison, si longtemps incertaine, est désormais assurée. Grâce à une surveillance étroite de la collection pharyngienne, le drainage déclive du foyer sous-occipital s'effectue d'une manière permanente et la plaie rétroauriculaire se répare rapidement. Un mois plus tard la guérison était à peu près complète ; céphalée et septicémie n'avaient plus laissé de traces : seule persistait encore un peu de raideur du cou.

Quand on parcourt les relations d'abcès rétro-pharyngiens consécutifs à des pyotites (mon interne Guillemin en a rassemblé une cinquantaine) on se convainc qu'en dehors des adénites, dont l'origine auriculaire demeure discutable et que je laisserai de côté, pour le moment, deux processus semblent intervenir pour diriger vers la gorge le pus ostéitique issu du temporal.

Ou bien il s'agit d'une effraction directe, de haut en bas d'un foyer situé à la face inférieure du rocher, soit que le pus de la caisse traverse le plancher nécrosé ou déhiscent, soit qu'il suive la trompe ou le conduit du muscle du marteau, soit enfin qu'interviennent les cellules sous-labyrinthiques bien étudiées et décrites par Mouret.

Ou bien il s'agit d'abcès intracrâniens développés au voisinage du sinus latéral ; ayant fusé dans la région sous-occipitale par un mécanisme que je chercherai à élucider tout à l'heure, puis migré dans une troisième étape vers l'espace rétro-pharyngé.

D'après mes relevés, les collections pharyngiennes d'origine otique pourraient se réclamer pour une part à peu près égale des deux processus ci-dessus. Sur 45 cas suffisamment explicites j'en rencontre 20 où le trajet était direct et où l'origine du pus se trouvait être la caisse, ou bien une cellule voisine de la face inférieure du rocher ; et 21 dans lesquels la suppuration n'était parvenue au pharynx que par un trajet détourné, après une étape sous-occipitale ou nucale : les 4 autres semblant se rapporter, non pas à des abcès ostéogènes rattachés à leur point d'origine

par un tractus suppuratif continu, mais à des collections développées à distance dans des ganglions.

La catégorie de beaucoup la plus intéressante, parce qu'elle offre des difficultés d'interprétation et de traitement toutes particulières, est constituée par le groupe des collections à trajet indirect. C'est elle seule que j'envisagerai ici.

Mes observations II et III offrent deux intéressants exemples de ces collections migratrices à étape sous-occipitale. Nous pouvons, je crois, les considérer comme des types. Comment donc expliquer la marche du pus en pareilles circonstances ?

Notons tout d'abord qu'il s'agit d'abcès extraduraux intéressant soit les deux versants de la pyramide pétreuse, soit le versant cérébelleux seul. Leur origine peut être une nécrose du toit tympanique ou antral. Ce doit être plus souvent une cellulite de la région intermédiaire au plancher antral, au labyrinthe statique, à la gouttière du sinus latéral et à la rainure digastrique (région sinuso-digastrique de Pérez, sur l'intérêt pathologique de laquelle G. Laurens avait, dès 1904, au Congrès international d'Otologie de Bordeaux, justement attiré l'attention).

Le pus collecté dans la concavité de la fosse osseuse de l'hémisphère cérébelleux pourra s'échapper de l'endocrâne vers la nuque par différentes voies :

En suivant le trajet des plexus veineux extraduraux entourant l'artère vertébrale.

En perforant l'écaille occipitale dans sa portion très mince et parfois lacunaire sise en dehors du trou occipital et en arrière des condyles.

En empruntant la scissure occipito-mastoïdienne.

C'est à ce dernier processus que je crois avoir eu affaire dans mon observation I[re] et je suis porté à croire qu'il intervient dans la majorité des cas. La suture occipito-mastoïdienne constitue, en effet, chez beaucoup de sujets et jusque dans l'âge avancé une fissure perméable courant parallèlement à la rainure digastrique en dedans de celle-ci, et souvent (Hyrtl) traversée par un rameau de l'artère occipitale sous-jacente. Le pus endocrânien, amené ainsi dans l'interstice celluleux parcouru par l'occipitale, pourra aisément, en suivant celle-ci, se glisser entre les aponévroses profondes de la nuque. Je ne crois pas, en effet, qu'il s'agisse habituellement d'un décollement du périoste de la face exocrânienne de l'écaille occipitale. Le périoste, confondu avec les multiples insertions des muscles nucaux, adhère très intimement à l'os, que je n'ai pas, pour ma part, trouvé dénudé.

Si maintenant nous nous reportons aux observations cliniques, nous voyons que ces abcès profonds de la nuque marquent plus de tendance à se diffuser vers en bas et vers en arrière, entre les plans musculo-aponévrotiques, qu'à filer en avant vers la gouttière pharyngée. Quand cette dernière éventualité se réalise, ce n'est qu'après une période de tension plus ou moins prolongée, ou bien grâce à un débridement heureux portant sur la région intermédiaire à la rainure digastrique et au condyle

occipital. Une étude attentive de l'anatomie de cette région me paraît de nature à éclairer le mécanisme du drainage pharyngé spontané et, mieux, à diriger l'effort thérapeutique.

A sa face inférieure, ou exocrânienne, l'écaille occipitale apparaît martelée d'insertions musculaires étroitement juxtaposées. Toutefois, entre la rainure digastrique, en dehors, et le condyle, en dedans, règne une zone relativement unie, que parcourent d'avant en arrière et parallèlement l'un à l'autre la suture occipito-mastoïdienne et le sillon de l'artère occipitale. Le pus, fusant par la suture hors de la fosse cérébelleuse, débouche dans une région circonscrite en dedans par le condyle occipital, en arrière par les attaches des muscles grand et petit droits postérieurs et petit oblique, en dehors par les insertions du digastrique du petit complexus, du splénius et du cleïdo-mastoïdien, en avant par une barrière musculo-aponévrotique continue, dont la constitution nous importe tout particulièrement.

Tandis, en effet, qu'en arrière les insertions des muscles ménagent entre elles des espaces celluleux parcourus par le tronc et les branches de l'artère occipitale, accompagnée de ses veines et nerfs satellites — offrant aux collections septiques des voies de diffusion vers la nuque — celles-ci demeurent isolées du pharynx par une sorte de cloison étanche tendue entre la mastoïde et le condyle occipital, qui apparaît bien sur une coupe horizontale pratiquée à quelques millimètres au-dessous de la base du crâne. Elle est constituée de dehors en dedans par :

1° Le feuillet postérieur, très résistant, de l'aponévrose parotidienne, fixé d'une part au bord antérieur de la mastoïde, d'autre part étroitement adhérent au corps de la styloïde et aux muscles qui l'engainent.

2° L'apophyse styloïde.

3° Un feuillet aponévrotique solide engainant le muscle droit latéral et tendu entre la styloïde et la face externe du condyle. Ce feuillet, fixé supérieurement à la lèvre postérieure du trou déchiré, se confond avec la gaine du paquet vasculo-nerveux, constitué par la jugulaire interne, les nerfs des neuvième, dixième et onzième paires et la carotide interne.

La même coupe met en évidence le feuillet externe de la gaine des vaisseaux, formé par l'aponévrose stylo-pharyngienne, ainsi que son feuillet interne, qui n'est autre que l'aponévrose prévertébrale. Il est aisé toutefois de se rendre compte par la dissection que l'adhérence est singulièrement plus étroite pour le paquet vasculo-nerveux avec le feuillet stylo-pharyngien qu'avec la gaine des muscles droits antérieurs, si bien qu'on peut considérer qu'il existe un espace cellulaire virtuel le long de son bord interne, espace qui n'est isolé de l'espace rétro-pharyngien que par la fragile expansion détachée de l'aponévrose latéro-pharyngienne vers l'épaisse lame prévertébrale.

En somme on peut admettre que l'unique obstacle à la migration d'une nappe purulente sous-occipitale vers le pharynx réside dans l'existence de la cloison musculo-fibreuse, tendue verticalement entre l'apophyse transverse de l'atlas et l'apophyse jugulaire de l'occipital, et, transversalement,

de la styloïde au condyle. Or il semble bien que dans la majorité des cas cet obstacle oppose aux progrès de l'infiltration suppurative une résistance supérieure à celle des interstices musculaires de la nuque : d'où la tendance habituelle à la diffusion en arrière.

Quand nous assistons, plus ou moins tardivement, à l'issue favorable constituée par l'abcès latéro-pharyngien, il n'est pas interdit de supposer que l'aponévrose du droit latéral a cédé sous l'effort du pus dans sa partie interne ; mais les recherches que j'ai effectuées avec M. Guillemin nous portent à faire intervenir une disposition anatomique particulière, d'ailleurs assez commune : je veux parler de l'existence accidentelle d'un *canal condylien intermédiaire.*

On désigne sous ce nom une gouttière (souvent transformée en canal), qui apparaît creusée au flanc externe du condyle, qu'elle parcourt d'arrière en avant. Ce conduit, intermédiaire au canal condylien postérieur, trou veineux, et au canal condylien antérieur, destiné à l'hypoglosse, donne passage à un canal veineux établissant une anastomose entre le vaisseau condylien postérieur et les plexus entourant le nerf de la douzième paire. Le canal condylien intermédiaire perfore donc d'arrière en avant l'aponévrose du droit latéral au voisinage de son attache à la face latérale du condyle. Nul doute que chez les sujets porteurs de cette disposition une collection, retenue sous l'écaille occipitale, ne trouve, en suivant le trajet de la veine condylienne intermédiaire, un accès relativement aisé vers l'espace celluleux occupant la région la plus interne de la gaine vasculo-nerveuse, et, de là, à travers le mince feuillet pharyngo-prévertébral, vers l'espace latéro-pharyngien. La voie est d'autant mieux tracée que le canal condylien intermédiaire vient en avant s'aboucher dans la terminaison du canal condylien antérieur, abordant à sa sortie l'hypoglosse, qui guidera plus sûrement encore l'abcès migrateur à travers la région postéro-interne de la gaine des vaisseaux.

Cette pathogénie admise pour la variété d'abcès pharyngiens otogènes que nous avons envisagée, je me suis demandé s'il ne serait pas loisible au chirurgien de favoriser, par une manœuvre opératoire, une éventualité que la nature réalise trop rarement et que je considère comme la plus avantageuse des issues dans les circonstances considérées. Et il m'a semblé qu'en ruginant avec précaution d'arrière en avant la face latérale du condyle, après résection de la pointe mastoïdienne et ligature de l'occipitale, nous pourrions, sans intéresser ni la jugulaire, ni les nerfs mixtes, suppléer au défaut du canal condylien intermédiaire et amorcer le drainage pharyngien. Sans avoir eu encore l'occasion de pratiquer systématiquement cette manœuvre j'ai l'impression d'avoir, chez le malade de ma deuxième observation, facilité l'effort de la nature en ruginant la face inférieure de l'occipital jusqu'au voisinage du condyle, dans le but de rechercher de ce côté une fistule qui ne fut d'ailleurs pas rencontrée.

DISCUSSION

Professeur Moure (Bordeaux). Je voudrais demander à M. Jacques si les parois du sinus étaient intacts et s'il ne s'agissait pas de phlébite suppurée ayant tendance à s'ouvrir dans le rétro-pharynx.

D'autre part il faut aussi tenir compte dans le diagnostic des ostéo-myélites profondes d'origine vertébrale dont la symptomatologie ressemble beaucoup à celle d'une mastoïdite profonde.

Reply

Professeur Jacques (Nancy). Le temps limité dont j'ai disposé ne m'a pas permis de relater en détail les observations sur lesquelles est basé mon travail. J'ai pu en effet éliminer, dans les deux cas relatifs aux collections rétro-pharyngées précédées d'abcès de la nuque, que j'ai observées, d'une part, l'origine jugulaire de la suppuration, d'autre part l'existence préalable d'une ostéite de l'atlas ou de l'axis. Il n'en reste pas moins que le diagnostic toujours délicat, sur lequel M. Moure vient d'attirer notre attention, devra toujours se poser et être résolu dans la mesure du possible avant ou pendant l'opération.

SECTION XVI

OTOLOGY

INDEPENDENT PAPER

SECONDARY SUTURE AFTER SIMPLE RESECTION OF THE MASTOID PROCESS

By HOLGER MYGIND, Copenhagen

There is no doubt that there are many drawbacks attached to the ordinary after-treatment of cases of simple resection of the mastoid process for acute osteitis by the filling up of the osseous cavity with granulations. The chief of these drawbacks is that the healing process is lengthy, taking generally from six to twelve weeks. Thus the average duration of the cases not complicated with cerebral abscess, sinus thrombosis, or meningitis treated at the Ear and Throat Department of the Copenhagen Commune Hospital is about 74 days.

Besides, there is an inclination, especially when the patients are young children, to eczema of the edges of the wound and of the skin of the adjacent parts, as also the frequent changing of the bandages is often troublesome.

It would, therefore, seem reasonable to suppose that the various efforts made to abbreviate and simplify this after-treatment should have been received with interest, and also that surgeons, who had been successful in their attempts, would have found followers. This, however, has not been the case, and two methods which in particular mark a distinct progress have not gained the recognition they deserve. I refer to the use of primary suture after filling up the cavity in the bone with blood-clot, as first suggested by Frank B. Sprague, of Providence, U.S.A., and first performed by Clarence Blake, of Boston, in 1906, and to a method briefly reported by Passow in 1908, by which periosteal flaps are loosened from the edges of the wound some time after the operation and sutured together, accompanied by suture of the edges of the skin. I have for some years, in a number of cases which were suitable for such treatment, used primary suture, and have often obtained primary healing. I tried Passow's method in some few cases, but gave it up, as I had, before I saw Passow's short account of it in his 'Ueber den Verschluss der Knochenwunden nach Antrum-Operationen' (*Beiträge*, vol. i, 1908, p. 70), commenced working with a method which has given me better and

better results the more I have been able to improve its technique.[1] It is, in my opinion, the technique which is the difficult part of this method, and not the indications, which are simple.

As far as the indications are concerned my method of secondary suture can be employed from eight to twenty days after resection of the mastoid process for acute osteitis when there is no swelling of the skin, subcutaneous tissue, or periosteum ; when there is a good formation of granulations in the osseous cavity and there are no pus-filled sinuosities in the bone ; when there are no necrotic parts in the bone or the edges of the wound ; when there are no intercranial complications or signs of labyrinthine inflammation ; and, finally, when the secretion of pus from the middle ear through the auditory meatus has ceased or is greatly diminished—all supposing the patient to be otherwise free from serious disease.

The process is as follows : Previous to the small operation, which, as a rule, is best performed under ether, there are a few preparations to be made. To begin with no iodoform tincture should be used at the initial operation, as it may, especially if the patient is a child, cause an irritation of the skin, and it is of the greatest importance that the edges of the wound should be in perfect condition. Eczema of the edges of the skin, so frequent in children, may often spoil the result. In order to prevent eczema or irritation of the skin, care should be taken that the iodoform gauze plugs placed in the cavity after the resection do not go beyond the edge of the granulations, and that they are changed daily the last three or five days before the secondary operation. Should the formation of granulations be excessive, isoform gauze should be substituted for the iodoform. Further, the edges of the wound and the outer ear should be covered every day to a considerable extent with a very thin layer of an ointment consisting of one part chloretum amidohydrargyricum and twenty parts pasta oxidi zinci. This ointment, which dries the skin and is antiseptic, almost always prevents eczema. Finally, in order to reduce the secretion of pus from the tympanic cavity, the auditory meatus ought to be dried every day with sterilized cotton and a 2 per cent. solution of nitrate of silver applied.

During the operation the skin surrounding the wound should be cleansed with benzine, which easily removes all traces of the ointment. The parts adjacent are then painted with a mixture of equal parts of solutio iodi spirituosa and spiritus dilutus, āā. The edges of the wound are now carefully scraped clean with a sharp spoon, all necrotic tissue is removed with scissors, as also small swollen glands, should there be any. Especial care should be taken that no necrotic remains of the fibrous insertion of M. sternocleido mastoideus are left in the wound. Finally, all soft granulations should be scraped away with a sharp spoon, leaving

[1] A survey of my results was reported at the Congress of Northern Otolaryngologists in Copenhagen 1911 (*Zeitschrift für Ohrenheilkunde*, vol. lxiv, 1912, p. 196).

everywhere a firm ground of granulations. The whole cavity of the wound is then thoroughly and repeatedly dried with iodoform gauze, which should be applied especially carefully to the granulations in aditus ad antrum. An iodoform gauze plug is then placed in the osseous cavity filling it entirely; with a sharp knife the skin and the subcutaneous tissue are loosened from the underlying periosteum to an extent of from $\frac{1}{2}$ to $\frac{3}{4}$ centimetre, so that the edges of the skin can be drawn close together in such a way that they lie, not only edge to edge, but that the deep surfaces also touch each other, facilitating thus the application of Michel's clips. The patient's head is now brought into a perfectly horizontal position, the iodoform gauze plug is removed and the osseous cavity is allowed to fill slowly with blood, which slowly forms a clot. Should the bleeding be too profuse, the edges of the wound and the cavity should be dried with iodoform gauze plugs pressed hard against the tissue; should the bleeding be insufficient, the edges of the wound should be scraped again with a sharp spoon. As soon as the coagulated blood has reached the level of the surface of the periosteum, the edges of the wound are drawn together, the periosteum being left untouched, and from three to five Michel's clips are applied. Finally, the wound is covered with a strip of perforated silk-gauze 'protective', outside which is placed sterilized gauze and a Körner's bandage. A strip of iodoform gauze is placed in the auditory meatus, after carefully drying up any pus with sterilized cotton wool.

With children the Michel's clips are removed 2 days or, still better, $2\frac{1}{2}$ days after the operation, with adults 3 days. Should there be healing *per primam* the patient can leave off the bandage 5 or 7 days after the secondary operation.

In some cases, however, there may be slight splitting of the superficial parts of the wound, arising from the straining of the edges, as also there may be a slight necrosis of the skin round one or more of the clips, &c.; in such cases it will be found necessary to retain the bandage somewhat longer.

In a minority of cases suppuration of the clot occurs. This manifests itself but rarely by slight pains in the wound, and a slight rise of temperature. As a rule there are only local phenomena: redness and swelling of the skin and an oozing of, first, serous, later on purulent, fluid from the lower angle of the wound. The wound, from which a reddish or chocolate-coloured pus will escape, must in this case be reopened and drained with iodoform gauze in the lower angle. I have never met with any other drawbacks with this method, and in any case the labour is not thrown away, as the wound heals more quickly than it otherwise would do.

I must, however, very emphatically point out that I have several times observed redness and swelling of the lips of the wound and even oozing of serous fluid without there being suppuration of the blood-clot. It is further important to remember that suppuration of the blood-clot may confine itself to its axial part. On the other hand, the cutaneous

wound and the parts adjacent sometimes present a perfectly normal appearance for some time, even for from 8 to 12 days, although the clot is suppurating. One is therefore now and again in doubt as to the advisability of reopening the wound, especially as, as before mentioned, there may be small superficial splits in the skin, slight necrosis, &c. In many persons the skin of the mastoid process, especially in children, is very thin, inelastic, and badly nourished, particularly in cases where resection has been performed and a number of blood-vessels are cut; bandaging contributes largely to these conditions. The skin of this part is, then, not particularly disposed to primary healing, for which reason skin treatment, as described above, is of great importance previous to suturing.

In cases where there is a suspicion of suppuration of the clot, and where it is unadvisable to delay, the following method may be performed successfully. The skin wound is opened to a slight extent with a probe, a small wooden sterilized stick with a little cotton wool on the end is introduced through the opening thus made. If there is suppuration of the whole clot, the end of the stick will easily enter it in every direction; if there is only suppuration in the axial part the stick will only run along the narrow canal towards aditus ad antrum, and, if the clot has kept all right, its soft, elastic surface will be distinctly felt by this slight probing, which causes no disturbance of the clot.

I will now proceed to give an account of my cases and the results obtained by the use of this method.

It has been used altogether in 91 cases of simple resection of the mastoid process for osteitis. As, in the $5\frac{1}{2}$ years I have employed this method, I have performed 221 simple resections of the mastoid process, it will be evident that in a great number of cases it has not been used. These consist, first, of a small group of cases in which primary suture was employed; secondly, of a larger group with intracranial complications; and lastly of a number of cases with bad granulations or where the patients were ill-nourished, with eczema around the wound, or suffering from serious disease. The following cases, however, will show that in the last group of cases good results may be obtained.

No. 181, 1908. Male; 9 months old. Purulent discharge from left ear, swelling behind the ear for 8 days. Resection of the mastoid process showed the soft parts considerably swollen, about 1 centimetre thick; corticalis perforated; diffused acute osteitis. Ten days later, secondary suture. A diffuse eczema appeared after this in the skin around the wound and some pus foci were formed in it; nevertheless the clot in the wound was not infected and the wound was completely healed one month after resection. The scar was not depressed.

No. 5, 1909. Male; 30 years. Resection of the mastoid process performed after 4 weeks' suppuration of the right middle ear. The mastoid process was found entirely diseased, the apex being also removed *in toto*. Three days after operation the patient had a croupous pneumonia. Secondary suture performed 19 days after operation; complete healing *per primam*, and the patient got up the day after.

No. 137, 1909. Female; 2½ years, pale, + Pirquet; purulent discharge from both ears of unknown duration. Resection of the left side showed a subperiostal abscess, partial destruction of corticalis, very extensive destruction of the osseous tissue. Thirteen days later, whilst there was still a purulent discharge from the tympanic cavity, secondary suture with a small drainage-tube in the lower angle of the wound. In spite of eczema, healing *per primam* with rapid closing of the drain aperture.

No. 312, 1909. Male; 27 years. Bilateral pulmonary phthisis. For 3 months purulent discharge from right ear with a 5 days' swelling behind the ear. Resection shows a subperiostal abscess, perforation of corticalis, and destruction of the whole mastoid process, the apex of which was removed *in toto*. Seventeen days later secondary suture, which healed *per primam* in spite of eczema of the skin of the mastoid region and outer ear.

No. 48, 1911. Female; 6 months. Fourteen days before admission, swelling behind the right ear, for which Wilde's incision was performed outside the hospital. Resection of the right mastoid process showed perforation of corticalis and diffuse osteitis. Ten days later discharge from left ear, and 11 days later swelling behind the same ear. Resection of the left mastoid process showed a large subperiosteal abscess and diffuse osteitis. Ten days after the last operation, secondary suture on both sides; healing *per primam*, excepting a slight splitting of the skin on the right side.

The majority of the patients treated were children, 41 being 15 years old or less. The youngest patient was 4 months old, and in this case healing *per primam* was obtained; the oldest patient was 60; in this case the clot suppurated. This last patient was, however, suffering from diffuse tuberculosis. The oldest patient with healing *per primam* was 54 years old. It would be natural to suppose that children with their greater power of regeneration would give better results than adults, but this is not the case. It is quite the opposite, owing probably to the greater tendency of children to eczema of the skin adjacent to the wound.

The extension of the osteitic process was, in most of the cases, very considerable. In one case only was it confined to the parts about the mastoid antrum; in 37 cases it extended over the greater part of the mastoid process, and the whole mastoid process was involved in no less than 60 cases; in 30 of these last cases the apex was removed *in toto*. The cavities on the whole were therefore very considerable, and by ordinary methods the after-treatment would have taken a long time. The circumstance that the dura mater was exposed in 50 and the sigmoid sinus in 35 cases proves also that the process was generally very extensive.

The following complications were present at the original operation: subperiostal abscess 44 times (of these 5 together with subcutaneous abscess), abscesses of the neck 6, abscessus perisinuosus 3, perisinuitis (granulations on the walls of sinus) 20 times, and pachymeningitis externa twice. None of these complications lessen the prospects of healing *per primam*, if only one waits with secondary suture until there is good

formation of granulations in all the parts and until all swelling of the soft parts has disappeared.

The nature of the pathogenic germ seems to be unimportant ; a great number of cases where the resection showed pus with streptococci healed *per primam.*

The results obtained were as follows : In 24 cases, viz. a little more than a quarter of the total number, there was suppuration of the clot. The wound had to be reopened and the cavity dressed in the usual way, healing taking on an average 66 days. In one case, that of a girl aged 10, where the blood-clot suppurated in consequence of post-operative angina, I performed a second secondary suture 8 days after the first ; the result was an ideal healing *per primam.* This case clearly proves how accidental circumstances can compromise the result, which is satisfactory when such can be avoided.

In 7 cases there was suppuration of the axial part of the clot ; a slight opening was made in the wound and a small iodoform gauze plug inserted. Healing occurred in these cases in from 28 to 40 days.

In 24 cases there were slight superficial cracks in the wound, small necrotic parts in the wound or around the Michel's clips, causing a more protracted healing than in cases of ideal healing *per primam.* In this group the healing took from 16 to 30 days from the date of the resection.

In 42 cases there was ideal healing *per primam,* the wound being completely healed in from 12 to 21 days from the date of the resection of the mastoid process.

Primary healing was then practically obtained in about two-thirds of the cases, and in the cases where it was not obtained the after-treatment was appreciably diminished.

The following figures will show the extent to which this method abbreviates the after-treatment of resection of the mastoid process. As I have already mentioned, 74 days is the average time at the Copenhagen Commune Hospital's Ear and Throat Department for the closing of the cavity in the mastoid process with the usual treatment, whilst the corresponding figures for cases of secondary suture, which healed practically speaking *per primam,* was only 20 days.

The following case may be quoted as an especially good instance of the happy results which may be obtained.

No. 25, 1913. A boy, one year old, was admitted on January 22, 1913, suffering from bilateral mastoiditis contracted during pneumonia ; on the right side there was a subperiosteal abscess. The operation revealed a diffuse acute osteitis in both mastoid processes. Ten days later secondary suture was performed on both sides. Ideal healing *per primam* was obtained and the patient was free from bandages 14 days after the operation.

In every case of failure I have endeavoured to discover the causes why the clot has become infected and suppurated, with the following results : eczema of the lips of the wound in 8 cases, general disease in 2,

tuberculosis in the mastoid process in 3, post-operative angina in 4, bad granulation and ill-nourished skin in 2, hæmorrhage in 2, and causes unknown in the remainder. As eczema is frequently the cause of the formation of small necrotic parts in and about the wound, it is evident that the condition of the skin is of the greatest moment in the attainment of favourable results.

This being my experience, I have endeavoured by various means and various methods of bandaging to get the skin of the mastoid process into as good a condition as possible before using secondary suture. Numerous experiments have convinced me that the method here described, both before and after operation, is that which is most reliable. I must therefore impress all confrères who may try my method with the necessity of closely following the instructions I have here given.

DISCUSSION

Professeur Moure (Bordeaux). Si j'ai bien compris la communication de notre collègue M. Mygind, il conseille de fermer la plaie rétro-auriculaire de l'antrotomie dix à douze jours ou même trois semaines après l'opération. Or, il y a déjà bien des années que je conseille et que je fais la réunion immédiate de cette plaie en laissant un simple drain dans l'antre. Dans ces cas, si le curettage mastoïdien a été complet, si la région a été bien désinfectée, lors du premier pansement non seulement l'oreille ne coule plus, mais le drain contient à peine un peu de sérosité sanguinolente. En faisant alors des pansements aseptiques on obtient la guérison entre trois ou quatre semaines au maximum et la cicatrice rétro-auriculaire est nulle. Si, au contraire, la plaie suppure, pour des raisons que je n'ai pas à envisager ici, on est quitte pour faire sauter deux ou trois points de suture, panser à plat pendant quelques jours, et ensuite faire la réunion secondaire dont a parlé notre confrère M. Mygind. Dans ces cas encore la cicatrice peut être nulle ou peu apparente. Il est inutile de mettre un caillot, il se fait tout seul par le sang qui s'écoule une fois l'opération terminée et la plaie suturée. Cette pratique de la réunion immédiate faite dans les conditions indiquées ci-dessus a les avantages de faciliter la cicatrisation rapide et de ne pas laisser les cicatrices disgracieuses, horribles parfois, des plaies rétro-auriculaires pansées à plat. Telle est la pratique que je suis systématiquement depuis plus de vingt ans sur plus de 600 opérés et j'en ai toujours retiré d'excellents résultats.

Professor Kubo (Fukuoka, Japan). Im Jahre 1911 habe ich einmal über die guten Resultate der sekundären Nahtanlegung nach einfacher Mastoidoperation berichtet. Seitdem tue ich regelmässig die sekundäre Nahtanlegung bei der unkomplizierten akuten Mastoiditis wenn die folgenden Bedingungen zustande kommen : 1. der äussere Gehörgang

muss trocken sein ; 2. die Granulation muss gesund sein ; 3. keine grosse Eiteransammlung darf in der Wundhöhle vorhanden sein. Diese Bedingungen folgen gewöhnlich in ein bis drei Wochen nach der Operation. Primäre Nahtanlegung direkt nach der Operation finde ich gefährlich, da ein versteckter Eiterherd vorhanden sein kann. Regelmässig operiere ich unter Lokalanästhesie, selbst bei Kindern. Die Mastoidealspitze nehme ich regelmässig weg, da die tote Höhle dadurch bedeutend verkleinert und die Gefahr des Zurückbleibens des Eiterherdes vermindert wird.

Dr. Gorham Bacon (New York). I have been very much interested in the paper of Professor Mygind, for I have used for several years a very similar method and have found it most satisfactory. I never had much success with the so-called blood-clot method as advocated by Dr. Blake, of Boston, and I have abandoned it. I employ the method as described by the speaker more especially in cases where I have performed the Schwartze-Stacke operation, and where the healing is very slow, and I have had much success with it, and I wish to endorse everything that Professor Mygind has said.

Dr. F. L. Jack (Boston). Professor Mygind's method of secondary suturing after having allowed the mastoid wound to fill with blood-clot has all the elements of safety and is not open to the same objections as the primary blood-clot method. Dr. Sprague suggested to me also the blood-clot dressing, and I have tried it a number of times. It seems to me poor surgery, and in most of my cases has been unsatisfactory. The conditions under which Professor Mygind employs the blood-clot is certainly safer than the primary use of it and I should think it a far preferable procedure.

Professor Alexander (Vienna), recommends (1) the use of only sterilized wicks, dusted with any kind of antiseptic powder ; (2) the frequent use of antiseptic gauze ; (3) the repeated change of dressing in all kinds of cases with much wound secretion, after the operation, for the purpose of an exact and good drainage, until it reaches one or two changes of dressing a day.

Il professore Ferreri (Roma). La questione posta dal collega Mygind è di capitale importanza per la pratica chirurgica otoiatrica, mirando a ridurre il tempo eccessivamente lungo delle medicature postoperative. Anche noi nella clinica di Roma procuriamo di regolarci in modo da portare al più presto a guarigione gli operati di mastoiditi acute, seguendo le seguenti norme : 1° Sutura periosto-cutanea per prima ipercutanea quando si tratta di mastoiditi acute da stafilococchi, pneumococchi, limitate alla mucosa di rivestimento dell' antro, senza lesione ossea. 2° Sutura periosto-cutanea dopo 10–15 giorni nelle mastoiditi d'altra

origine, allorchè il fondo della ferita granuli bene e non vi sia perdita di sostanza esagerata.

Professor DENKER (Halle a. d. Saale) hat mit der primären und der sekundären Naht besonders bei Kindern gute Erfolge gesehen ; er warnt jedoch vor der primären Naht in den Fällen, wo in Rücksicht auf die allgemeinen Symptome schon frühzeitig, d.h. zu einer Zeit operiert wird, wo noch keine grösseren Hohlräume durch die Einschmelzung der Zellenwände gebildet sind und sich der Prozess deswegen nicht gut abgrenzen lässt. Redner hat nach primärer Naht in zwei Fällen tiefe extradurale Abszesse entstehen sehen mit Arrosion des hinteren Bogengangs.

REPLY

Professor MYGIND (Copenhagen). The interest taken by so many speakers in my paper shows that there is a general desire to shorten the after-treatment following mastoid operations, and several of the speakers have used my method almost exactly as I have described it. The method seems, therefore, so to speak, to lie in the air.

To Professor Moure I should say that my experience with primary suture is not so good as his ; there are besides many cases where it cannot be used—for instance, in cases of subperiosteal abscess, &c.

Professor Kubo generally lets three weeks elapse before using secondary suture, but in a great many cases it is possible to do it earlier. A slight discharge from the auditory meatus does not in most cases prevent primary healing.

The use of sterile dressing only, as suggested by Professor Alexander, has the disadvantage that the dressing must be changed much more frequently than when antiseptic dressing is used, and I consider this a great drawback in children, who are often—at least in my country—difficult to manage during the after-treatment.

The character of the infection, as mentioned by Professor Ferreri, whether streptococcal or otherwise is—according to my experience—without any influence on the healing process.

In response to Professor Denker I may state that it seems as easy to get primary healing in large osseous wounds as in small ones.

I am glad to hear that Dr. Gorham Bacon and Dr. Jack, like most of the other speakers, have had the same favourable experience as I have had with secondary suture.

SECTION XVI

OTOLOGY

INDEPENDENT PAPER

DE L'OSTÉOMYÉLITE DU ROCHER

(Observations nouvelles)

Par M. le Dr. BAR, Chirurgien Oto-Rhino-Laryngologiste des Hôpitaux de Nice (France)

Au cours des affections inflammatoires aiguës ou chroniques de l'oreille ou même spontanément sans maladie antérieure,[1] se déclare parfois, d'une manière soudaine, une complication extrêmement grave, douloureuse, ayant quelque analogie avec l'ostéite mastoïdienne et qui sous forme d'inflammation osseuse rapidement nécrosante et diffuse à l'ostentation constitue l'ostéomyélite du rocher. Cette affection rare n'a attiré l'attention que depuis que Kuster, Bergmann en Allemagne, Lannelongue en France ayant démontré que l'ostéomyélite ne frappe pas seulement l'épiphyse des os longs mais aussi le tissu spongieux des os du crâne, Pauzat [2] d'abord, 1893, Schilling [3] ensuite, 1904, eurent décrit l'ostéomyélite du temporal comme complication des otorrhées. Au jeune âge surtout, les maladies intercurrentes et antérieures et les rétentions purulentes déterminées paraissent sans spécificité de tel ou tel microbe, la cause probable de la diffusion du processus infectieux à travers le tissu spongieux du rocher.

Observation Ire (personnelle). — Ostéomyélite du rocher consécutive à une otite suppurée chronique chez un scrofuleux.

C. P., âgé de 10 ans, Italien, habitant Nice, se présente à la consultation le 12 février 1913, se plaignant de violentes douleurs dans la tête et d'une tuméfaction énorme de la région mastoïdienne, survenue très rapidement depuis la veille, toutes choses qu'il attribue à un coup de froid sur son oreille droite en suppuration depuis une rougeole de son bas âge. Cet enfant est issu de parents bien portants.

Le malade est abattu, d'un facies qui dévoile ses souffrances ; avec un aspect terreux, un état fébrile et anxieux, indice d'une profonde intoxication.

[1] Pearson, *British Medical Journal*, mars 1883.
[2] Pauzat, *Annales de Laryngologie*, septembre 1893.
[3] Schilling, *Zeitschr. f. Ohrenheilk.*, décembre 1904.

L'attention est aussitôt attirée du côté de l'oreille par un écoulement fétide, formé d'un pus grisâtre qui sort d'une façon continue du conduit auditif externe, et aussi par ce fait que le pavillon de l'oreille semble abaissé, décollé et tuméfié. L'examen de la région auriculaire révèle en effet un gonflement généralisé à la partie postérieure, empâtement dur, œdémateux, ressemblant à celui du phlegmon diffus, mais en différant en ce qu'il n'est pas rouge franchement, que tendu, il cesse brusquement au point présumé où s'arrête l'altération osseuse. Ce gonflement s'étend en haut de façon à dépasser un peu la crête sous-mastoïdienne, dépasse de beaucoup l'apophyse mastoïde qu'il déborde largement en arrière, et inférieurement semble gagner de proche en proche toute la région carotidienne, ayant déjà franchi la pointe de la mastoïde. La palpation de cette région œdématiée et douloureuse dans toute la région postérieure de l'oreille ; l'œdème garde facilement l'empreinte du doigt, mais nulle part on ne constate de fluctuation nette.

Fièvre autour de 40,5°. Ni frissons, ni vertiges.

Première opération. — Opéré le 15 février par un de nos confrères, en notre absence. Large incision rétroauriculaire qui mène sur l'os nécrosé. Issue de pus fétide, abondant. Trépanation mastoïdienne, simple. Chute de la température à 37°. Mais le deuxième jour de l'opération la température revient à 38,5°.

Lorsque nous sommes amenés à visiter le malade nous trouvons une suppuration fétide profuse avec lésions d'ostéite très étendue. Os mastoïdien dénudé, noir, d'aspect nécrotique sur sa table externe. A travers la brèche on voit cet os mou et friable, celluleux, infiltré d'un pus grisâtre et fétide. Les lésions osseuses paraissent s'étendre très loin en arrière et l'œdème se diffuse davantage vers le bas du cou. L'observation journalier indique que l'état général est mauvais, la température s'accroît de façon à monter à 39,6°. Le malade se cachectise de plus en plus. Pansements quotidiens par lavages à la solution phéniquée à 4 pour cent. La fièvre s'atténue et la température s'abaisse à 37°, mais ce n'est qu'une accalmie provisoire.

Seconde opération le 22 février avec notre distingué confrère le docteur Sargnon, de Lyon. En présence de la persistance de tous ces symptômes nous nous mettons en mesure de pratiquer un évidement aussi complet que possible de la mastoïde. L'os mou et friable se laisse enlever facilement à la pince-gouge et à la curette et nous pratiquons un évidement complet jusqu'à la pointe qui est celluleuse et infiltrée. Le sinus est dénudé sur plus d'un centimètre, mais devient tout à coup le siège d'une hémorragie lorsque, nous dirigeant vers les parties hautes, nous venons à détacher quelques fongosités périsinusiennes qui aveuglaient une ulcération existante de ce gros vaisseau sanguin. Forcés aussitôt d'arrêter l'opération nous faisons un tamponnement du sinus à la gaze iodoformée après avoir pratiqué à l'eau oxygénée à 12 vol. dédoublée, un soigneux lavage de la région opérée. Curettage des fongosités, ablations d'esquilles volumineuses, de séquestres vers les zones postérieures en haut au-dessus du sinus, et en bas vers la pointe ont rendu la région aussi nette que possible, sans nous laisser néanmoins la certitude que toutes parties nécrosées aient été enlevées. C'est ainsi que vers les parties profondes du coli du massif osseux du facial, du côté des canaux semi-circulaires, et en bas du côté du plancher de la caisse et vers l'apophyse styloïde, un stlyet dirigé perçoit des rugosités symptomatiques d'os nécrosé, laissant ainsi envisager avec méfiance l'avenir du malade. Dans les profondeurs on est naturellement limité, car non seulement l'abord n'est pas facile, mais rendu dangereux par les nerfs et les vaisseaux qui s'y trouvent.

En effet trois jours après cette importante opération le malade fait

une poussée fébrile à 39,4° remontant ainsi de 38° où il était descendu ; le cou devient luisant, plus empâté et on perçoit bientôt que les glandes de la région carotidienne se prennent d'inflammation purulente que nous libérons bientôt par des incisions appropriées sur cette région dangereuse du vaisseau. (Cette troisième opération fut faite le 6 mars.)

Dès les premiers jours qui suivent cette troisième opération, une amélioration se déclare ; la fièvre tombe, l'état général devient meilleur et un bourgeonnement convenable se fait dans toute la région opérée. Aussi, réserve faite au sujet des points d'ostéite qu'on sent dans les diverses zones du rocher, il y a-t-il lieu d'espérer une guérison rapide ? En effet, quatre semaines après, la santé générale paraissait retenue, mais était à faire localement pour un point douteux de la région mastoïdienne opérée. Là, en effet, à travers quelques bourgeons atones et saignants au niveau de la région antrale, un stylet s'enfonce profondément et perçoit un os dénudé. Un mois après on remarque, allant en s'accentuant, un peu de déviation de la face, le facial étant sans doute intéressé par la marche progressive du mal. On ne peut désormais que manifester des craintes et devenir suspect quant à la possibilité et au résultat d'opérations curatives futures.

La région, à n'en pas douter, a été non seulement atteinte dans son ensemble, mais encore après la violence de l'attaque initiale paraît n'avoir point cessé sa marche progressive vers les couches profondes, attaque brusque et marche progressivement envahissante caractérisant ce genre d'ostéomyélite.

Ce n'est point la première fois que nous observons des cas de ce genre. En voici le résumé d'un autre que nous avons publié autrefois et dont la brusquerie et les résultats de l'attaque peut faire songer à une véritable ' apoplexie osseuse '.

Observation II. — Ostéomyélite du rocher pendant l'évolution d'une scarlatine compliquée d'otites purulentes doubles.

En février 1905 nous vîmes dans la soirée un enfant de sept ans qui, en pleine scarlatine, avait été pris depuis deux jours d'un écoulement otorrhéique double. Dans le courant de la journée il s'était plaint d'une douleur derrière l'oreille droite. Au moment de notre examen, non seulement elle était persistante et accrue, mais la région était déjà œdémateuse. Quelques heures avaient suffi à le mettre dans cet état. L'écoulement auriculaire se faisait bien. La fièvre était autour de 39°. On décida de l'opérer le lendemain à la première heure. Une large incision sur la région mastoïdienne conduit sur cet os déjà exsangue, mat, privé de circulation et de vitalité. La trépanation fut analogue à celle qu'on eut pratiquée sur un os de cadavre, et donna fort peu de pus : os nécrosé nettement défini quant à ses trabécules qui paraissent vides et friables. On eut dit d'un os où la circulation et la vie étaient arrêtée, tandis que l'antre en suppuration et le conduit auditif suppurant indiquaient l'abcès phlegmoneux dont la région avait été atteinte.

Il n'y eut pas de marche envahissante. Un séquestre superficiel de toute la région mastoïdienne se forma et par bourgeonnement sous-jacent se limita dans l'espace d'une année, après laquelle nous pûmes en faire l'exérèse. La guérison du malade fut complète en trois points malgré un terrain lymphatique et des antécédents familiaires suspects de tuberculose.

Dans ce second exemple la brusquerie d'attaque et ses effets nécrotiques paraissent avoir été équivalentaires d'une véritable apoplexie de l'os, ainsi qu'en témoigne encore l'état dans lequel il fut trouvé à l'ouver-

ture opératoire. Cette rapidité d'évolution initiale aussi bien que la marche insidieuse et envahissante de l'ostéomyélite mise en lumière par des travaux récents (Schilling, Luc, Guisez [1]) mérite quelque attention.

Classiquement, en effet, nous savons que pendant l'enfance et l'adolescence, lorsque les os qui s'accroissent sont le siège d'une grande activité nutritive, on peut observer des accidents d'une gravité très variable et qui vont d'une hyperhémie, d'une congestion simple à une suppuration totale. Une cause morbide quelconque, diathésique (scrofule, diabète, tuberculose, syphilis, etc.), une maladie antérieure, intercurrente (une otorrhée,[2] une sinusité frontale [3]), lorsqu'il s'agit des os du crâne provoque les accidents et ceux-ci affectant la forme inflammatoire aiguë évoluent d'une manière plus ou moins rapide sous les types de ' périostite circonscrite et d'ostéomyélite phlegmoneuse diffuses '. Dans des cas tout à fait exceptionnels ces accidents peuvent même se manifester sous une forme foudroyante dont la deuxième observation est un bon exemple.[4] En quelques heures, dans ce cas, l'os y avait été partiellement gangréné, d'une façon irrémédiable, comme s'il avait été frappé d'une véritable apoplexie. C'est qu'avec leur richesse en tissu spongieux les os du crâne sont éminemment propres au développement et à la diffusion du processus ostéomyélitique, pathogénie à laquelle la disposition et le développement du réseau sanguin intra-osseux contribue encore. Pearson [5], sous le nom de ' nécrose aiguë des os du crâne ', remarqua leur évolution rapide. Ce sont des cas d'ostéomyélite qui parfois, faute d'en distinguer la cause, sont peut-être envisagées comme primitives et qui peuvent toujours être considérées comme les analogues des ostéomyélites spontanées de Reynaud. Leur évolution nécrotique initiale, sans être fréquemment foudroyante est toujours très rapide, à peine quelques jours, comme les cas présents en témoignent. En moins d'une semaine en effet après le début des accidents on rencontre du pus, quelquefois même sous l'aponévrose, s'il y a eu effraction de la table externe de la mastoïde, pus jaune verdâtre, contenant un grand nombre de globules de graisse et de gouttelettes huileuses. Un stylet introduit au fond de la plaie perçoit un os dénudé, et parfois très au loin.

Un foyer osseux apparaît souvent dans un ou plusieurs autres points, permettant de se prononcer en faveur de l'ostéomyélite, ainsi que c'était le cas du malade que nous signalons aujourd'hui.

Non moins intéressante est la marche de l'affection, soit en effet, à la faveur d'une disposition spéciale du diploé, soit à celle d'une virulence particulière des germes infectieux ayant présidés à son déve-

[1] Guisez, ' Ostéomyélite des os plats du crâne consécutive dans otites et sinusites suppurées,' *Rapport à la Société française d'Otologie, Rhinologie et Laryngologie*, Paris, 1906.

[2] Pauzat, *Annales de Laryngologie*, 1893.

[3] Schilling, *Zeitschr. f. Ohrenheilk.*, 1904.

[4] Bar, ' De la nécrose aiguë des os du crâne,' *Société française d'Oto-Rhino-Laryngologie*, Congrès de Paris, 1907.

[5] Pearson, *British Medical Journal*, 1883.

loppement, cette marche est insidieuse et progressivement envahissante. Les observations curieuses de Schilling,[1] de Laurens,[2] de Broeckaert,[3] de Luc,[4] de Guisez,[5] de Richardson [6] (de Washington) montrent qu'elle peut faire littéralement le tour de la calotte crânienne. Uniquement cantonnée au rocher, elle n'en est pas moins extrêmement dangereuse et parfois rapidement mortelle. Après avoir en effet rayonné dans toutes les directions, conformément au schéma si lucide que Lermoyez [7] a dressé par la marche des mastoïdites aigus, et occasionné, malgré les opérations les plus hâtives et les plus radicales, la nécrose rapide des parties atteintes, l'ostéomyélite du rocher, dans certains cas heureusement rares, continue sa course par continuité osseuse aussi bien que par voie vasculaire, elle avance et malgré la barrière que lui présente le massif osseux du facial, elle parvient à gagner en profondeur.

Dès lors la participation du facial, la participation de la dure-mère, la participation du sinus sont devenus inévitables ; la paralysie faciale, la pachyméningite, les abcès du cerveau et du cervelet, la thrombose du sinus et principalement du sinus latéral en sont la conséquence. Enfin après une marche essentiellement rémittente, avec accalmie, après chaque intervention, le malade aboutit à une issue fatale que les délabrements opératoires les plus étendus n'ont pu éviter, n'ayant pu suffire à enrayer l'infection ostéomyélitique.

[1] Schilling, *Zeitschr. f. Ohrenheilk.*, 1904, p. 52.
[2] G. Laurens, *Congrès de Paris*, 1900.
[3] Broeckaert, *Gazette hebdomadaire de Med. et Chir.*, 1899.
[4] Luc, *Ann. de Laryngologie*, 1905.
[5] Guisez, *Archives de Laryng.*, 1905, et *Rapport à la Soc. d'Otologie*, 1906.
[6] Richardson (de Washington), *Archiv. of Otol.*, i, 1904.
[7] Lermoyez et Boulay, *Thérapeut. des maladies de l'oreille*, i. 400.

SECTION XVI

OTOLOGY

DISCUSSION No. 2

THE PATHOLOGY OF ACQUIRED DEAFMUTISM

(For Report by Dr. H. Mygind, see Part I, pp. 291–307)

Referat von Professor Dr. ALFRED DENKER, Halle a. S.

Die Pathologie der angeborenen Taubstummheit

In dem nachstehenden Berichte sollen die *Pathogenese* und die *pathologische Anatomie* der *angeborenen*, d. h. der *intrauterin entstandenen* Taubstummheit behandelt werden, gleichviel, ob in dem einzelnen Falle das Gebrechen durch eine intrauterine Meningitis oder infolge syphilitischer Placentarinfektion erworben oder aber durch Entwicklungsstörungen irgendwelcher Art bedingt war. Bevor wir uns der Schilderung der Entstehung der Krankheitsbilder und der charakteristischen pathologischen Veränderungen zuwenden, ist es notwendig, auch an dieser Stelle auf die von verschiedenen Seiten aufgestellten Prinzipien der Einteilung der Taubstummheit kurz einzugehen.

Die von altersher gebräuchliche und schon Aristoteles bekannte Einteilung unterscheidet eine *angeborene* und eine *erworbene* Form der Taubstummheit, je nachdem die pathologischen Veränderungen im Gehörorgan *intrauterin* oder *nach der Geburt* entstanden sind. Dass dieser ältesten Einteilung gewisse Mängel anhaften, ist gerade durch die in den letztverflossenen Jahrzehnten vorgenommenen histologischen Untersuchungen immer deutlicher hervorgetreten. Die sorgfältige Forschung hat ergeben, dass es oftmals sehr schwierig ist, zu entscheiden, ob die dem Leiden zu Grunde liegenden krankhaften Veränderungen im inneren Ohr intrauterin entstanden oder im ersten oder zweiten Lebensjahr im Anschluss an eine abortiv verlaufene Meningitis erworben sind. Die von den Eltern des taubstummen Kindes angegebenen anamnestischen Daten werden uns bezüglich der Aetiologie des Gebrechens in einzelnen Fällen irre führen, nicht nur weil für das innere Ohr verhängnisvolle Krankheiten von den Angehörigen übersehen werden, sondern andererseits auch von den Eltern bisweilen Affektionen im frühen Kindesalter, welche für das Gehörorgan bedeutungslos sind, irrtümlich als die Ursache einer in

Wirklichkeit intrauterin entstandenen Taubheit angeführt werden. Die Schwierigkeiten bei der Einteilung vermehren sich weiterhin noch dadurch, dass nach den neueren Untersuchungen die Möglichkeit einer im *Fötalleben* im Anschluss an eine intrauterine Meningitis oder im Gefolge einer syphilitischen Placentarinfektion auf *entzündlicher* Basis sich entwickelnden Labyrinthitis als Ursache für die Zerstörung des Gehörorgans durchaus zugegeben werden muss; wir werden demnach bisweilen bei der histologischen Untersuchung des Ohres die gleichen Bilder bei einer intrauterin, aber auf entzündlichem Wege entstandenen Taubheit finden, wie bei dem im Leben erworbenen Gebrechen.

Diese der alten Einteilung anhaftenden Mängel haben im Jahre 1902 den Wiener Forscher Victor Hammerschlag veranlasst, neue Vorschläge für die Bezeichnung der verschiedenen Formen der Taubstummheit zu machen. Auf Grund seiner sorgfältigen, an kretinischen Taubstummen vorgenommenen Untersuchungen schlägt er vor, die Taubstummheit in folgende zwei Hauptgruppen einzuteilen:

I. Die durch *lokale* Erkrankung des *Gehörorgans* bedingte Taubstummheit.

Diese Form ist der Ausdruck einer *auf das Gehörorgan beschränkten* Erkrankung (Entzündung, Exsudation, Blutung, Trauma) in einem *sonst gesunden* Körper; sie ist immer — fötal oder postfötal — *erworben* (Meningo-Encephalitis, durch Placentarinfektion erworbene Syphilis).

II. *Die konstitutionelle Taubstummheit.*

Bei dieser Form ist das Gebrechen aufzufassen als *Teilerscheinung* einer *allgemeinen konstitutionellen Anomalie* des betroffenen Individuums. (*Fehlerhafte Keimesanlage.*) Die konstitutionelle Taubstummheit teilt Hammerschlag dann weiterhin ein in:

(*a*) die *endemische* Form (kretinische Taubstummheit),

(*b*) die sporadische Form (*hereditär-degenerative* oder *kongenitale* Taubstummheit).

Nach Bircher ist die endemische Form der Taubstummheit auf dieselbe endemische Noxe zurückzuführen, die für die Entstehung des endemischen Kretinismus verantwortlich gemacht werden muss, sie sei demnach nur ein Glied in der Kette der *kretinoiden Degeneration.* Die letztere ist aufzufassen als der Ausdruck einer durch den Genuss des *Trinkwassers* gesetzten chronischen Schädigung der Schilddrüse, einer chronischen *Hypo-* resp. *Athyreosis.* Die Hypothyreosis führt zu den bekannten Erscheinungen des endemischen Kretinismus, wenn sie in die Entwickelungsperiode, in die Kindheit resp. in das Embryonalleben zurückdatiert. Es muss demnach die endemische Taubstummheit in vielen Fällen als ein fötal *oder* postfötal erworbener Zustand aufgefasst werden. Dazu kommt, dass die erworbene Hypothyreosis der Erzeuger zu einer in dem gleichen Sinne wirkenden *Disposition* bei den Kindern führt, so dass gewisse Formen der kretinischen Degeneration sich ätiologisch als ein *Produkt* aus der *erworbenen* und der *hereditär bedingten* Funktionsherabsetzung der Schilddrüse darstellen (cf. Hammerschlag, ‘ Ein neues Einteilungsprinzip

für die verschiedenen Formen der Taubstummheit,' *Archiv f. Ohrenh.* Bd. lvi, S. 177).

Unter der sporadischen Form der konstitutionellen Taubstummheit, die er auch als *hereditär-degenerative* oder *kongenitale* Form bezeichnet, versteht Hammerschlag die durch *Konsanguinität* der — aus einer *hereditär-degenerativen* Familie stammenden — Erzeuger bedingte oder mit *sonstigen hereditär-degenerativen Symptomen* vergesellschaftete Taubstummheit. Als hereditär-degenerative Symptome sind nach E. Urbantschitsch aufzufassen: Retinitis pigmentosa, ungleiches Brechungsvermögen beider Augen, Reste der Pupillarmembran, Epikanthus, ungleiche Irisfarbe, Strabismus, partieller Albinismus, Schädeldeformitäten, Wolfsrachen, Abnormitäten der Gaumenbogen, Kryptorchismus und Syndaktylie, Cerebellar-ataktischer Gang.

Wenn wir die Hammerschlag'schen Vorschläge einer kritischen Betrachtung unterziehen wollen, so lässt sich nicht leugnen, dass beim ersten Blick die Abgrenzung einer durch Konstitutionsanomalien bedingten Taubstummheit von einer durch lokale Erkrankung des Gehörorgans erworbenen Taubstummheit mancherlei für sich hat, und dass es auch wohl in vielen Fällen gelingen dürfte, an der Hand der Anamnese und der objektiven Untersuchung diese beiden Hauptgruppen voneinander zu unterscheiden. Es scheint mir allerdings zweifelhaft, ob alle Fälle, welche nicht als kretinische oder hereditär-degenerative Taubstummheit aufzufassen sind, als erworben bezeichnet werden müssen in dem Sinne, dass sie entzündlichen Prozessen oder einem Trauma ihre Entstehung verdanken; ich halte es für möglich, dass auch noch andere ätiologische Faktoren für das Auftreten von Bildungsanomalien im Ohre in Betracht kommen können. — Hammerschlag sagt ferner, dass die Fälle seiner I. Hauptgruppe durch lokale Erkrankung in einem *sonst gesunden* Körper bedingt sind; dagegen ist zu bemerken, dass die in den ersten Lebensjahren infolge einer bei Meningitis oder schwerer Scharlachotitis erworbene Labyrinthitis sich doch keineswegs in einem gesunden Organismus abgespielt hat.

In seinen weiteren Ausführungen plädiert Hammerschlag dafür, die hereditär-degenerative Taubstummheit speziell als *kongenital* zu bezeichnen; damit könnte man sich einverstanden erklären, wenn Hammerschlag nicht die Bezeichnung '*kongenital*' als gleichbedeutend mit dem Ausdruck '*in der Keimesanlage begründet*' gebrauchen würde. Durch diese Auffassung setzt er sich in *Widerspruch mit der Ansicht sämtlicher Biologen und der Vertreter der pathologischen Anatomie*; *kongenital* bedeutet nicht das Gleiche wie '*angezeugt*', sondern *kongenital* ist '*mitgeboren*' oder '*angeboren*', es ist ein Sammelbegriff, unter dem man allgemein *alles zusammen* versteht, was im intrauterinen Leben teils durch entzündliche Vorgänge *erworben* wird, teils *fehlerhaft angelegt* war.

Wenn Hammerschlag ferner meint, dass die *hereditär-degenerative* Form der Taubstummheit natürlich *nicht erworben* sein kann, da sie in der Keimesanlage begründet sei, so lässt sich dagegen nichts einwenden; nur

werden uns keinerlei Mittel an die Hand gegeben, diese Form *pathologisch-anatomisch* von anderen Formen der angeborenen Taubstummheit zu unterscheiden. Die einfache anamnestische Feststellung der Tatsache, dass die hereditär-degenerativ belasteten Erzeuger eines taubstummen Kindes miteinander verwandt sind, kann uns keineswegs genügen, da auch ein *normalsinnig* geborenes Kind solcher Eltern im frühen Kindesalter durch *Meningitis* Taubheit erwerben kann.

Hammerschlag macht keine Vorschläge, wie seine I. Hauptgruppe und die beiden Unterabteilungen seiner II. Hauptgruppe nun nach den anatomischen Befunden weiterhin einzuteilen sind, und ich glaube deswegen, dass seine Prinzipien der Einteilung und Unterscheidung der verschiedenen Formen der Taubstummheit uns nicht wesentlich weiter gebracht haben.

Mit der Hammerschlag'schen Einteilung kann sich auch Goerke nicht einverstanden erklären ; er lehnt sie als zur Zeit undurchführbar ab und ist der Ansicht, dass uns klinische und ätiologische Einteilungsprinzipien vorläufig im Stich lassen. In seiner im Jahre 1908 erschienenen Arbeit, *Pathologie der Taubstummheit* (' Ergebnisse der Pathologie ' von Lubarsch-Ostertag) schlägt Goerke eine Einteilung nach entwicklungsgeschichtlichen Gesichtspunkten vor. Er unterscheidet zwei Gruppen :

1. *Embryonale Taubstummheit.*
2. *Postembryonale Taubstummheit.*

Unter die 1. Gruppe werden diejenigen Veränderungen gerechnet, welche das Ohr *während seiner Entwicklung* treffen, also seinen Aufbau, seine Form, die Ausbildung seiner einzelnen Teile stören (= *Missbildungen*, und zwar meistens *Hemmungsbildungen, selten Excessivbildungen*).

Zu der 2. Gruppe gehören alle übrigen Formen von Taubstummheit, die bedingt sind durch Veränderungen, welche das Ohr erst *nach vollendeter Entwickelung* treffen.

Auch diese von Goerke empfohlene Einteilung bringt uns meines Erachtens nicht aus den bestehenden Schwierigkeiten heraus. Denn die Frage, ob bei mangelhaften anamnestischen Daten ein Fall ausschliesslich nach dem pathologisch-anatomischem Bilde als *embryonal* oder *postembryonal* anzusehen ist, wird meistens ebenso schwer zu entscheiden sein wie die Frage, ob *angeborene* oder *erworbene* Taubstummheit vorliegt. Als *sicher embryonal* entstanden sind wohl nur die Fälle von *Aplasie des Schläfenbeins* zu betrachten. Nachdem die neueren Forschungen ergeben haben, dass jede *Missbildung hypoplastischer* Art die Folge von *intrauterinen Entzündungen* sein kann, ja, dass auch Labyrinthanomalien bei *Anencephalie* entzündlichen Ursprungs sein können, da die Gehirnveränderung selbst durch entzündliche Prozesse bedingt ist, wird es die grössten *Schwierigkeiten* bereiten, die *Entwicklungsanomalien entzündlicher und nicht entzündlicher* Provenienz voneinander zu unterscheiden.

Dass wir bei der Beurteilung der Pathogenese des einzelnen Falles äusserst vorsichtig sein müssen, wird immermehr klar und besonders durch die von Manasse publizierten Untersuchungsergebnisse illustriert.

Manasse fand bei *chronischer progressiver* Schwerhörigkeit atrophische Degeneration des Cortischen Organs und des Ganglions, Lageveränderungen der Reissner'schen Membran, etc., also Veränderungen, die man früher als sicher kongenital ansah. Er ist deshalb der Meinung, dass es *unstatthaft* ist, eine Taubheit *lediglich auf Grund dieser Befunde als kongenital* zu bezeichnen.

Einer anderen Auffassung in Bezug auf die Beurteilung der angeführten pathologischen Labyrinthveränderungen redet — wie mir scheint, nicht mit Unrecht — Hammerschlag das Wort, indem er sagt: 'Wenn wir in den Fällen vom Typus Scheibe (Taubstummheit durch Epithelmetaplasie und Lageveränderungen im Ductus cochlearis) neben den sicher kongenitalen Befunden auch solche finden, die zum Bilde der von Manasse als erworbene Krankheit angesprochenen labyrinthären Taubheit gehören, dann wird nicht die Frage entstehen, ob die Fälle von Typus Scheibe erworben sind, sondern vielmehr die Frage, ob nicht die Fälle von Manasse "*Spätformen einer kongenital* angelegten Veränderung des Gehörorgans" sind.'

Um diese Hypothese zu stützen, zieht Hammerschlag die Otosklerose als eine ebenfalls von hereditären Einflüssen zweifellos abhängige Affektion zum Vergleiche heran; er weist darauf hin, dass es Familien gibt, in denen *ererbte Taubheit* mit *Otosklerose* zusammen beobachtet wird, dass ferner in Fällen *kongenitaler Taubstummheit* auch das Vorkommen der für die Otosklerose charakteristischen *Spongiosierungsprozesse* im Knochen konstatiert worden ist. (Fälle von Alexander, Politzer, Lindt.) Sodann sprechen auch die von Alexander und Katz bei Tieren vorgenommenen Untersuchungen, die neben den für die Otosklerose typischen Knochenalterationen noch andere Veränderungen (Pigmentanomalien, Kretinismus u.s.w.) ergeben, für den hereditären Charakter der Otosklerose.

Wenn man endlich die auffallende Uebereinstimmung der atrophisch degenerativen Nervenveränderungen bei der kongenitalen Taubstummheit und der Otosklerose in Betracht zieht, so lässt sich der Gedanke, dass es sich bei beiden Affektionen um verwandte Prozesse handelt, nicht von der Hand weisen.

Halten wir uns vor Augen, dass die hereditär angelegte Otosklerose bekanntlich nicht schon bei der Geburt oder kurz nachher, sondern meistens erst in späteren Lebensjahrzehnten in die Erscheinung tritt, so muss die Möglichkeit zugegeben werden, dass die Hammerschlag'sche Hypothese richtig ist, dass es sich bei der *progressiven labyrinthären* Schwerhörigkeit um *Spätformen* einer *kongenital* angelegten Labyrinthveränderung handeln kann. Und mit Goerke können wir weiterhin annehmen, dass es sich bei der progressiven, labyrinthären Schwerhörigkeit um eine Aufbrauchkrankheit im Sinne Edingers handelt, die dadurch zustande kommt, dass einzelne Nervenbahnen nicht stark genug angelegt worden sind, um den normalerweise an sie herantretenden Anforderungen zu entsprechen.

Auf der Versammlung der deutschen otologischen Gesellschaft in

Dresden (1910) hat E. Urbantschitsch eine Einteilung empfohlen, die sich an die Vorschläge Hammerschlags eng anschliesst ; er teilt ein in :

1. *Erworbene Taubstummheit,*
   (*a*) intrauterin erworben,
   (*b*) postfötal erworben.
2. *Kongenitale Taubstummheit,*
   (*a*) *hereditär-degenerative* Form, die
   α, *manifest*, β, *latent* auftreten kann ;
   (*b*) *endemische* Form.

Bei dieser Einteilung entspricht die *erworbene Taubstummheit* der Hammerschlag'schen 1. Gruppe, der ' durch lokale Erkrankung des Gehörorgans bedingten Taubstummheit ', die *kongenitale Taubstummheit* der Hammerschlag'schen *konstitutionellen Taubstummheit.*

Von den Vorschlägen Hammerschlags weicht die von E. Urbantschitsch gegebene Einteilung insofern ab, als er die hereditär-degenerative Taubstummheit weiterhin in eine manifeste und latente Form zergliedert. Urbantschitsch will unter hereditär-degenerativer Taubstummheit jene Form verstanden wissen, bei der die *Ursache* der Taubstummheit in der fehlerhaften Keimesanlage begründet ist, ohne aber dabei zu präjudizieren, dass dieser ererbte Defekt bereits zur Zeit der Geburt ganz zur Geltung kommen müsste. Er nimmt demnach unter die *latente* Form der hereditär-degenerativen Taubstummheit diejenigen Fälle auf, bei welchen das Kind hörend zur Welt kommt, bei dem sich erst später, eventuell sogar im Anschluss an eine scheinbar auslösende Ursache, Taubstummheit entwickelt, vorausgesetzt, dass sich die kranke Keimesanlage nachweisen lässt.

Mit seinen beiden Hauptgruppen greift Urbantschitsch scheinbar auf die alte Einteilung in *erworbene* und *kongenitale* Taubstummheit zurück ; die Uebereinstimmung ist aber in der Tat nur eine *scheinbare*, denn Urbantschitsch reiht unter die erworbene Taubstummheit nicht nur die Fälle ein, bei welchen das Gebrechen nach der Geburt im Leben acquiriert wurde, sondern auch die intrauterin durch entzündliche Prozesse oder durch Syphilis der Mutter bedingte Taubstummheit. Es muss zugegeben werden, dass die Zusammenfassung aller intrauterin und postfötal erworbenen Fälle der Taubstummheit Manches für sich hat, da es sich im ganzen um ätiologisch gleichartig entstandene Affektionen handelt ; dabei wäre allerdings zu betonen, dass die intrauterin ' erworbene ' Taubstummheit wohl ausschliesslich solche Fälle umfasst, welche durch eine Meningitis oder Meningo-Encephalis bedingt werden, während bei der postfötal erworbenen Taubstummheit ausserdem durch Mittelohrprozesse und Trauma bedingte Labyrinthitiden als ursächliche Faktoren für die Taubstummheit in Betracht kommen.

Keineswegs kann ich mich aber einverstanden erklären mit der von Hammerschlag übernommenen Auffassung der Bezeichnung des Wortes ' kongenital ' ; ich muss nochmals darauf hinweisen, dass *kongenital* nach

der Auffassung der Biologen und Pathologen *nicht* gleichbedeutend ist mit dem Ausdruck 'in der Keimesanlage begründet'; *wir Otologen haben nicht das Recht, den allgemein anerkannten Sinn eines Ausdruckes umzudeuten,* einem Worte eine andere als die allgemein gebräuchliche Deutung zu geben; wollten wir die von Hammerschlag vorgeschlagene und von E. Urbantschitsch übernommene unrichtige Auslegung des Wortes 'kongenital' acceptieren, so würden wir in der Literatur eine *heillose Verwirrung anrichten.*

Wenn E. Urbantschitsch bei seiner Einteilung nur bei der 'kongenitalen' Taubstummheit von einer *endemischen* Form spricht, so wird dabei die postfötal erworbene Form der endemischen Taubstummheit (cf. S. 500) vernachlässigt, die in seine 1. Hauptgruppe, 'erworbene Taubstummheit,' einzureihen wäre. — Erwähnt sei noch, dass E. Urbantschitsch damit einverstanden ist, die von ihm nach *ätiologischen* Prinzipien aufgestellten Hauptgruppen weiterhin nach den *pathologisch-anatomischen* Befunden—wie es Siebenmann vorgeschlagen hat—zu zergliedern.

Aus den vorstehenden Ausführungen ist zu ersehen, dass uns die Vorschläge von Hammerschlag, Goerke und E. Urbantschitsch noch nicht aus den vorhandenen Schwierigkeiten herausführen. Die verschiedenen Mängel, die ihnen anhaften, veranlassen mich, auch heute noch in der gleichen Weise, wie ich es in dem 7. Heft der *'Anatomie der Taubstummheit'* zum Ausdruck gebracht habe, im ganzen und grossen mit einigen von mir angegebenen Modifikationen an der alten Einteilung und der Gruppierung nach den Siebenmann'schen Vorschlägen festzuhalten. Dementsprechend möchte ich empfehlen, die nachstehende Einteilung zu acceptieren:

I. — *Die angeborene oder kongenitale Taubstummheit*; sie umfasst alle Fälle, deren Entstehung zurückzuführen ist auf intrauterine Störungen. Man kann sie einteilen:

(*a*) in Fälle, bei welchen die Veränderungen bedingt sind durch *intrauterine entzündliche Prozesse* — (*Meningo-Encephalitis*, durch Placentarinfektion erworbene *Syphilis*).

(*b*) Fälle von *Bildungsanomalien*, die auf *nichtentzündlicher Basis* entstanden sind. Unter diese Gruppe würden auch die durch *ererbte kretinische* Degeneration (*endemische* Taubstummheit Hammerschlags), die durch *Konsanguinität* der hereditär-degenerativ belasteten Erzeuger bedingte und die mit *sonstigen hereditär-degenerativen* Symptomen vergesellschaftete Taubstummheit (*sporadische* Form der *konstitutionellen* Taubstummheit nach Hammerschlag) fallen.

Diese Gruppe wäre dann weiterhin mit Siebenmann zu zergliedern:

1. in Fälle mit *Aplasie* des *ganzen Labyrinths*,

2. in Fälle, bei welchen das ganze knöcherne und häutige Labyrinth vorhanden, dagegen das *Epithel mehr oder weniger degeneriert* ist.

Bei dieser Gruppe kann man ferner unterscheiden:

(*α*) Fälle, bei denen die *Epithelmetaplasie beschränkt* ist au die *Membrana basilaris*,

(β) Fälle mit *ausgedehnter Epithelmetaplasie*, Fehlen oder mangelhafter Entwickelung des *Sinnesepithels* kombiniert mit *Ektasie* und *Kollapszuständen* der häutigen Labyrinthwand der Pars inferior.

Ob es sich empfiehlt, diese Gruppen noch weiter, wie es Siebenmann vorgeschlagen hat, in verschiedene Typen zu zerlegen, erscheint zweifelhaft, da sich in den letzten Jahren die Fälle gemehrt haben, die sich nicht unter die vorhandenen Typen einreihen liessen.

II. — *Die nach der Geburt erworbene oder postfötale*[1] *Taubstummheit*; diese Gruppe wäre weiterhin einzuteilen:

(*a*) in Fälle *meningitischen Ursprungs*,

(*b*) in Fälle *tympanalen* Ursprungs (im Anschluss an Mittelohraffektionen entstehend),

(*c*) in Fälle mit *primären Labyrinthveränderungen*, bei denen die Labyrinthitis nicht durch Uebergreifen eines entzündlichen Prozesses in der Nachbarschaft (Meningen, Mittelohr) bedingt ist (Mumps, Trauma, Lues acquisita).

Ferner würden unter diese Hauptgruppe die *postfötal erworbene endemische* Taubstummheit zu rechnen sein, wenn es gelingen sollte, sie von der *ererbten* kretinischen Degeneration abzugrenzen.

Wenn wir uns nun der Besprechung der pathologischen Veränderungen zuwenden, durch welche die Taubstummheit bedingt wird, so ist bezüglich der *Lokalisation* dieser Veränderungen auf Grund der vorliegenden histologischen Untersuchungen zunächst zu sagen, dass fast ausnahmslos nur schwere Störungen im *inneren Ohr* als Ursache für die Entstehung des Gebrechens in Betracht kommen. Erkrankungen im Mittelohr, selbst wenn sie mit dem Verlust der grossen Gehörknöchelchen und der Binnenmuskeln einhergehen, sind nicht imstande, die Hörfähigkeit so stark herabzusetzen, dass die Sprache nicht auf natürlichem Wege erlernt wird oder, wenn sie schon entwickelt war, wieder verloren geht.

Dagegen ist es in hohem Masse wahrscheinlich, dass die *Ausschaltung der Funktion beider Labyrinthfenster* durch pathologische Prozesse bei *Intaktheit* des *inneren* Ohres die Ursache für das Gebrechen abgeben kann. Der erste Fall, der für die Richtigkeit dieser Ansicht spricht, wurde von Politzer 1892 publiziert; es fand sich bei einem Taubstummen, als Ursache seines Gebrechens ausser Stapesankylose durch Verwachsung des Köpfchens mit dem hinteren oberen Rand der Fensternische, knöcherner Verschluss des runden Fensters durch eine das ganze Promontorium bedeckende Knochenmasse.

Der zweite Fall wurde von Habermann im 53. Bd. des *Archivs f.*

[1] Die früher von mir vorgeschlagene Bezeichnung dieser Hauptgruppe als 'im Leben erworbene Taubstummheit' möchte ich auf den von E. Urbantschitsch erhobenen Einwand, dass unter dieser Bezeichnung auch die Veränderungen in dem lebenden Fötus verstanden werden könnten, fallen lassen, und an seine Stelle, den wohl nicht misszuverstehenden Ausdruck *nach der Geburt erworbene oder postfötale Taubstummheit* setzen.

Ohrenheilkunde veröffentlicht; es betraf einen 13 jährigen Taubstummen, bei dem beiderseits die Nische des Schneckenfensters durch Hyperostose der knöchernen Wände bis auf einen bindegewebig ausgefüllten Spalt vollständig verlegt und die Nische des Vorhofsfensters ebenfalls knöchern sehr verengt war; der Stapes und der untere Teil des langen Ambosschenkels war mehrfach mit den Nischenwänden bindegewebig verwachsen. Dabei wiesen *Labyrinth* und *Nervus acusticus normale* Verhältnisse auf und auch am Gehirn liessen sich wenigstens makroskopische Veränderungen nicht erkennen.

Der dritte hier in Betracht kommende Fall, bei dem einseitig ganz ähnliche Verhältnisse wie in den Fällen von Politzer und Habermann vorlagen, ist von mir (Denker, Fall II, *Anatomie der Taubstummheit*, 5. Lieferung) beschrieben worden. Hier lag auf der linken Seite ein vollständig knöcherner Verschluss der Nische des Schneckenfensters und Fixation der verdickten Stapesplatte in der knöchern stark verengten Vorhofsfensternische vor. In der Schnecke fanden sich nur Altersveränderungen, und der von dem Erlanger Neurologen Prof. Jamin untersuchte Hirnstamm liess insbesondere im Kerngebiet des Hörnerven keine primären degenerativen Veränderungen erkennen. Dass es sich in diesem Falle um Taubstummheit handelte, bei welcher *einseitig allein die schweren Veränderungen* an den Labyrinthfenstern das Gebrechen bedingten, das wird umso wahrscheinlicher, wenn man in Betracht zieht, dass bisher bei keinem Taubstummenschläfenbein, welches mit den Hülfsmitteln der modernen Technik untersucht wurde, Veränderungen am Nervenstamm, im Labyrinth oder an den Labyrinthfenstern vermisst wurden.

Immerhin scheinen diese drei angeführten Fälle nach den bisherigen Untersuchungen seltene Ausnahmefälle darzustellen und man darf annehmen, dass in der Regel Veränderungen am Nervus cochlearis oder an seiner Endausbreitung im Labyrinth als die Voraussetzung für die Entstehung der Taubstummheit anzusehen sind.

Von den beiden Hauptgruppen der *angeborenen* Taubstummheit wollen wir zunächst diejenige einer Betrachtung unterziehen, welche die Fälle umfasst, bei denen die Veränderungen bedingt sind durch *intrauterine entzündliche Prozesse* (*Meningo-Encephalitis*, durch Placentarinfektion erworbene *Syphilis*). In Uebereinstimmung mit *Siebenmann* möchte ich annehmen, dass es uns auf Grund der histologischen Untersuchung möglich sein wird, die Fälle abzugrenzen, welche ihre Entstehung einer *intrauterin, aber relativ spät überstandenen Meningitis* verdanken; dagegen wird es nach meiner Ansicht kaum möglich sein, die im *früheren Embryonalleben auf entzündlicher Basis entstandenen Entwickelungsanomalien* von den durch andere Ursachen (fehlerhafte Keimesanlage) bedingten Hemmungsbildungen zu differenzieren.

Die Entstehung der durch eine intrauterine Meningitis bedingten Labyrinthitis werden wir uns in ähnlicher Weise zu denken haben, wie bei den postfötal erworbenen Fällen meningitischen Ursprungs; in

beiden Fällen dürfte es sich um eine Propagation des sich im Subarachnoidalraum abspielenden Prozesses zu den Labyrinthräumen handeln, und zwar wohl meistens auf den präformierten Wegen des Aquaeductus cochleae und der perineuralen und perivasculären Scheiden der durch den Meat. aud. int. in das Labyrinth eintretenden Nerven und Gefässe.

Pathologisch-anatomisch charakterisieren sich die Veränderungen bei dieser Form in ähnlicher Weise wie bei der nach der Geburt durch Meningitis erworbenen Labyrinthitis, nur treten sie meistens *nicht in so destruktiver* Weise auf wie bei der letzteren. Wir finden eine mehr oder weniger ausgedehnte *Degeneration* des *Schneckenepithels*, die oftmals kombiniert ist mit *Ektasie* und *Kollapszuständen* der *häutigen* Labyrinthwand in der Schnecke. Die *Aplasie* und *Pigmentierung* im Ductus cochlearis ist im ganzen nicht so stark ausgeprägt wie in manchen Fällen der II. Hauptgruppe. Bisweilen traten eigentümliche beiderseits identische *Mittelohrveränderungen* auf: *Verengerung* resp. *Verschluss der Fensternischen, Verdickung der Stapesplatte, Verlötung* des langen *Ambosschenkels* und des hinteren *Stapesschenkels* mit dem an der betreffenden Stelle nur bindegewebig verschlossenen Canalis Fallopiae, und der vorgetriebenen *hinteren Paukenhöhlenwand*; dabei kann der Musc. stapedius fehlen.

Bei der *funktionellen* Prüfung dieser Fälle finden sich nicht selten *bedeutende Hörreste.*

I. Hauptgruppe

Zu dieser durch intrauterine entzündliche Prozesse bedingten *I. Hauptgruppe* der angeborenen Taubstummheit, die im ganzen und grossen dem *Typus I* (Siebenmann) in der von Siebenmann angegebenen Einteilung entspricht, sind zu rechnen:

1. Der von Siebenmann in der *Anatomie der Taubstummheit*, I. Lief., beschriebene Fall.
2. Nager, *Anatomie der Taubstummheit*, III. Lief.
3. Habermann, *Archiv f. Ohrenheilk.*, Bd. lxiii, S. 201.

In diesem Falle finden sich Verwachsungen resp. Adhäsionen zwischen Hammerkopf und oberer Wand und zwischen Steigbügel und ovalem Fenster, Hyperostose des hinteren Teiles des Promontoriums; rechts ausserdem umschriebene Ankylose am hinteren unteren Rand der Stapesplatte, Atrophie der Nerven des Sacculus und der Schnecke, sowie der peripheren Ganglienzellen im Spinalkanal, Ektasie resp. Collaps des Ductus cochlearis; Formveränderungen der Stria vascularis und Hypoplasie des Cortischen Organs.

Mit Wahrscheinlichkeit sind unter diese Gruppe auch die folgenden Fälle zu rechnen:

4. Panse, *Archiv für Ohrenheilk.*, Bd. lxiv, S. 118.

Beiderseits knöcherne *Ankylose* des hinteren Stapesschenkels mit der Nische des Vorhofsfensters. *Membrana Reissneri* verbreitert, zum Teil auf die Basilarmembran herabgesunken und mit ihr verwachsen. *Stria*

wenig verändert. *Cortische Membran* missgestaltet, in den Sulcus spiralis internus verlagert und mit Epithel überzogen. *Papilla basilaris* überall degeneriert. Im *Spiralkanal* nur *wenig Ganglienzellen. Nerv. acust.* teilweise degeneriert. Endarteriitis der Art. cochlearis. Macula rec. utriculi rechts kolloid degeneriert, links normal. Macula sacculi rechts degeneriert, links fehlend.

5. Denker (Fall II, *Anatomie der Taubstummheit*, 5 Lief.).

Ob dieser Fall unter die angeborene Taubstummheit zu rechnen ist, wird von Goerke (*Internationales Centralblatt f. Ohrenheilk.*, Bd. viii, S. 391) angezweifelt. Es fanden sich in diesem Falle an dem *rechten* Ohr atrophisch-degenerative Veränderungen am Nerven-Ganglienapparat, Defekt resp. Verlötung der Membrana Reissneri mit der Membrana basilaris, Fehlen der Membrana Corti, der Papilla basilaris und zum grossen Teil auch der Lig. spirale. Auf der *linken* Seite fanden sich nur in der unteren Hälfte der Basalwindung degenerativ-atrophische Veränderungen am Corti schen Organ, während die weiter nach der Schneckenspitze gelegenen Partien normale Verhältnisse aufwiesen, ein Befund, wie wir ihn im Gehörorgan älterer Individuen anzutreffen pflegen. Da solche Bilder uns auch bei der chronischen progressiven Schwerhörigheit entgegentreten, ist es nach Goerkes Ansicht wahrscheinlich, dass auch auf dem rechten Ohr der gleiche Prozess sich abgespielt hat, nur dass er dort bereits viel weiter gediehen sei. Die Taubheit in diesem Falle sei zu erklären durch die Veränderungen an den Fenstern (knöcherner bezw. bindegewebiger Verschluss der Schneckenfensternische, Verengerung der Vorhofsfensternische, Verdickung der Stapesplatte u.s.w.).

Der Auffassung Goerkes, dass es sich hier um eine postfötal erworbene Taubstummheit handle, kann ich nicht beistimmen, einesteils, weil keineswegs feststeht, ob die bei der chronischen progressiven Schwerhörigkeit festgestellten Veränderungen in einem vollkommen normal angelegten Gehörorgan auftreten können, oder ob sie, wie Hammerschlag vermutet, als Spätformen einer kongenital angelegten Alteration des Gehörorgans zu betrachten sind. Ferner aber ist es mir bei dem Fehlen von Residuen entzündlicher Vorgänge, wie wir sie nach Ablauf einer nach der Geburt entstandenen Labyrinthitis in Gestalt von Bindegewebs- und Knochenneubildungen und von perilymphatischen Strangbildungen doch meistens auftreten sehen, doch mindestens höchst wahrscheinlich, dass es sich in diesem Falle um angeborene Taubstummheit gehandelt hat, zumal so schwere Veränderungen im Labyrinth wie auf der rechten Seite bei erworbener Taubstummheit *ohne* entzündliche Ursache wohl kaum vorkommen dürften. Auch das Fehlen von Veränderungen im Nervenstamm spricht eher für angeborene als für postfötal erworbene Taubstummheit.

6. G. Alexander (*Anatomie der Taubstummheit*, 2. Lief.).

Auch in diesem Falle liessen sich stärkere Veränderungen im Mittelohr nachweisen: *Stapesankylose* beiderseits, Einengung der *Nische des Schneckenfensters*. In der Paukenhöhlenwand der Labyrinthkapsel in

der Umgebung des Stapes *Knochenherde vom Typus des bei Otosklerose* gefundenen pathologischen Knochens. Hochgradige *Atrophie des Nerv. acustico-facialis* und seiner Ganglien, Fehlen des Ramus saccularis. Epithel der Macula utriculi niedriger, Canalis utriculi-saccularis, vestibularer Teil des Ductus endolymphaticus, Saccus endolymphaticus beiderseits fehlend. Sacculus rechts rudimentär, Nervenendstellen beiderseits fehlend. *Ductus cochlearis* in den Mittelwindungen durch *Collaps* der *Membrana Reissneri* und stellenweise Verlötung derselben mit der Basilarmembran verkleinert und zum Teil in zwei Räume geteilt. Alle *Nervenendstellen* vollkommen *atrophisch* und degeneriert. *Stria vascularis* fehlend oder an ihrer Stelle abnorme Zellenwucherungen. *Membrana Corti* verlagert, ohne Verbindung mit dem Cortischen Organ. Auf dem Epithel der Membrana Reissneri homogene Schleimkugel.

7. Alexander und Neumann (Fall I, *Anatomie der Taubstummheit*, 6. Lief.).

Neben den degenerativ-atrophischen Veränderungen im Nervenendapparat und den Ganglien, Ektasien resp. Verengerung oder Aufhebung des Lumens der Hohlräume etc. fanden sich hier *rudimentäre Entwickelung des äusseren Gehörganges, des Stapes, Fehlen von Hammer und Ambos und Trommelfell. Auffüllung der Paukenhöhle* und des Antrums durch *pathologischen, reich vascularisierten Knochen.* In den perilymphatischen Räumen pathologische Knochenbalken, Bindegewebs- und Knochenneubildung an den Skalenenden der Schneckenbasis mit Verschluss der Scala tympani gegen den Vorhof. Bindegewebsstränge und Knochenbalken in den Scalen. Die Veränderungen im Mittelohr deuten darauf hin, dass die Störungen schon in eine frühe Entwickelungsperiode vor der Anlage des fehlenden Tympanicum, des Hammers und des Ambosses zu legen sind.

8. R. Panse (*Anatomie der Taubstummheit*, 8. Lieferung).

Nach der Anamnese will der Patient von Geburt an taub gewesen sein. Die histologische Untersuchung ergab deutliche Residuen einer entzündlichen Affektion des inneren Ohres. *Rechts Mittelohr* normal, *links* Veränderungen durch tuberkulöse Mittelohrentzündung ohne Einbruch ins innere Ohr. *Links* fehlen Macula sacculi und rec. utriculi. Sacculuswände miteinander verklebt. Im Labyrinth degenerative Vorgänge, wobei zu erwähnen ist, dass die atrophischen Veränderungen im cochlearen Teil wesentlich stärker ausgeprägt waren als im vestibularen. *Rechts* Veränderungen noch stärker. *Sacculus, Utriculus* und *Cochlea* fehlend. An Stelle der Schnecke alter Knochen ohne interglobulären Knorpel. Verf. ist der Ansicht, dass es sich um *Residuen* einer Labyrinthitis handelt, die auf eine intrauterine Meningitis zurückzuführen sind.

9. Walker Downie (*Zeitschrift f. Ohrenheilk.*, Bd. xxx, S. 236).

In diesem Falle handelt es sich *ebenso wie in beiden folgenden Fällen* um eine durch *hereditäre Syphilis* erworbene Taubheit. *Stapesplatte* mit dem Rande des Vorhofsfensters innig verbunden oder knöchern mit ihm

verwachsen, so dass sie *unbeweglich* an der medialen Paukenhöhlenwand fest anhaftet. Der Warzenteil bestand aus kompaktem Knochen. Verengerung des inneren Gehörgangs fast bis zur Aufhebung des Lumens. Vestibulum stark verengt. *Modiolus* und *Lamina spiralis ossea* stark verdickt. Von den Bogengängen war *nur eine Spur des äusseren* Kanals auffindbar. Die Sklerosierung des Proc. mastoid., die Obliteration des äusseren Drittels des Meat. audit. int. und eines grossen Teils des Labyrinths waren die Folgen eines chronisch-entzündlichen Prozesses von unzweifelhaft syphilitischem Charakter.

10. Gradenigo (*Archiv f. Ohrenheilk.*, Bd. xxv, S. 46 und 237).

Beiderseitige *vollständige Zerstörung des häutigen Labyrinths* und der Elemente der beiden Labyrinthfenster, *Neubildung von Faser- und Knochengewebe*: Das neugebildete Knochengewebe war teils aus dem Endost des Labyrinthraums, teils aus direkter Umwandlung des neugebildeten Bindegewebes entstanden. *Bogengänge* vollständig *fehlend, Vorhof* bedeutend *verengt*, in der Schnecke nahm das neugebildete Knochengewebe umsomehr an Masse ab, je mehr man sich der Spitze näherte. Auf Grund der Anamnese und nach dem Vorhandensein der Hutchinson'schen Trias handelt es sic hnach Ansicht Gradenigos zweifellos in diesem Falle um durch hereditäre Syphilis hervorgerufene Taubstummheit.

11. Moos und Steinbrügge (*Zeitschrift f. Ohrenheilk.*, Bd. xv, S. 87, Fall I).

Hyperostotische Verengerung des äusseren Gehörganges, Verkleinerung der Paukenhöhle und des Antrums, *knöcherne Obliteration der Zellen des Warzenfortsatzes*, dachförmige *Ueberragung der Nische des Vorhofsfensters*, *Dickenzunahme* des *Promontoriums*. Bindegewebige Adhäsionen in der linken Scala tympani. In den erweiterten Markräumen fanden sich rote Blutkörperchen, blutkörperchenhaltige Zellen und Pigment, in der Umgebung des Nerv. facialis Blutextravasate. *Rechts Collaps* der *Membrana Reissneri*, Cortisches Organ stellenweise erhalten, an einigen Schnitten nur die inneren Pfeiler, daneben unregelmässige zellige Massen. *Links* im Anfangsteil der Scala tympani bindegewebige Massen und kolloidartige Körper. Ob es sich in diesem Falle um die Erscheinungen einer *hereditären* oder *erworbenen Syphilis* gehandelt hat, wagen die Verfasser bei der mangelhaften Anamnese nicht zu entscheiden.

II. Hauptgruppe

Wir kommen nun zu der Besprechung derjenigen Fälle von *angeborener Taubstummheit, bei welchen die im Labyrinth konstatierten Bildungsanomalien* auf *nicht entzündlicher Basis entstanden sind*, und zwar haben wir zunächst die Unterabteilung zu betrachten, welche die Fälle mit *Aplasie des ganzen Labyrinths* umfasst.

Gruppe 1. — Von dieser Gruppe ist bisher nur ein *einziger* obduzierter Fall (Michel, *Gazette Médicale de Strassbourg*, 1863, p. 55) bekannt; es handelt sich um angeborenen doppelseitigen *Defekt des ganzen Labyrinths*.

Bei normalem Gehörgang und Trommelfell war die Paukenhöhle verengt, das Antrum nur angedeutet. Chorda tympani und Proc. mast. fehlen, ebenso das ganze Labyrinth samt der entsprechenden Knochenmasse und dem Nerv. acusticus. Von der Paukenhöhle aus gesehen erscheint die Labyrinthwand *transparent*, da direkt hinter ihr die Schädelhöhle liegt. Siebenmann erklärt das Zustandekommen der Aplasie aus dem Ausbleiben der unter normalen Verhältnissen schon im ersten Embryonalmonat erfolgenden Ektodermeinstülpung, die später zur Bildung des Labyrinths führt. Die normale Entwickelung von Gehörgang und Trommelfell biete nichts Auffallendes, da es erst später zur Bildung des Deckknochen des Annulus tympanicus und der Schuppe komme; dagegen sei es höchst merkwürdig, dass die Gehörknöchelchen, welche schon am Ende der 4. Woche als dichtere Blastemmasse sich am dorsalen Ende des Meckel'schen und Reichert'schen Vorknorpels differenzieren und nahe an das Labyrinthbläschen sich anlegen, durch dessen Fehlen in ihrer Entwickelung nicht gehemmt worden sind.

Gruppe 2. — Bei dieser Gruppe ist, wie oben erwähnt, das ganze *knöcherne* und *häutige* Labyrinth vorhanden, dagegen das Epithel *degeneriert*; je, nach dem Grad und der Ausdehnung der Epitheldegeneration kann man 2 Unterabteilungen unterscheiden:

a. Fälle, bei denen die *Epithelmetaplasie beschränkt* ist auf die *Membrana basilaris*.

Hierher gehören:

1. Der von L. Katz im *Archiv f. Ohrenheilk.*, Bd. xliii, S. 167 veröffentlichte Fall.

Bei normalem, äusserem und mittlerem Ohr fand sich eine Verdünnung der Lamina spiralis ossea und eine auf das Epithel der *Membrana basilaris* und die Nervenelemente der Schnecke beschränkte *Hypoplasie*.

2. In dem von E. Oppikofer (*Zeitschr. f. Ohrenheilk.*, Bd. xliii, S. 177) beschriebenen Fall fand sich Hypoplasie des Ganglion spirale und der von ihm abzweigenden Fasern, mangelhafte Entwickelung und stellenweises Fehlen des Cortischen Organs, Metaplasie der Claudius'schen Zellen und ausserdem Unipolarität eines Teils der Zellen des Spiralganglions, embryonales Verhalten der Membrana tectoria in der Spitzenwindung und der Stria vascularis in allen Windungen.

3. Watsuji (*Anatomie der Taubstummheit*, I. Lief.). Die pathologischen Veränderungen betreffen ausschliesslich die membranöse Schnecke, die Spiralganglien und den Schneckennerven sowohl im Stamm als auch während seines Verlaufs in der Schnecke; der übrige Teil des häutigen Labyrinths, die Schneckenkapsel und das Mittelohr verhalten sich gänzlich normal.

4. Denker (Fall I, *Anatomie der Taubstummheit*, 4. Lief.). Bei normalem Mittelohr und unverändertem Stamm des Nervus acustico-facialis waren die pathologischen Veränderungen lokalisiert in dem Verlauf des Nervus cochlearis innerhalb der Schnecke, in den Spiralganglien und im Ductus cochlearis, Pars superior und Sacculus normal.

5. Friedrich (*Verhandlungen der Vers. Deutscher Naturf. und Aerzte zu Dresden*, 1907, S. 356).

Wenn auch Friedrich selbst die in seinem Fall gefundenen Anomalien als das Produkt einer degenerativen Neuritis auffasst, so scheint mir doch das Fehlen von entzündlichen Bindegewebsneubildungen mehr für eine angeborene Taubstummheit zu sprechen. Auch sind Fälle von degenerativer Neuritis cochlearis in den ersten Lebensjahren — bei einem Taubstummen kann es sich doch nur um diese Zeit der Entstehung des Leidens handeln — meines Wissens noch nicht beobachtet worden.

β. Fälle mit *ausgedehnterer Epithelmetaplasie, fehlender* oder *mangelhafter Entwickelung* des *Sinnesepithels*, kombiniert mit *Ektasie* und *Collapszuständen* der häutigen Labyrinthwand der Pars inferior.

Zu dieser Unterabteilung sind zunächst folgende acht Fälle zu rechnen.

1. Mondini (Mygind, *Taubstummheit*, Fall IV).

2–6. Ibsen-Mackeprang, die Fälle 12–13, 24–25, 28–29 u. 36–37.

7. G. Alexander (*Archiv f. Ohrenheilk.*, Bd. lxi, S. 183).

8. A. Iwanow (*Russische Monatsschrift f. Ohrenheilk.*, März 1907) (Ref. im *Archiv f. Ohrenheilk.*, Bd. lxv, S. 309).

Diese Fälle weisen als *gemeinsame* pathologische Veränderungen eine *Erweiterung des Aquaeductus vestibuli* und der *häutigen Schneckenspitze* auf (Siebenmann hat diese Fälle als Typus *Mondini* zusammengefasst). In dem von Mondini beschriebenen Fall waren die beiden Scalen der Schnecken nur in den unteren $1\frac{1}{2}$ Windungen vorhanden. In der oberen Partie der Schnecke befand sich eine weite gemeinsame Höhle, durch deren Mitte ein *dünnes Säulchen* von dem Modiolus senkrecht zur Decke verläuft; dieses Säulchen ist teils knöchern, teils knorpelig. Die *Schnecke* ist im ganzen von der Spitze bis zur Basis etwas *abgeplattet*. Die Bogengänge sind normal. Der prall gefüllte Saccus endolymphaticus liegt in einer entsprechend grossen Mulde der hinteren Pyramidenfläche. Der Aquaeductus vestibuli bildete eine nach hinten nur membranös geschlossene Rinne.

Ausser den oben erwähnten charakteristischen Merkmalen fanden sich in dem von Alexander untersuchten Falle noch folgende Einzelheiten:

1. *Atrophie des Hörnerven* im Stamm, in seinen Aesten und Ganglien. Das Schneckenganglion besitzt im oberen Abschnitt keine spirale Anordnung; Maculae und Cristae etwas reduziert, Zahl der Sinneszellen etwas verringert. 2. *Herdförmige Atrophie und Degeneration des Epithels* aller Wände des Ductus cochlearis in verschieden hohem Grade. 3. Die unter normalen Verhältnissen *konkave* (*axiale*) Wand der oberen Schneckenwindung ist *aufgebläht* und legt sich über die Stelle, wo im gesunden Ohr die Helicotrema-Lücke liegt. Die Nervenendstellen der ektasierten Spitzenwindung haben embryonalen Charakter. Stellenweise setzt sich die Vestibularmembran der Mittelwindung in die Crista spiralis der Spitze fort und infolge Fehlens des axialen Schneckenabschnittes fliessen

die Cristae beider Seiten der Mittelwindung axial zusammen. 4. Meistenteils findet sich auffallende Armut an Blutgefässen und überall besteht gänzlicher Pigmentmangel des inneren Ohres. Es bestand in diesem Falle *Totaltaubheit.*

Auch in den von Iwanow veröffentlichten Fällen waren *Modiolus* und *Lamina spiralis ossea* nur im Bereich der untersten Windung vorhanden. Scala *vestibuli* der mittleren Windung kommunizieren mit einander, da die dazwischen liegende Wand fehlt. Das Helicotrema fehlt dementsprechend ebenfalls. *Schnecke* im ganzen *abgeplattet.* Der Patient starb an eitriger Meningitis, die von akut entzündlichen Veränderungen in der Schnecke begleitet war ; *Collaps des Ductus cochlearis* und des *Sacculus* links, Bluterguss in der Stria vascularis. Als Ausdruck von *Entwickelungshemmungen* fanden sich ausserdem beiderseits *Atrophie* des *Ganglion spirale,* links *Degeneration* und *Atrophie* des Cortischen Organs und der *Membrana Corti,* rechts *Atrophie des Cortischen Organs,* der *Stria vascularis* und des *Ligamentum spirale.*

Die folgenden sieben ebenfalls zur Unterabteilung β der Gruppe II gehörigen Fälle zeichnen sich zum Teil durch eine *Verdünnung der knöchernen Lamina spiralis,* des *knöchernen Gerüstes der Spindel* und der Zwischenwände der oberen Schneckenhälfte aus. (Diese Fälle — von Siebenmann als Typus *Scheibe* bezeichnet — lassen sich vielleicht als ein Vorstadium der bei den ersten acht Fällen der Unterabteilung gefundenen Veränderungen auffassen.) *Hochgradige Ektasie des Sacculus.* Epitheldegeneration und stärkere Faltenbildung im Ductus cochlearis, die als *Collaps* oder *Ektasie* der Membrana Reissneri zu Tage tritt. Es ist nicht mit Sicherheit auszuschliessen, dass auch diese Fälle als durch eine intrauterin überstandene Meningitis bedingte Labyrinthitis aufzufassen sind. Die beschriebenen Veränderungen liessen sich in folgenden Fällen nachweisen :

9. Scheibe, I. Fall (*Zeitschrift für Ohrenheilk.,* Bd. xxii, S. 11).
10. Scheibe, II. Fall (*Zeitschrift f. Ohrenheilk.,* Bd. xxvii, S. 95).
11. E. Oppikofer (*Zeitschrift f. Ohrenheilk.,* Bd. xliii, S. 192).
12. Siebenmann (*Verhandlungen der Deutschen otol. Ges.,* 1904).
13. L. Katz, II. Fall (*Zeitschrift f. Ohrenheilk.,* Bd. xli, S. 89).
14. Schwabach (*Zeitschrift f. Ohrenheilk.,* Bd. xlviii, S. 293). In diesem Falle fanden sich keinerlei Residuen voraufgegangener entzündlicher Prozesse. *Cortisches* Organ *rechts* in der Basalwindung nur rudimentär vorhanden, in den übrigen Windungen und ebenfalls links fehlend. *Rechts Atrophie* des Ganglion spirale und der Nervenfasern von der Basis nach der Schneckenspitze zu an Intensität abnehmend, links die Atrophie nur in der Basalwindung zu erkennen. *Membrana Reissneri rechts* ausgebuchtet, links vollständig kollabiert, spaltförmige Verengerung des Ductus cochlearis. Vorhof, halbzirkelförmige Kanäle, Vestibularnerv beiderseits normal.
15. Lindt (*Deutsches Archiv für klin. Medizin,* Bd. lxxxvi). Bei annähernd normalen Verhältnissen in der Pars superior fand sich *Collaps*

des Sacculus und der *Membrana Reissneri*, Degeneration des Neuroepithels in der Cochlea und im Sacculus.

Mit diesen sieben Fällen haben zwei von Goerke (*Anatomie der Taubstummheit*, 3. Lief.) publizierte Fälle grosse Aehnlichkeit; sie weisen folgende Einzelheiten auf:

16. In dem einen Falle war das Lumen des Ductus cochlearis durch Strangbildungen eingeengt, seine äussere Wand in ihrer zelligen Struktur stark verändert; teils Fehlen, teils starke Verbreiterung der Stria vascularis, das Lumen des Ductus cochlearis zur Hälfte ausfüllend. Von spezifischen Zellenelementen ist auf der Membrana basilaris nichts zu erkennen. Teilweise Verlötung der *Membrana Reissneri* mit der *Membrana basilaris*. Zahlreiche kolloide Kugeln in der *Stria vascularis* bei Atrophie der Blutgefässe.

17. Auch in dem II. Fall war die *Stria vascularis* stark verändert, das Vas prominens überall fehlend; Cortische Membran nicht zur völligen Entwickelung gelangt. An Stelle der Papilla basilaris ziemlich flaches, strichförmiges Epithel. *Macula sacculi* rechts fehlend, links wenig entwickelt. Die *Otolithenmembran* hat ihre Jugendform bewahrt. *Collaps* der *äusseren Sacculuswand*. N. acusticus und seine Ganglien relativ gut ausgebildet. Das *embryonale* Verhalten der *Stria vascularis*, des *Epithels*, der *Membrana basilaris*, der *Macula sacculi* und der *Otolithenmembran* sprechen dafür, dass die Veränderungen als Entwickelungshemmungen aufzufassen sind.

18. Alt (*Monatsschrift f. Ohrenheilk.*, 1908, No. 1). In diesem Falle war das *Mittelohr* im Ganzen normal, das *Labyrinth* in typischer Weise angelegt. Die *Nervenendstellen* waren im ganzen Labyrinth nur unvollkommen zur Ausbildung gelangt und hatten nachträglich eine weitgehende *Atrophie* durchgemacht, die im vestibulären Abschnitt den grössten Teil, in der *Cochlea* das *ganze Sinnesepithel* betrifft und hier sogar zum Collaps des Ductus cochlearis geführt hatte. Die Schneckenachse war *rechts* mässig *atrophiert*, *links* stark *reduziert*; sie war in der *II. Windung* nicht knöchern sondern *bindegewebig* und *fehlte in der III. Windung* gänzlich. Reichliche Ausbildung von Hyalinkörpern und pigmentführenden Schollen im ganzen Bereich des Ductus cochlearis. *Cupulae* rechts *fehlend*, links atrophisch. Residuen vorausgegangener entzündlicher Prozesse im Labyrinth nirgends zu erkennen.

19. Schwabach (*Anatomie der Taubstummheit*, 4. Lief.). In diesem Falle fehlen Residuen von voraufgegangenen entzündlichen Prozessen am Nerven und in der Schnecke gänzlich. Im Stamm des Nerv. acust. *Metastasen eines Adenocarcinoms*, dessen primärer Herd sich im Magen befand. Die Veränderungen in der Schnecke beschränken sich auf die Pars inferior. Der *Sacculus* ist *rechts* verengt. Die Macula sacculi beiderseits ohne Neuroepithel. Links ist das Lumen des Sacculus in seiner hinteren Partie mässig verengt. Die *Stria vascularis* weist die Form eines teils kegelförmig zugespitzten, teils halbkugelig geformten Tumors auf. *Membrana Reissneri* stellenweise *ektasiert*, an anderen

Stellen herabgesunken, das Lumen des Ductus cochlearis einengend. *Membrana tectoria* überall von normaler Faserung weicht besonders *links* in Bezug auf Gestalt und Lageverhältnis von der Norm ab.

Das Aussehen der *Papilla basilaris* entspricht im grossen und ganzen dem von Böttcher und Kölliker bei Raubtierembryonen beschriebenen ‘kleinen Epithelialwulst’. Haarzellen fehlen am Cortischen Organ gänzlich. Nur im Vorhofsteil und in der unteren Partie der Mittelwindung Andeutung des Cortischen Tunnel. In der im übrigen normalen *Scala vestibuli* findet sich im Vorhofsabschnitt ein dreieckiges Gebilde, das in das Ligament. spirale übergeht, dem es auch seinem Aussehen und seiner Struktur nach gleicht. *Scala tympani* beiderseits unverändert. Neben dem *Canalis Fallopiae* abnorme Muskelbildung.

20. Quix und Brouwer (*Anatomie der Taubstummheit*, 7. Lief.). Mittelohr, Labyrinthkapsel und Pars superior des häutigen Labyrinths ohne Veränderungen. *Sacculus* und *Macula sacculi* normal, Ramus saccularis mässig verdünnt. *Vollständiges Fehlen* des *Ganglion spirale* sowie der peripheren und zentralen Nervenfasern. Ductus cochlearis verengt, seine *epithelialen Gebilde vollständig degeneriert*, die *Stria vascularis*, die *Membrana Corti*, das *Cortische Organ* fehlen oder zeigen Metaplasie. Die von Brouwer vorgenommene Untersuchung des *zentralen Verlaufs des Nerv. acusticus* ergeben beiderseitige Zerstörung der in die Medulla oblongata einstrahlenden Cochlearisfasern, des Tuberculum acusticum und der Zellen aus den zentralen Acusticuskernen. Degeneration in der Formatio reticularis, im Corpus trapezoideum, im Gebiet der in der Raphe kreuzenden Fasern, in der lateralen Schleife und im Corpus quadrigeminum posticum.

Quix ist im Gegensatz zu Brouwer der Meinung, dass die intercerebralen Abweichungen der Hörbahn sekundär sind, und zwar entstanden teilweise durch aufsteigende Degeneration des peripheren Neurons, teilweise durch sekundäre Inaktivitätsatrophie.

Goerke ist der Ansicht, dass der vorliegende Fall nicht als angeborene resp. embryonale Taubstummheit anzusehen ist, weil die beschriebenen pathologischen Veränderungen auch bei postfötaler Taubstummheit und bei chronischer progressiver Schwerhörigkeit beobachtet worden sind. Demgegenüber ist einzuwenden, dass es sich bei der *progressiven labyrinthären* Schwerhörigkeit auch um Spätformen einer *kongenital* angelegten Schwerhörigkeit handeln kann. Da ferner *nirgends Residuen* einer überstandenen Entzündung gefunden wurden, möchte ich mit dem Verfasser den Fall als *angeborene* Taubstummheit ansehen.

21. Schönemann (*Anatomie der Taubstummheit*, 7. Lief.). Dieser Fall hat Aehnlichkeit mit der weiter oben (Fälle 1–8) beschriebenen Form kongenitaler Taubstummheit, welche sich durch eine *Erweiterung der häutigen Schneckenspitze* und des *Aquaeductus vestibuli* auszeichnet. Auch hier erwies sich am *linken* Ohr die knöcherne *Grundlage der mittleren und der Spitzenwindung* als *völlig defekt*, während das knöcherne Stützgerüst der Basalwindung ziemlich erhalten war. Ausser den bekannten atro-

phisch-degenerativen Veränderungen im Ductus cochlearis fand sich links ein von der *Lamina spiralis ossea ausgehendes, in das Lumen des knöchernen Schneckenkanals völlig frei hineinragendes Osteom.* Entgegen der Ansicht Goerkes, der das intrascalär gelegene Osteom als eine auf entzündlicher Basis entstandene Knochenneubildung auffasst, und es für fraglich hält, dass es sich um angeborene Taubstummheit handelt, scheint mir der Fall doch deswegen zur kongenitalen Taubstummheit zu gehören, weil der festgestellte *Defekt des Modiolus* in der II. und der Spitzenwindung bei postfötal erworbenem Gebrechen *noch niemals* beobachtet worden ist.

Wir haben nun noch die Fälle zu betrachten, *bei welchen die Taubstummheit bedingt war durch ererbte kretinische Degeneration*, oder wo das Gebrechen vergesellschaftet war mit sonstigen *hereditär degenerativen Symptomen.*

Nach Siebenmann liegen die Störungen bei der *kretinischen* (oder *endemischen*) *Taubstummheit* Veränderungen des *inneren Ohres* oder Schädigungen des Zentralnervensystems zu Grunde ; die Taubheit lässt sich keineswegs — wie Wagner und Jauregg meinten — durch eine Fortsetzung der myxoedematösen Schwellung der Nasen- und Rachenschleimhaut auf das Mittelohr erklären, da die exakte funktionelle Prüfung die Symptome einer *Erkrankung des inneren* Ohres ergibt. Die Taubheit der Kretinen ist nach Siebenmann aufzufassen als Teilerscheinung der schweren funktionellen Störung, welche das ganze Nervensystem erleidet bei Ausfall oder qualitativer Aenderung der Schilddrüsenfunktion. Aus dem Umstand, dass das Gehör durch die Schilddrüsentherapie *nicht* beeinflusst wird, zieht Siebenmann den Schluss, dass schwere anatomische Defekte im inneren Ohr hier die Regel bilden. Beweiskräftige Sektionsergebnisse für die Richtigkeit dieser Annahme lagen bis zum Jahre 1904 noch nicht vor, wenn auch in zwei von Siebenmann und von Habermann berichteten Fällen Entwickelungsstörungen in der Schnecke gefunden wurden.

22 und 23. G. Alexander (*Archiv f. Ohrenheilk.*, Bd. lxxviii, S. 54). Eine wertvolle Erweiterung unserer Kenntnisse von den Veränderungen bei kretinischer Taubheit bildet die von G. Alexander veröffentlichte Arbeit über ' das Gehörorgan der Kretinen '. Dieser Publikation liegen die Untersuchungsergebnisse von *zwei Fällen kretinischer Taubstummheit*, einem Fall von hochgradiger Schwerhörigkeit bei einem kretinösen Kinde und einem Fall von Schwerhörigkeit bei einem kretinösen Hunde zu Grunde. In sämtlichen untersuchten Schläfenbeinen fanden sich myxomatöse Verdickung der Mittelohrschleimhaut, Verödung der Nische des Schneckenfensters durch Bindegewebe und Fettgewebe, auffallend geringe Beteiligung des Nervenganglienapparates des Acusticus, ausserdem ausgedehnte degenerative Veränderung des peripheren Sinnesorgans und hochgradige Substanzarmut des perilymphatischen Bindegewebes.

Das Gehirn erwies sich frei von krankhaften Veränderungen, so dass die Hörstörungen ausschliesslich auf Rechnung des *peripheren Sinnes-*

organs zu setzen sind. Von Bedeutung war der Befund von pathologischen Knochenherden in der Labyrinthkapsel, wie sie in ähnlicher Weise bei *Otosklerose*, ausserdem aber auch bei folgenden Fällen angeborener Taubstummheit gefunden wurden: Habermann (*Archiv f. O.*, 1904, Bd. lxiii), Panse (*Archiv f. klin. Med:*, Bd. lxxxvi, S. 140), Denker, Fall II (*Anatomie der Taubstummheit*, 5. Lief.), Schwabach (*Zeitschrift f. O.*, Bd. xlviii, S. 295). Diese Befunde machen es in hohem Masse wahrscheinlich, dass auch die Knochenveränderungen bei Otosklerose kongenitaler (wahrscheinlich entzündlicher) Natur sind.

Im Gegensatz zu den positiven Untersuchungsergebnissen G. Alexanders sei darauf hingewiesen, dass Siebenmann in einem Falle von totaler Aplasie der Schilddrüse bei einem 13 jährigen Kretin irgendwelche Anomalien im inneren Ohr nicht nachweisen konnte. Es geht daraus hervor, dass die Schilddrüse für die Entwickelung des häutigen Labyrinths während des intrauterinen Lebens entbehrlich ist.

Erwähnt sei auch, dass bei von *mir* untersuchten *thyreodektomierten* Hunden (Denker, 'Schilddrüse und Gehörorgan,' *Verhandl. d. Deutschen otol. Ges.*, 1909) das Fehlen der Reaktion auf Schalleindrücke *weder bedingt war* durch *myxoedematöse Veränderungen* in der *Mittelohrauskleidung* noch durch *degenerative Vorgänge am Nervenstamm und in der Schnecke. Auch im Wurzel- und Kerngebiet des Nerv. acusticus und in seinem weiteren Verlauf bis zur Vierhügelgegend* (Professor Jamin) liessen sich *keine Anhaltspunkte* finden, die als Erklärung für die scheinbare Taubheit hätten dienen können. Es ist höchstwahrscheinlich, dass das Ausbleiben der Reaktion auf acustische Reize bedingt ist durch *Störungen im Zentralnervensystem*, die als *Folge* der im Anschluss an die Schilddrüsenexstirpation auftretenden *Stoffwechselveränderungen* aufzufassen sein dürften.

In den Fällen, wo bei den Kretinen die Schwerhörigkeit ausschliesslich als eine Folge der myxomatösen Verdickung der Paukenhöhlenschleimhaut aufzufassen ist, kann nach Ansicht Alexanders durch Thyreoidinbehandlung vollständige Heilung erfolgen; er glaubt, dass auch bei schwerhörigen Kretinen mit Symptomen pathologischer Veränderungen im schallperzipierenden Apparat noch Besserung erzielt werden kann, während naturgemäss nach eingetretener Taubheit Thyreoidintherapie keinerlei Erfolg mehr haben kann.

24–26. Manasse (*Zeitschrift für Ohrenheilk.*, Bd. lviii, S. 105). Von den *drei Individuen*, deren Schläfenbeine Manasse untersuchte, machte der erste direkt einen kretinischen Eindruck und hatte zwei taubstumme Brüder; der 2. Fall betraf einen 24 jährigen von Geburt an taubstummen Mann, der zwar nicht kretin, aber schwachsinnig war; in dem 3. Falle handelte es sich um einen 68 jährigen mit Struma behafteten Mann, der einen geistesschwachen Bruder hatte, im übrigen aber einer gesunden Familie angehörte. Die beiden ersten Fälle stellen zweifellos eine exquisit familiäre Erkrankung degenerativer Natur dar, die ihren Ausdruck in der Strumabildung, der geistigen Minderwertigkeit und der Taubstumm-

heit fand. Ob auch der 3. Fall als kretinische Taubstummheit anzusehen ist, wagt Manassè nicht zu entscheiden. Neben den auch sonst bei Taubstummheit beobachteten degenerativ-atrophischen Veränderungen im häutigen Labyrinth, konnte Verfasser in allen drei Fällen *erhebliche, wenn auch nicht völlig gleichartige, so doch sehr ähnliche Veränderungen am knöchernen Ohrskelett feststellen, die wenigstens zum grossen Teil mit erheblicher Wahrscheinlichkeit als kongenital anzusprechen sind*; die Alteration des häutigen Labyrinths dagegen sieht Manasse wenigstens zum Teil als erworben an. — Für den *embryonalen* Charakter der knöchernen Veränderungen in der Labyrinthkapsel, die durch *Knochenneubildung zu Einengung der Paukenhöhle, Verlegung der Labyrinthfenster und zu einer Verzerrung der Labyrinthhohlräume geführt hatte*, spricht nach Manasses Meinung vor allem der Umstand, dass eine *Grenze zwischen normalem und pathologischem Knochen nirgends* zu finden war, wie dies bei Knochenneubildungen *entzündlicher* Art der Fall zu sein pflegt.

Zu der mit hereditär-degenerativen Symptomen vergesellschafteten Form der angeborenen Taubstummheit sind vor allem die Fälle zu rechnen, welche mit *Atresia auris congenita* einhergehen und ferner die Fälle, bei denen sich *Pigmentanomalien* finden. Von ersteren sind nach Siebenmann 10 doppelseitige und 3 einseitige Fälle klinisch beobachtet und publiziert. Von *Sektionsbefunden* bei Taubstummen mit Atresia auris congenita liegt nur ein einziger von

27. Ogston veröffentlichter Fall von einem 1½ Jahr alten Kinde vor; das Schläfenbein der atresierten Seite wies makroskopisch erkennbare Entwickelungsanomalien auf: Atrophie des Nerv. acusticus, Hemiatrophie von Gross- und Kleinhirn, Hypoplasie der Pyramide, Fehlen des Nerv. oculomotorius, Vagus, Accessorius, ferner des Foramen stylomastoideum und des Proc. mastoideus.

Von *Pigmentanomalien* sind es hauptsächlich die Fälle von Taubstummheit mit *Retinitis pigmentosa*, welche unser Interesse beanspruchen. Während eine grössere Reihe von klinischen Beobachtungen bei Fällen dieser Art vorliegt, verfügen wir nur über *einen* Sektionsbericht; derselbe stammt von

28. Siebenmann und Bing (*Zeitschrift f. Ohrenheilk.*, Bd. liv, S. 265), und zeigte folgenden Befund: Ramus cochlearis und Ganglienzellen im Rosenthal'schen Kanal stark atrophisch. Lumen des Ductus cochlearis unverändert. *Cortisches Organ* im *untersten* Teil der Schnecke *fehlend*, am besten im *mittleren Teil der mittleren Windung entwickelt*, es sitzt aber abnormerweise der Crista das Ligament. spirale bedeutend *näher* als der Crista spiralis. *Stria vascularis* hypoplastisch und gegen die *Membrana Reissneri* heraufgerückt. Auffallend *spärliche Vascularisation* im ganzen intralabyrinthären Kapillargebiet. Nervenendstellen der Ampullen und des Vorhofs streckenweise degeneriert; Ramus *vestibularis* und seine *Ganglien* anscheinend *normal*. Pigmentation etwas spärlich.

Während sich bei den meisten Fällen von *angeborener* Taubstummheit

die Veränderungen auf die Pars inferior beschränken, findet sich hier auch *Degeneration* der *Maculae und Cristae.* — Die Untersuchung des zentralen Verlaufs des Nerv. acusticus ergab in Bezug auf die Vestibularbahn normale Verhältnisse, dagegen schwere *Alteration* der *Cochleariskerne* und *Atrophie* der *Cochlearis*fasern, Veränderungen, die als Inaktivitätsatrophie aufzufassen sein dürften. Von besonderem Interesse ist, wie Siebenmann hervorhebt, dass das Cortische Organ in der mittleren Windung relativ am besten entwickelt war im Zusammenhalt mit der Beobachtung Bezolds, dass sich bei drei von 4 Gehörorganen, deren taubstumme Besitzer mit Retinitis pigmentosa behaftet waren, eine Hörinsel in dem mittleren Teil der Skala fand. Sollte es sich durch weitere Untersuchungen bestätigen, dass die mit Retinitis pigmentosa behafteten Taubstummen Hörinseln im mittleren Teil der Skala und bei der Obduktion eine relativ gute Entwickelung des Cortischen Organs der Mittelwindung aufweisen, so würden derartige Befunde als Stütze der *Helmholtz'schen* Resonanztheorie zu verwerten sein.

Zum Schluss mögen hier noch *drei Fälle* Erwähnung finden, bei denen es zweifelhaft ist, ob die *Taubstummheit angeboren* oder *nach der Geburt erworben ist*:

1. Politzer (*Anatomie der Taubstummheit*, 1. Lief.). In diesem Falle war die *Vorhofsfensternische* ausgefüllt mit einer neugebildeten *Knochenmasse*,die vom Promontorium ausging und Aehnlichkeit mit den Knochenveränderungen bei Otosklerose aufwies. Auch die *Schneckenfensternische* war stark verengt durch eine vom Endost des Promontoriums ausgehende Knochenwucherung. In der Schnecke fiel am Boden der Scala tympani eine *faltenförmige Erhebung* des *Endosts* auf, dessen Durchschnitt verschieden gestaltet, bald kolbig, bald pilzartig, den papillären Zotten nicht unähnlich erscheint. *Fehlen*, resp. Verminderung der *Ganglienzellen* im Rosenthal'schen Kanal. Die *Papilla basilaris* weist überall die typische Gestalt auf, jedoch ist ihre *Zellstruktur* nur in der Spitzenwindung ausreichend: *Hier fehlen die Haarzellen.* — Die Veränderungen in der Labyrinthkapsel und im Endost sind nach Politzers Ansicht zweifellos als Produkte einer Entzündung anzusehen, ob sie jedoch im *Fötalleben* oder *nach der Geburt entstanden* sind, wagt er *nicht* zu entscheiden.

2. R. Panse (*Archiv f. Ohrenheilk.*, Bd. lxiv, S. 130). Mittelohr und Pars superior beiderseits normal. *Rechts Schneckenkapsel* normal. Membrana Reissneri fehlt in der Mittelwindung, in der Basal- und Spitzenwindung aufgefasert. *Membrana Corti* teils fehlend, teils in Kugel- oder Pinselform vorhanden, mit Epithel überzogen. Die sehr gefässarme *Stria vascularis* und *Cortisches Organ* nur in der Mitte und oben erhalten, letzteres als Hügel von Colloidklumpen und atypischen Zellen. Sehr spärliche Spiralganglienzellen und zerfallene Nervenfasern. *Links Membrana Reissneri* teils fehlend, teils basal angelötet. *Membrana Corti* missgestaltet, aber meist vorhanden. *Cortisches* Organ nur ein undeutlicher Zellhaufen. *Stria vascularis* colloid entartet, gefässlos. Nur spärliche *Spiralganglien* und *Acusticusfasern*. Da abgesehen von der

Verlötung der Membrana Reissneri mit der Basilarmembran keine Veränderungen vorhanden waren, die man als Residuen eines entzündlichen Prozesses mit Sicherheit ansehen kann, und da ferner noch ein *Bruder* von Jugend an *taub* war, ist es wohl wahrscheinlicher, dass es sich hier um angeborene Taubstummheit gehandelt hat.

3. Linck (*Anatomie der Taubstummheit*, 6. Lief.). Beiderseits, Mittelohr und Labyrinthkapsel ohne Besonderheiten. *Stria vascularis*, *Cortisches Organ* und zum grossen Teil auch *Cortische Membran fehlend*. *Ektasie* des Ductus cochlearis und *Atrophie* des *Nerv. cochlearis* und des *Ganglion spirale*. Starke Pigmentablagerungen im *bindegewebigen Schneckengerüst*. In der Basalwindung beide Skalen mit lockerem Maschengewebe ausgefüllt, das in das histologisch gleichgeartete *Ligament. spirale* übergeht. Verf. hält dieses Maschengewebe nicht für das Produkt einer abgelaufenen Entzündung, sondern sieht es an als restierendes embryonales Bindegewebe, das nicht zur Involution gekommen ist. Für die intrauterine Provenienz der Veränderungen spricht ferner nach Lincks Ansicht die Gleichartigkeit des Befundes bezüglich der Ausdehnung und der histologischen Beschaffenheit des Fasergewebes auf beiden Seiten. Demgegenüber betont Goerke (*Centralblatt f. Ohrenheilk.*, Bd. viii, S. 392) nach meiner Ansicht mit Recht, dass die Ausbreitung und Intensität der entzündlichen Veränderungen bei meningeal entstandenen Labyrinthitiden sich oftmals auf beiden Ohren vollkommen gleichen, und dass das in der Basalwindung gefundene weitmaschige Bindegewebe auch als Residuum einer abgelaufenen Labyrinthentzündung aufgefasst werden kann. Es erscheint mir demnach zweifelhaft, ob der von Linck publizierte Fall der angeborenen oder der postfötal erworbenen Taubstummheit zuzurechnen ist.

DISCUSSION

Dr. Castex (Paris). D'après mon expérience personnelle, les sourds-muets ont souvent des altérations dans la région auditive du cortex cérébral, l'oreille moyenne est peu atteinte. La cause en est dans des méningites parfois intra-utérines de l'enfant.

Prof. Uchermann (Christiania). I regret having received the report of Prof. Mygind so late that I can only make a few remarks. Mygind ends his report by saying : The pathology of deafmutism acquired after birth is *really* the pathology of infantile labyrinthitis, &c. I agree with him if instead of *really* he would say *chiefly*. From my investigation of about 800 deafmutes in Norway, published in my work, *La Surdi-mutité en Norvège*, 1896 (to be found in the library of the College of Surgeons), I found eight cases in which the examination showed Rinné − and Schwabach + to the same extent, and in one case, a grown-up deafmute, the hearing by applying an artificial tympanum became *normal*. That also

shows the necessity of examining the living deafmutes in the same way as we examine other deafs with the help of tuning-forks, and especially bone-conduction in relation to air-conduction (Rinné, Schwabach). The opinion of Bezold, that it is impossible to get reliable results with deafmutes, is wrong if you get the aid of an intelligent, educated deafmute and the deafmutes themselves have learnt to distinguish between feeling and hearing. In this way I have examined many hundred cases, and, previous to Bezold, have shown that most of the cases were of labyrinthine origin, a few following disease of the middle ears. In regard to Dr. Castex's remarks, I do not think he is right in believing that most cases of deafmutism are caused by changes in the cerebral cortex. In most cases cerebral changes are secondary and due to want of development caused by inactivity of the temporal part of the brain (mutism, deafness).

Prof. BRÜHL (Berlin) demonstriert Präparate von den seltenen Fällen einer sicher congenital tauben Patientin, bei der gefunden wurden: starke Hyperostosen an beiden hinteren Pyramidenflächen; Aplasie in der Ausbreitung der rechten Cochlearis; im Cortischen Organ, Spiralganglion, in der rechten Cochlearis selbst, in beiden Felsenbeinen symmetrisch.

Prof. KÜMMEL (Heidelberg) fand bei den ausgedehnten Untersuchungen von Taubstummen in den badischen Anstalten (etwa 1500 Schüler sind im Laufe der Jahre genau untersucht worden), dass unter den 8 bis 9 Prozent (von etwa 300 nach Wassermann untersuchten) hereditärsyphilitischen Zöglingen mehrmals mehrere Kinder gleicher Eltern waren, und dass bei diesen mindestens einmal die Taubstummheit kongenital war. Es fanden sich aber auch taube Geschwisterpaare ohne Wassermann'sche Reaktion, und mehrfach waren diese die Kinder von Eltern, die beide taubstumm waren.

Prof. ALEXANDER (Vienna) found as characteristic signs of an important percentage of *congenital deafness* (about 70–75 per cent.):

(1) The sacculo-cochlear degeneration (pars superior intact — pars inferior diseased).

(2) Remains of hearing.

In cases of acquired deafness we find in about the same percentage of cases:

(1) The whole membranous labyrinth degenerated.

(2) Complete deafness, no remains of hearing.

Dr. JOBSON HORNE (London) said that in a short contribution which he had made to the subject [1] he had expressed the opinion that meningitis, especially epidemic cerebro-spinal meningitis, and scarlet fever were more frequently the cause of acquired deafmutism than all other infec-

[1] *The Treasury of Human Inheritance*, Pts. i and ii, p. 27, Deafmutism University of London, Francis Galton Laboratory for National Eugenics, 1909.

tious diseases of childhood put together. At the same time he considered that mumps and hereditary syphilis as causative factors had not been taken sufficiently into account. Direct heredity from the parent and the grandparent as shown by the pedigrees collected by the Galton Eugenics Laboratory was not so very rare as some authorities had stated. A child need not be born stone deaf in order to develop into a deafmute. Many of the congenital cases did not come under observation during the first two or three years of life. The mother may not know that her child is dumb until the age of school life is reached, up to that time the speech may be regarded only as backward. The parents are naturally reluctant to disclose a family defect, and the tendency is then to attribute to some illness or accident in the first years of life what is really an inheritance. Furthermore, it would seem that a weakness or tendency to deafness is at times hereditary and may become patent with disease. Those were some of the difficulties to be contended with in arriving at a true estimate of the pathology of deafmutism alleged to have been acquired.

Dr. SPIRA (Krakau) spricht über Heredität von Ohrenkrankheit überhaupt, die häufiger vorzukommen pflegt, als gewöhnlich angenommen wird. Abgesehen von den bekannten erblichen Krankheiten, wie Otosklerose, Taubstummheit und progressive nervöse Schwachhörigkeit, kann man auch bei anderen Ohrenkrankheiten bemerken, dass sie in manchen Familien gehäuft vorkommen. Die Ursache kann nur in einer ererbten *lokalen* verminderten Widerstandsfähigkeit gesucht werden, welche eine gesteigerte Disposition zu Ohrenkrankheiten verursacht und auf ererbte lokale anatomische, zirkulatorische, trophoneurotische oder neuropathologische Anomalien, oder auf gemeinsame konstitutionelle Anomalien zurückzuführen sind.

REPLIES

Prof. DENKER (Halle a. d. Saale) stellt mit Befriedigung fest, dass Prof. Mygind ebenfalls der Ansicht ist, eine Einteilung in Untergruppen und Typen nicht zu empfehlen. Er ist einverstanden, anstatt ' im Leben erworbene Taubstummheit ', zu sagen ' nach der Geburt erworbene oder postfötale Taubstummheit ', und hat das bereits in seinem Referat zum Ausdruck gebracht. Redner bezweifelt die Richtigkeit der Annahme von Castex, dass die angeborene Taubstummheit häufiger sei als die erworbene. Er empfiehlt bei der Untersuchung nach Konsanguinität der Erzeuger darauf zu achten, ob die Eltern sonstige hereditär-degenerative Symptome aufwiesen. Gegen die Ansicht Uchermanns scheint dem Referenten der Umstand zu sprechen, dass doppelseitige angeborene knöcherne Atresie des Gehörorgans keine Taubstummheit hervorzurufen pflegt.

Prof. Denker demonstriert zur Einleitung der Diskussion über die Pathologie der Taubstummheit eine Reihe von Diapositiven von Fällen,

die den verschiedenen Gruppen der angeborenen Taubstummheit angehören. Es wurden mit dem Projektionsapparat gezeigt Fälle von Siebenmann, Denker, Alexander, Siebenmann-Oppikofer, Manasse.

Prof. MYGIND (Copenhagen). Several of the gentlemen who have taken part in the discussion have spoken about the causes of deafmutism. As this is a very big question and does not come under the topic which has formed the subject of the papers read, I shall not enter upon them.

Prof. Castex lays great stress upon atrophic alterations of the temporal lobe of the brain. I must say that I cannot agree with him that they form a basis for the deafness. In my opinion these alterations are either caused by inactivity of the centres of speaking, or they are caused by the meningitis which has been the primary cause of the labyrinthine deafness.

To Prof. Uchermann I should say that as the results arrived at in my paper are deduced from post-mortem examinations, and as none of the reports published as yet have proved the existence of deafmutism caused by middle-ear disease, I am justified in using—in our present state of knowledge—the word ' really ' in my final remarks instead of ' chiefly ' as proposed by Prof. Uchermann.

Prof. Alexander has in his remarks pointed out the fact that the pars superior of the labyrinth is often preserved in cases of congenital deafmutism. I do not think, however, that this circumstance is of much value in distinguishing congenital cases from acquired ones, as this part of the membranous labyrinth is often also little affected in cases of acquired deafmutism.

As to Dr. Jobson Horne's remarks, I want to state that I have said in my paper that there is no marked line between the alterations formed in acquired deafmutism and those described in *some* congenital cases.

At the close of the discussion a demonstration of the pathology of deafmutism was given by Prof. SIEBENMANN (Basle).

SECTION XVI

OTOLOGY

INDEPENDENT PAPER

ZUR 'THERAPIE' DER 'SUBJEKTIVEN OHRGERÄUSCHE'

Von Professor W. KÜMMEL, Heidelberg

Schäfer und Hegener [1] haben vor drei Jahren der Deutschen otologischen Gesellschaft ein ausgezeichnetes Referat über subjektive Geräusche und Töne erstattet, dem ich die Anregung verdanke, mich mit diesen für unsere Patienten so oft bedeutungsvollen Erscheinungen eingehender zu beschäftigen. Ich will aber hier keineswegs neue Anschauungen über die Entstehung der subjektiven Geräuschempfindungen vortragen, sondern nur über eine Erkenntnis bezüglich der Therapie sprechen, die mir immer klarer und zweifelloser geworden ist: dass nämlich alle die vielen Behandlungsmethoden, die in unseren Lehrbüchern und in allen Einzelarbeiten immer wieder empfohlen werden, und die auch ich früher immer verwendet habe, absolut unrationell, und deshalb unzulänglich sind. Ich muss heute, auf die Gefahr des schärfsten Widerspruchs hin, die begründete Ueberzeugung aussprechen, dass *jede eigentliche Therapie*, die sich gegen die subjektiven Geräusche wenden will, *eine systematische Schädigung* der Patienten bedeutet; und dass die einzige rationelle Behandlung darin besteht, dass man die Patienten lehrt, sich mit den subjektiven Geräuschen abzufinden, wenn man nicht etwa in der Lage ist, die schuldige Ohrerkrankung und die dadurch bedingte Hörstörung ganz oder nahezu ganz zu beseitigen.

Bei Manchen würde ich eher Zustimmung finden, wenn ich diese Behauptung auf eine nicht eben zahlreiche Gruppe von Patienten beschränken würde: diejenigen, bei denen subjektive Geräusche bestehen, ohne dass eine Hörstörung nachweisbar ist. Neuerdings finde ich wenigstens bei Wittmaack [2] für diese Gruppe von Pat. die Notwendigkeit hervorgehoben, dass man da die psychischen Faktoren in erster Linie berücksichtigen müsse. Für die Fälle mit tatsächlicher Gehörsverminderung aber gibt auch W. eingehende therapeutische Vorschriften; und in dieser

[1] Schäfer, 'Die subj. Töne u. Geräusche'; Hegener, 'Klinik, Pathol. etc. der subj. Ohrgeräusche,' *Verhandl. d. Deutsch. otolog. Ges.*, Basel, 1909.

[2] 'Die Prognose u. Behandl. der Ohrgeräusche,' *Deutsch. med. Woch.*, 1912, No. 39.

Richtung besteht eine gewisse Meinungsdifferenz zwischen uns, die aber, wie ich hier gleich feststellen will, eigentlich nur scheinbar ist. Ich verzichte auf alle Literatur, die ich übrigens eifrig studiert habe, und möchte rein praktisch meine Auffassungen darstellen in Form eines, Ihnen hoffentlich nicht zu langweiligen, Dialogs mit einer gebildeten älteren Dame, die mich wegen der 'unerträglichen Geräusche im Kopfe' konsultiert. Ich frage: 'Hören Sie denn nicht schlecht?' 'Ganz gut höre ich nicht; aber das wäre mir ganz gleich, wenn ich nur das fürchterliche Brausen und Klopfen und Singen im Kopfe los werden könnte. Gibt es denn gar kein Mittel dagegen?' 'Nein, gegen das Sausen gibt es nur ein Mittel, aber das möchten Sie schwerlich angewendet sehen.' 'Doch, ich lasse mir gern alles gefallen, wenn ich nur die entsetzliche Quälerei los werde.' 'Dann müsste man das Herz zum Stillstand bringen: darauf werden Sie aber wohl verzichten?' 'So müsste ich also ewig diese Quälerei tragen?' 'Ja: die Geräusche lassen sich nicht *wegbringen*, solange der Patient lebt, aber sie lassen sich sehr leicht *ertragen*, wenn man sich nicht krankhaft mit ihnen beschäftigt.' 'Oh, ich beschäftige mich gar nicht mit ihnen; ich gebe mir alle Mühe, sie zu vergessen, aber das ist vollständig unmöglich. Sie wecken mich in der Nacht und hindern mich am Schlafen. Wenn ich einen Brief schreibe, verwirren sich mir die Gedanken durch das fortwährende Toben in meinen Ohren, und ich bin vollständig unfähig, irgend etwas Vernünftiges zu tun, wenn die Ger. so stark sind.' Ich frage ihren Mann: 'Ist Ihre Frau Gemahlin nicht schon, ehe sie über die Ger. geklagt hat, ein wenig nervös gewesen?' Die Antwort ist ein sehr energisches: 'Jawohl! Meine Frau hat schon immer sich sehr leicht aufgeregt, dieses wohl nach aussen verborgen, aber selbst sehr darunter gelitten, und ihre Schlaflosigkeit ist schon seit Jahren sehr lästig gewesen.' Und die Frau gibt schliesslich wenigstens zu, dass die Schlaflosigkeit auch schon früher gelegentlich bestanden hätte.

Den Befund bei der Untersuchung übergehe ich als gleichgültig für unser Thema: alles spielt sich in genau der gleichen Weise ab bei dem Befunde einer typischen Acusticuserkrankung, wie bei der klassischen Bezold'schen Trias, bei der doch fast alle Kollegen die Diagnose auf 'Otosklerose' stellen. Die Hörstörung ist ziemlich erheblich, aber auch das ist belanglos: man beobachtet genau die gleichen Dinge bei nahezu vollständiger Taubheit, wie bei ganz leichten Hörstörungen. Wesentlich für die Beurteilung der Therapie ist nur der Umstand, dass eine Besserung des Gehörs wenig aussichtsvoll erscheint, und dass man der Pat. eigentlich nur einen *Versuch* zur Behandlung empfehlen kann. Ich bringe ihr das mit Schonung bei. Sie nimmt es mit verhältnismässiger Resignation hin, fragt dann aber umso ängstlicher, ob sich denn gar nichts gegen die Geräusche tun lasse, wenn schon die Schwerhörigkeit einmal ertragen werden müsste. Ich lasse mir erzählen, was bisher geschehen ist. 'Zuerst war ich bei Herrn Dr. X. Der blies mehrere Male Luft in die Paukenhöhle ein, sagte aber von vornherein, es würde vielleicht nicht viel helfen. Dann machte er noch mehrere Male eine elektrische Massage, und die schien

auch zuerst ganz gut zu tun, aber dann wurde nach jeder Behandlung das Sausen viel schlimmer, und schliesslich bin ich nicht mehr hingegangen. Mein Hausarzt hat mich dann veranlasst, zum Dr. Y. zu gehen. Der hat mich sehr genau untersucht und mir gesagt, dass die Schwerhörigkeit langsam zunehmen werde, zu machen sei nichts dabei, sie sei aber noch gar nicht schlimm, liesse sich auch vielleicht durch Phosphor oder andere Mittel aufhalten, und es würde wohl noch lange dauern, ehe ich schwerere Störungen merken würde. Vollständige Taubheit würde auch nicht eintreten, wenn nicht etwa eine Gehörnervenerkrankung dazu käme. Gegenwärtig handle es sich nur um einen trockenen chronischen Mittelohrkatarrh. Gegen die Ger. hat er mir vorerst neben dem Phosphor Bornyval verschrieben, nachdem das früher vom Hausarzt gegebene Bromkali nichts genutzt hat. Beides habe ich genommen, aber dann wurden auch dabei die Ger. immer ärger, und so habe ich's denn weggelassen. Schliesslich habe ich noch Herrn Prof. Z. in N. aufgesucht, weil mir Bekannte gesagt haben, dass er einen ganz besonderen Ruf für solche Erkankungen hätte. Der hat mich eine Zeitlang elektrisiert und mir dann noch einen Brief an unsern Hausarzt wegen elektrischer Behandlung mitgegeben. Der Hausarzt meinte aber, das würde mich sehr nervös machen, und deshalb hat er gar nicht damit angefangen. Er hat mir nur gesagt, ich müsste die Ger. vergessen und nicht beachten ; aber das kann ich nicht.' 'Welcher Art sind denn die Geräusche ?' Ich gebe der Pat. die Matte'sche Liste zur Auswahl, und sie bezeichnet alsbald eine grosse Menge der dort verzeichneten Geräuschqualitäten, sagt aber, dass diese ausserordentlich wechseln, und dass sie eigentlich fast alle diese Geräusche schon einmal gehört hätte. Sehr oft handelt es sich natürlich auch um Geräusche ganz bestimmter Qualität und Stärke. Aber auch darauf möchte ich gar keinen Wert legen, ebensowenig auf die Vergleiche, die sie bezüglich der Stärke gibt, und die Ihnen allen zum Ueberdruss bekannt sind. Dann kommt wiederum die Frage : 'Was soll ich aber nun gegen diese Geräusche tun ?' 'Vor allen Dingen müssen Sie wissen, dass *jeder Mensch*, mit gesunden oder mit kranken Ohren, an jedem Tage seines Lebens in der Lage ist, solche Geräusche wahrzunehmen, wie Sie sie empfinden.' 'Aber doch nicht so kolossal stark, wie ich sie höre ?' 'Ja, dabei müssen Sie berücksichtigen, dass die Stärke irgendeiner Gehörsempfindung immer von zwei Dingen abhängt : einmal von der wirklichen Stärke des Geräusches oder Tones, der sie hervorruft, und zweitens von der subjektiven Empfindlichkeit, die Ihr Hörapparat besitzt. Ich brauche Sie nur daran zu erinnern, dass man die Mücke in der Nähe ebenso gross sieht, wie den Adler in der Ferne. Es kommt also schon viel darauf an, wo Sie sich die Entstehung dieser Geräusche im Geiste vorstellen. Und ferner : Sie werden doch gewiss, wenn Sie jetzt eine Lokomotive pfeifen oder den Sturm heulen hören, die Vorstellung haben, dass das sehr laut ist ; und trotzdem dürfen Sie sicher sein, dass Sie diese Geräusche mit Ihrer Hörstörung vielleicht nur so laut hören, wie Normal-Hörende das Zwitschern eines Sperlings oder das Summen einer Biene. Also wird

der Normal-Hörende eine Geräuschempfindung, die *Ihnen als sehr laut* erscheint nur als ganz leise bezeichnen können. So ist es doch ganz natürlich, wenn Sie Geräusche als sehr stark empfinden, die, objektiv genommen, äusserst schwach sind, die Sie aber deutlich wahrnehmen, weil sie ganz dicht an Ihrem Ohre, oder gar in Ihrem Ohre, entstehen, obwohl sie objektiv so schwach sind, dass kein anderer Mensch sie wahrnehmen kann.' ' Ja, das mag wohl richtig sein, aber für mich sind doch einmal die Geräusche so furchtbar stark, mag das auch nur subjektiv sein. Und was mich am meisten quält, ist, dass sie mich immer zwingen, an mein Ohrenleiden zu denken ! ' ' Dann sind Sie wohl auch der Ueberzeugung, dass die Geräusche ein Zeichen dafür sind, dass die Ohrerkrankung noch fortschreitet ? ' ' Jawohl, das hat mir aber doch auch Herr Dr. Y. gesagt ! Ist das denn nicht der Fall ? ' ' Ja, darüber sind sich die Gelehrten noch nicht einig ; es gibt allerdings Kollegen, die behaupten, dass die Geräusche durch eine innere Reizung des Gehörnerven entständen, und dass diese eben durch den Krankheitsprozess herbeigeführt werde, aber andere sind durchaus der Ansicht, dass die Geräusche vielmehr nichts anderes seien, als Geräusche in den Blutgefässen, übrigens auch in den Muskeln. Am Ohr entlang gehen so viele und so grosse Blutgefässe, dass man sich eigentlich nur wundern muss, wenn man nicht *mehr* von diesen Geräuschen hört. Jedermann ist auch in der Lage, diese Gefässgeräusche bei ganz normalem Gehör wahrzunehmen, wenn man sie in geeigneter Weise verstärkt ; also z. B., wenn die Kinder eine Muschel vors Ohr halten und dann sagen, sie hörten das Meer darin brausen. In Wirklichkeit hören sie dann nichts anderes als eben die Blutgefässgeräusche.' ' Aber *das* kann nicht stimmen : diese Geräusche in einer solchen Muschel habe ich als Kind, und so lange mein Gehör noch gut war, sehr deutlich hören können, das war aber stets ein richtiges Brausen. Was ich jetzt höre, ist nicht blos das, sondern manchmal ein Singen oder Sieden, wie von einem Teekessel, manchmal ist es ein Klopfen, und manchmal ein richtiges Heulen, wie wenn der Wind ins Kamin bläst.' ' Ja, dieser Einwand wird immer gemacht, aber Sie müssen bedenken, dass Schlagadern und Blutadern direkt am Ohr vorbei gehen, und dass die ersteren mit dem Herzschlag regelmässig klopfende, die letzteren dagegen gleichmässige Geräusche verursachen, die man eher als ein Sausen oder Heulen bezeichnen kann. Haben Sie denn noch nicht bemerkt, dass die Geräusche besonders heftig sind, wenn Sie sich aufgeregt haben, und dass sie nahezu unhörbar werden, wenn Sie in einer lebhaften und Sie interessierenden Unterhaltung begriffen sind ? ' ' Ja, das ist allerdings richtig, besonders bei Aufregungen werden die Geräusche oft ganz unerträglich.' ' Und Sie geben auch zu, dass Sie in behaglicher, vergnügter Unterhaltung oder bei lebhaft interessierender Lektüre u. dgl. die Geräusche nicht bemerken ? ' ' Ja, aber sie sind sofort wieder da, wenn ich darauf achte ! ' ' Also, da geben Sie ja selbst zu, dass die Wahrnehmung der Geräusche davon abhängig ist, ob Sie darauf Acht geben ! Andererseits werden Sie selten z. B. solche starken klopfenden Geräuschempfindungen haben, wenn Sie etwas sehr Erfreuliches erleben :

Sie sehen daraus, dass die Stärke der Geräusche sicher nicht nur abhängt von der Stärke des Herzschlages, also von ihrer wirklichen Stärke, die bei freudigen und unangenehmen Erregungen annähernd gleich ist, sondern auch von der Empfindlichkeit des Nervensystems, die augenblicklich gerade vorhanden ist.' 'Nun, dann müssten doch die Geräusche zeitweilig aufhören ?' 'Das ist auch wohl wirklich der Fall, d. h. nicht das Geräusch hört auf, sondern Sie hören auf, das Geräusch wahrzunehmen. Ganz dasselbe können Sie auch bei äusseren Geräuschen beobachten. Denken Sie nur daran, wie oft Sie das Quietschen einer Türe oder den Lärm, den die Kinder beim Spielen machen, gar nicht hören, wenn Sie eifrig mit irgend etwas beschäftigt sind, oder wenn Sie in guter Stimmung mit irgend jemand sich unterhalten. Und denken Sie, wie gewaltig Sie dasselbe Geräusch wieder stört, wie gellend Ihnen das Geschrei der spielenden Kinder erscheint, wie Sie zusammenfahren, wenn die Tür quietscht, sobald Sie sich aus irgendeinem Grunde in schlechter Laune befinden, oder ängstlich auf irgend etwas warten, oder sich körperlich unwohl fühlen, oder auch nur einmal schlecht geschlafen haben!' 'Zugegeben. Aber die Geräusche müssen doch wirklich sehr stark sein, denn sie hindern mich doch z. B. sehr oft am Schlafen.' 'Umgekehrt, wenn Sie schlecht schlafen, dann hören Sie die Geräusche stark, denn dann ist einmal durch den schlechten Schlaf Ihre Empfindlichkeit grösser, und zweitens bleiben in der Nacht die Geräuscheinwirkungen von draussen für Sie aus. So machen Ihnen die Geräusche in Ihrem Kopf in der schlaflosen Nacht einen viel stärkeren Eindruck. Dass sie Sie aber am Schlafe *hindern*, kann ich bestimmt in Abrede stellen.' 'Ich muss aber doch glauben, dass diese Geräusche sehr stark sind, denn zuweilen machen sie mir das Hören direkt unmöglich, selbst wenn ganz laut gesprochen wird.' 'Nun das werden Sie aber besonders bemerken, wenn Sie in Gesellschaft mit verschiedenen Personen sich unterhalten. Dann verstehen Sie naturgemäss schlecht, geben sich aber viel Mühe, Ihre Aufmerksamkeit auf das Gespräch zu konzentrieren, weil Sie gern an der Unterhaltung teilnehmen möchten: durch das Lauschen wird in genau der gleichen Weise die Empfindlichkeit für die Geräusche im Innern Ihres Kopfes, wie für die Sprachgeräusche draussen gesteigert. Kommt dazu noch das begreifliche Unbehagen wegen der mangelhaften Verständigung, dann werden Sie auch viel empfindlicher sein für die Geräusche in Ihrem Innern, und je mehr Sie sich ärgern, um so schlechter werden Sie dann die äussern Geräusche wahrnehmen, und um so stärker muss Ihre Empfindlichkeit für die in Ihrem Kopfe entstehenden Geräusche sein, und selbstverständlich werden Sie die dann als störend empfinden. Hat Sie nicht schon öfter ein leichtes Geräusch, das in Ihrem Haus alltäglich vorkommt und das Sie sonst gar nicht beachten, sehr erheblich gestört, wenn Sie mit einem undeutlich sprechenden Menschen sich zu unterhalten gezwungen waren ? Und doch ist dieses Geräusch genau dasselbe, das Sie bei der Unterhaltung mit einem deutlich sprechenden Menschen oder mit einem Angehörigen, dessen Stimme Sie gewöhnt sind, nicht gestört hat.' 'Also da meinen Sie,

dass die ganzen Geräusche, die ich höre, nichts Krankhaftes wären ?' 'Ganz richtig, die Geräusche sind nicht krankhaft ; nur dass Sie sie hören, ist krankhaft.' 'Ja, dann verstehe ich nur eines nicht : wie ist es dann möglich, dass gesunde Leute diese Geräusche, die auch bei ihnen vorhanden sind, überhaupt nicht hören ?' 'Auch das ist sehr einfach. In Wirklichkeit kann jeder Mensch, der sich einige Mühe gibt und sich zu beobachten versteht, auch bei ganz normalem und sogar bei hervorragendem Gehörvermögen an jedem Tage seines Lebens mehr oder weniger Geräusche wahrnehmen. Allerdings ist dazu eine besondere Aufmerksamkeit und Fähigkeit zur Selbst-Beobachtung nötig. Diese lernt aber ein Mensch, der, ob er will oder nicht, sein Ohr täglich anstrengen und mit ihm besonders scharf aufpassen muss, sehr viel leichter als ein gesunder. Immerhin haben wir eine Menge von Beobachtungen von ganz gesunden Menschen über solche Geräusche. Als der Physiologe Zwaardemaker sich einen ganz schalldichten Raum hergestellt hatte, was gar nicht leicht ist, glaubte er zuerst zu seiner grossen Überraschung, immer eine Wasserleitung darin rauschen zu hören. Erst nach einiger Zeit kam er dahinter, dass das nur die Blutgeräusche in seinem eigenen Ohr waren. Der berühmte Akustiker Stumpf, ein Mann mit einem ganz ausserordentlich feinen Gehörvermögen, hat durch viele jahre die subjektiven Gehörsempfindungen genau aufgezeichnet, die bei ihm vorkamen. Er hat jeden Tag solche Geräusche wahrgenommen, und zwar in allen erdenklichen Tonlagen, gleichmässige und unterbrochene, hohe und tiefe, richtige Geräusche und mehr an Klänge erinnernde Wahrnehmungen, und sogar gelegentlich ganz reine Töne. Von andern Leuten mit vorzüglichem Gehör haben wir ähnliche Angaben ; z. B. war der Königsberger Berthold sogar in der Lage, seine Geige nach dem Ton zu stimmen, den er in seinem Ohr dauernd hörte. Alle diese Leute haben ein ausgezeichnetes Gehör gehabt und bis in ihr hohes Alter behalten. Aber ich kann Ihnen auch Beobachtungen aus dem täglichen Leben nennen, die das Vorkommen von solchen 'störenden' Geräuschen beweisen. Es gibt Menschen, die sehr fein hören, die aber sehr ungern telephonieren, einfach deshalb, weil sie, sobald sie das Telephon ans Ohr halten, ein lautes Sausen und Klopfen hören und deshalb die Sprache im Telephon gar nicht verstehen zu können glauben. Bei denen würde also ganz dasselbe gelten wie bei Ihnen, wenn Sie wegen Ihrer Geräuschempfindungen jemand andern nicht verstehen können. Aber einige Geduld lässt dann stets sehr bald die Geräusche so verschwinden, dass sie gar nicht mehr bemerkt werden. Und das gilt auch für Sie ! Also, damit wir einmal mit unserer Unterhaltung fertig werden : die Geräusche sind in der Tat nichts Krankhaftes bei Ihnen, sondern Sie haben es nur gelernt, sie zu hören. So gut Sie's aber *gelernt* haben, können Sie es auch wieder *verlernen*. Für das Gehör müssen wir sehen, was sich tun lässt, aber Sie müssen sich an die Tatsache gewöhnen, dass die Geräusche sich nicht beseitigen lassen ; und ich weiss auch, dass Sie es fertig bringen werden, sie wieder ertragen und nicht beachten zu lernen, so, wie Sie sie vor Ihrer Ohrerkrankung und zu Zeiten, wo der Zustand

Ihrer Nerven besser war, als jetzt, ohne irgendwelche Beschwerden ertragen und überhaupt nicht bemerkt haben. Versuchen Sie es nur, dann dürfen Sie überzeugt sein, dass es Ihnen auch gelingen wird. Aber gewiss werden Sie noch oft genug an das Vorhandensein dieser Klopfgeister und Brummkreisel erinnert werden. Wenn Sie aber wissen, was sie zu bedeuten oder vielmehr nicht zu bedeuten haben, dann können sie Ihnen nichts tun, und Sie können leicht mit ihnen fertig werden.'

Jedenfalls sehen Sie aus dieser möglichst getreu kopierten Unterredung, dass ich bei solchen Pat. irgendeine Behandlung gegen die Ohrgeräusche einzuleiten durchaus vermeide, weil ich bestimmt glaube, ihnen damit einen schlechten Dienst zu erweisen. Das Medikament oder die sonstige Therapie, die wir anwenden, helfen gegen die Erscheinung, die wir bekämpfen wollen, nichts ; oder wenn wir etwas damit erreichen, so ist das nur eine Suggestionswirkung, und eine solche Suggestionswirkung ist allemal sehr unsicher und erschöpft sich nach kurzer Zeit. Dann kommt sehr bald der Moment, wo die Pat. sich sagen : ' Jetzt hilft also auch dieses soundsovielte Mittel nicht : man möchte verzweifeln ! ' Es ist selbstverständlich, dass dann die Neurasthenie zur kräftigsten Blüte gelangt. Freilich ist nicht einmal immer die Mitwirkung des Arztes dazu erforderlich, denn sehr häufig haben sich die Pat. selbst so eifrig mit den Geräuschen beschäftigt und haben eine so bestimmte Vorstellung davon, wo und wie sie entstehen, dass es sehr schwer wird, diese oft absurden Vorstellungen ihnen wieder auszureden. Meiner Meinung nach können wir uns bei der Frage nach der *Behandlung* der subjektiven Geräuschempfindungen die an sich so interessante Frage nach ihrer *Entstehung* vollständig schenken. Es genügen *drei Tatsachen* zur richtigen Beurteilung der Therapie : einmal die, dass vollkommen gesunde Menschen mit normalem Hörvermögen und gesundem Nervensystem subjektive Geräusche häufig, und selbst dauernd, wahrnehmen, ohne irgendwie dadurch gestört zu sein ; zweitens die gleichfalls feststehende Tatsache, dass es sehr viele Pat. mit gleichartigen und gleich schweren Hörstörungen gibt, bei denen keinerlei Belästigung durch subjektive Geräusche zustande kommt, obwohl wir bei genauerer Nachforschung von ihnen die Existenz solcher oftmals mitgeteilt bekommen ; schliesslich drittens die Tatsache, dass auch manche Menschen mit normalem Gehör schwer an solchen subjektiven Geräuschen laborieren. Wenn wir uns zu der Annahme berechtigt glauben — und daran zweifelt wohl niemand — dass solche subjektive Geräusche *bei normalem Gehör neurasthenischen* Ursprungs sind, so berechtigt uns meiner Meinung nach nichts, bei den Pat. *mit objektiven Gehörsstörungen* anzunehmen, dass bei ihnen die subjektiven Geräusche tatsächlich stärker als bei Ohrgesunden, und deswegen die dadurch bedingten Beschwerden *nicht als psychogen*, neurasthenisch anzusehen seien.

Wenn ich den Patienten gegenüber für die Entstehung der Geräusche regelmässig die Blutgefässe verantwortlich mache, so bin ich mir vollkommen darüber klar, dass manche Geräusche sicher anders entstehen.

Aber die Entstehung der Geräusche in den Blutgefässen ist für den Laien am leichtesten verständlich, und deshalb ziehe ich vor, ihnen diese Deutung zu geben. Man muss den Patienten vor allem eine plausible Erklärung für die Geräusche geben, die ihnen nicht die Vorstellung rechtfertigt, dass diese das Zeichen eines fortschreitenden Krankheitsprozesses im Ohr, oder, wie sie nicht so selten auch denken, im Gehirn seien. Darüber dürfen wir uns doch keine Illusionen machen, dass wir die Geräusche selbst tatsächlich nicht objektiv abschwächen können; hätten wir dafür keinen anderen Beweis, so genügte schon der Umstand, dass subjektive Geräuschempfindungen von erheblicher Stärke und Dauer auch bei ganz gesunden Menschen vorkommen, wie das ja schon deren volkstümliche Deutungen demonstrieren. Wir dürfen uns doch nicht einbilden, dass wir das Gehörorgan eines Mannes wie Stumpf durch eine noch so raffinierte Behandlung besser machen könnten, als es ihm die Natur gegeben hat! Können wir bei *ihm* die subjektiven Geräusche *nicht* beseitigen, was Sie mir wohl nicht bestreiten werden, so dürfen wir *ebenso wenig bei dem kranken Ohre* einen solchen Erfolg erwarten. Wenn gesunde Menschen starke subjektive Geräuschempfindungen haben, einerlei aus welcher Ursache immer sie entstehen mögen, so können auch bei Ohrenkranken die subjektiven Geräusche nicht krankhaft sein. Vielmehr dürfen nur die Beschwerden, die die Pat. dadurch empfinden, als krankhaft, und zwar als psychogen bezeichnet werden. Damit haben wir meiner Meinung nach den Schlüssel zur Therapie in dem Sinne, den ich Ihnen angedeutet habe, nämlich, dass man den Pat. klar machen muss, die subjektiven Geräusche seien nicht zu beseitigen, aber sie seien auch für das Leiden selbst bedeutungslos, und wenn man sich nicht unnötig den Kopf darüber zerbreche, sei es auch leicht, die Beschwerden, die sie machen, los zu werden.

Ich bitte Sie, sich einmal die Mühe nicht verdriessen zu lassen und in ähnlicher Weise die psychogenen Faktoren bei Ihren Pat. zu berücksichtigen, wie ich das vorher durch ein Beispiel anzudeuten versucht habe. Ich gebe vollständig zu, dass sich die Zeit, die man auf eine solche Unterhaltung verwendet, für den Arzt angenehmer und gewinnbringender mit irgendeiner mechanischen Therapie oder dem Verschreiben eines Rezeptes ausnutzen lässt; ein Vergnügen sind solche Unterhaltungen selten, und die vorher angeführte ist noch lange keine der unangenehmsten. Ich habe selbst immer die Empfindung, man könnte eine solche Unterhaltung eigentlich recht gut durch einen Phonographen abwickeln lassen! Für den Pat. ist aber ein solcher Dialog oft sehr gewinnbringend: ich kenne vielleicht besonders viele, die von Arzt zu Arzt gewandert sind, und bei denen es sich erst durch eine solche Auseinandersetzung erreichen liess, dass sie auf weitere ärztliche Behandlung verzichteten und sich mit ihrer Schwerhörigkeit abfanden, an der sie immer noch genug zu tragen haben. Wenn wir schon das schlechte Gehör eines Pat. nicht verbessern können, so ist das auch für uns gewiss betrüblich genug; es ist aber schliesslich besser, das einzugestehen, und

den Pat. in einen psychischen Zustand zu bringen, der es ihm ermöglicht, sein Leiden verhältnismässig gut zu ertragen, als wenn wir ihn durch nutzlose therapeutische Bemühungen immer mehr in das Elend der Neurasthenie hineinjagen. Natürlich gibt es Fälle, in denen auch solch eine Behandlung vergeblich ist ; aber das sind Leute, deren psychischer Zustand sich eben nicht aufbessern lässt. Natürlich hängt auch bei solchen Bemühungen viel, wenn nicht Alles, von den persönlichen Eigenschaften des betr. Arztes und Patienten ab. Es gibt Patienten, die angeschnauzt sein wollen, und solche, die man sanft streicheln muss ; Patienten, denen man seine eigene Unsicherheit und Unkenntnis in Einzelheiten ruhig eingestehen darf und muss, und solche, die verlangen, dass man ihnen irgendeinen blauen Dunst vormacht ; die Letzteren werden wir als Aerzte wohl niemals ganz richtig behandeln können. Ihre Krankheiten sind das Feld, auf dem Kurpfuscher und Wallfahrtsorte ihre schönsten Ernten einheimsen.

DISCUSSION

Dr. Halász (Miskolcz). Ich glaube es sind viele Fälle bei der Behandlung der Ohrgeräusche, welche ohne mechanische Einwirkung zu behandeln und verbessern sind, nämlich durch diätetisches Verhalten. Ich behandelte sieben Fälle von Ohrgeräuschen, welche durch *Fernhalten von Fleischessen und animalischem Eiweiss* (Eiern) für längere Zeit eine Heilung der Ohrgeräusche nach sich zogen. Ich möchte den Herren Kollegen sehr anraten, diese diätetische Behandlungsmethode zu versuchen.

Dr. Spira (Krakau). Es muss vor allem auf objektive lokale Veränderungen gefahndet werden. Wo Arteriosklerose vorhanden ist, können solche Geräusche durch den Blutdruck herabsetzende Mittel (Diuretin), wo Katarrhe der oberen Luftwege bestehen, durch entsprechende Behandlung derselben günstig beeinflusst werden. Wo absolut eine objektive ursächliche Veränderung nicht aufgefunden werden kann, kann das von Professor Kümmel vorgeschlagene Verfahren sich segensreich erweisen.

Dr. Louis Bar (Nice). M. le docteur Kümmel a dit qu'on ne doit pas soigner les bruits d'oreille, mais la maladie qui les occasionne. C'est peut-être un peu excessif, quoique très sage. En effet, les bruits d'oreille sont tantôt le résultat d'une maladie acquise (bruits aigus, congestion, etc.), tantôt le reliquat d'une infirmité, la sclérose, vraie ou fausse. Tous cessent quand la surdité est comprimée mais jusque-là ils se présentent sous deux formes isolées ou associées sous forme de bruits endogènes et bruits exogènes. Les bruits endogènes sont continus, ceux exogènes sont intermittents, plus ou moins fréquents, plus ou moins continus. Ce sont ceux qu'on doit guérir, car on le peut et leur présence active les bruits endogènes souvent imperceptibles. Les bruits congestifs, qui se présentent sous forme de bruissements bourdonnants, sont à traiter comme

on traiterait une arthrite profondément placée. Les autres (bruits d'oiseaux, bruits de pluie, bruits de vents à travers les forêts) seront tantôt d'origine anémique et traités de la même manière que l'anémie, tantôt, toujours fixes, ils sont moins sentis lorsque l'infirmité les occasionne, et ne s'améliorent que par les moyens capables d'améliorer l'infirmité.

Mr. Sydney Scott (London) drew attention to cases of mild tinnitus, due to inaccessible causes, benefited by 'Rational Therapy', such as applied by Dr. Du Bois of Berne. As to the severe intractable cases, in England they had tried operation by division of the auditory nerve, and also by extirpation of the labyrinth, but with results so disappointing that the speaker could say nothing in favour of advocating either measure for 'tinnitus'.

Mr. G. J. Jenkins (London) agreed that certain cases of tinnitus, the cause of which cannot be reached by operation or general treatment, are treated most satisfactorily along the lines Professor Kümmel suggests.

Professor Alexander (Wien) stimmt Dr. Kümmel bei und meint, dass die psychische Behandlung von Patienten mit subjektiven Geräuschen sehr notwendig ist; zumeist gelingt es, die Stärke der Geräusche einzudämmen. Die chirurgische Behandlung (Labyrinthotomie, Lumbalpunktion oder Acusticusdurchschneidung) subjektiver Geräusche ist nach seiner Meinung *nicht* zu empfehlen.

Reply

Professor Kümmel (Heidelberg). Es freut mich, dass ich von mehreren Seiten Zustimmung gefunden habe zu meinen Einwänden gegen eine Therapie, welche sich nicht gegen die genau bekannte *Krankheit* wendet, sondern gegen das *Symptom* der Ohrgeräusche. Wodurch diese letzteren entstehen, habe ich absichtlich ganz unerörtert gelassen: die hypothetische Erklärung, welche Herr Neumann für manche Fälle gab, ist sicher sehr interessant und verdient alle Beachtung, aber sie bleibt eine Hypothese und kann als solche keine gute Grundlage für die Therapie geben. Selbstverständlich müssen bei einer solchen Erkrankung alle Mittel verwendet werden, die entweder die *lokale* Ohrerkrankung oder die *allgemeine* Neurasthenie günstig beeinflussen; Herrn Hialász hat mit Recht an die Wichtigkeit der Diät erinnert: aber niemals sollen die subjektiven Geräusche *als solche* behandelt werden.

SECTION XVI

OTOLOGY

INDEPENDENT PAPER

NOTE CRITICHE SULLA CURA CHIRURGICA DELLE SUPPURAZIONI CRONICHE AURICOLARI

Per il Professore GHERARDO FERRERI, Roma

Dopo un breve periodo di insolito ardore che nell' ultimo ventennio del secolo scorso pervase la maggioranza degli otologi preferendo la cura chirurgica a quella conservativa nelle suppurazioni dell' orecchio medio, chiunque segua la letteratura otoiatrica più recente resta sorpreso del silenzio che ora regna su questo argomento. Dobbiamo al precedente periodo non solamente importanti perfezionamenti di tecnica operativa nella chirurgia dell' orecchio medio e sovratutto dell' epitimpano, ma numerosi contributi di anatomia patologica e di fisiologia sulle conseguenze funzionali dei vari atti operativi compiuti sull' apparecchio di trasmissione del suono. Tutto questo poderoso lavoro che senza dubbio ha recato notevole progresso alla medicina operatoria dell' orecchio ed ha fatto conseguire risultati migliori che con gli antichi metodi più o meno conservativi, aumentando il prestigio della nostra specialità, non sembra vero sia stato purtroppo soffocato dall' ulteriore progresso compiuto dalla chirurgia otoiatrica nella cura delle complicazioni delle malattie acute e croniche suppurative auricolari. Per modo che, ad esempio, non è raro il caso di riscontrare che in un' otite media purulenta cronica, per la quale, almeno in primo tempo, alcuni anni or sono si sarebbe prima esperita la pratica endotica, ora senz' altro non si indugia ad aggredirla per la via esterna.

Questo è il soggetto che mi propongo di sottoporre ad esame critico basato su di una lunga esperienza personale, beninteso restringendomi a considerare soltanto le lesioni suppurative croniche che non oltrepassano il confine della cassa e dell' epitimpano. Noi sappiamo che interventi chirurgici attraverso il condotto uditivo nelle suppurazioni croniche della cassa e loro esiti non possono varcare certi limiti, perchè le parti malate o sfuggono all' esame otoscopico o non sono raggiungibili con strumenti introdotti nel condotto uditivo esterno. Il compito operativo, abbastanza facile se le lesioni sono localizzate alla cavità timpanica propriamente

detta, si rende più arduo nelle affezioni dell' attico, allorchè si sospetti una carie più o meno diffusa dell' epitimpano. In tali casi la maggioranza degli operatori gira la posizione, aggredendo l'antro mastoideo per demolire poi la parete esterna dell' adito e raggiungere così l' epitimpano. Naturalmente (essendo circoscritta la lesione all' orecchio medio propriamente detto o all' attico) sorge la domanda se non sia un precetto erroneo di tecnica operativa aggredire le parti sane per giungere a quelle malate, col pericolo talvolta di complicare i disturbi funzionali inerenti alla lesione della cassa con quelli accidentali prodotti dall' intervento sull' attico (paralisi del facciale, lesione dei canali semicircolari, apertura dei seni venosi situati in posizione anomala, ecc.).

La pertinacia che presentano le suppurazioni dell' orecchio medio anche dopo un intervento endotimpanico, più che ad insuccesso operatorio, è imputabile alla struttura dell' osso, che senza dubbio ha peculiare influenza nell' evoluzione delle suppurazioni auricolari. Oltre a doversi tener conto delle variazioni che subisce normalmente lo sviluppo della cassa timpanica al pari della mastoide, dobbiamo considerare per la cassa tre varietà fondamentali di struttura : diploica, pneumatica e sclerotica.

Sezionando temporali di individui affetti da lesioni dell' orecchio medio, sia di bambini come di adulti, non è raro spiegarci la persistenza delle suppurazioni della cassa con la costituzione spongiosa delle sue pareti, quel tipo cioè che Cheatle ha chiamato infantile. Ora ricordando che alla formazione della mastoide e della cassa concorrono la porzione petrosa, la squammosa e l'anulo timpanico e che ha prevalenza la petrosa, ci spiegheremo la presenza più frequente dell' osso spongioso nella parete posteriore della cassa e nella porzione posteriore dell' epitimpano, per la ragione che l' ala squammosa mastoidea, contenendo meno diploe di tutte le ossa del cranio, nel punto in cui si adatta alla porzione petrosa si riduce alle due corticali esterna ed interna accollate tra loro. La divisione delle due porzioni petrosa e squammosa lungo la fessura petro-squammosa talvolta poi permane. Avremo in questi casi nell' antro mastoideo propriamente detto la presenza della lamina detta di Schwartze-Eysell, mentre nella cassa si troverà una cresta lungo l' epitimpano, detta di Rouvière, che fu studiata anche da Canèpele.

Nel tipo a struttura pneumatica la pneumatizzazione è talora notevole nell' ala squammosa, tanto vero che si è anche riscontrata nel 7° mese della vita fetale, ed in questi casi si possono trovare delle cellule epitimpaniche, specie della parete esterna dell' attico. Quando poi la corticale endo- ed esocranica è molto sottile, avremo allora sovente a notare che la superficie dell' osso, sia in corrispondenza del tegmen tympani, sia al disopra dell' anulo timpanico, assumerà l'aspetto di una stoffa divorata dai tarli od almeno sarà talmente sottile da essere trasparente e facile ad essere usurata ; ecco perchè in alcuni individui anche adulti il pus si fa strada in alto come nei bambini e si comprende perchè le suppurazioni croniche dell' orecchio in questi casi di eccessiva pneumatizzazione siano ribelli alla cura chirurgica endotimpanica.

L' evoluzione delle otiti purulente può essere pure influenzata dalla struttura compatta o sclerotica dell' osso temporale, quando l' eburneizzazione non sia la conseguenza di una osteosclerosi determinata dall' otorrea e quindi un mezzo di difesa contro le propagazioni del male, ma un fatto fisiologico. È certo che anche quando la suppurazione cronica dell' attico e dell' antro è limitata alla mucosa, se esiste una eburneazione completa fisiologica, non sono sempre favorevoli i risultati dell' *exenteratio cavi tympani*, in quanto che tanto la cassa che l' antro sono più piccoli del normale ed hanno rapporti topografici atipici e dipendenti, per nostra personale opinione (che è in contrasto con quella di Mouret), non da un processo di ossificazione normale, ma invece da una deviazione di esso nel periodo di sviluppo dell' orecchio medio. Si spiegano così tutte le infinite opinioni che è inutile riepilogare sull' origine fetale e post-fetale dell' antro.

La cavità attico-antrale non appartiene alla categoria dei cosidetti organi fissi od essenziali dell' individuo che compaiono a seconda della specie ad un epoca fissa embrionale, come l' orecchio interno, l' occhio, il cuore ecc. L' orecchio medio invece è un organo accessorio, senza periodo fisso di formazione, e come tale deve obbedire a quelli essenziali che lo sopportano e ne subirà tutte le vicende dipendenti da speciali condizioni inerenti all' individuo e non alla specie, donde uno sviluppo o precoce o ritardato o anche arrestato da determinate affezioni ereditarie.

È precisamente nel periodo di sviluppo che la cassa timpanica, come organo accessorio, sarà strettamente legata ai processi normali o patologici dell' osso temporale e potrà quindi, nella stessa guisa che presentare o struttura pneumatica o diploica, subire degli arresti di sviluppo per una precoce eburneazione forse legata a pregresse lesioni ossee di origine rachitica o osteomalacica.

Si è dato poca importanza come fattore etiologico alle deformazioni ossee che avvengono nello sviluppo del temporale a causa delle quali l'anatomia normale dell' orecchio medio e cavità annesse non si può sistematizzare.

Anche orientamenti non patologici nello sviluppo delle ossa determinano condizioni per le quali i processi morbosi assumono decorso e gravità diverse da un individuo all' altro. Così dalla differente inclinazione colla quale l' ala squammosa mastoidea tende a saldarsi con la petrosa consegue che la cavità epitimpanica al di sopra della testa e del corpo dell' incudine sarà di ampiezza variabile, per cui in alcuni adulti si può talora vedere che l'estremo superiore degli ossicini tocca quasi il tetto dell' attico, come nei bambini, mentre normalmente dovrebbe esistere un più o meno largo spazio.

Dagli studi anatomici più recenti è dimostrato che la cavità epitimpanica, che fisiologicamente tende sempre più ad ingrandirsi, molte volte pare che subisca un arresto di sviluppo e con essa di conseguenza l' *aditus* e l' antro. Normalmente la cavità antrale, già a completo sviluppo al momento della nascita, non si accresce di molto in seguito. Talora

aumenta invece di volume in senso verticale tanto da raggiungere una altezza di due centimetri, per cui, stabilendo come norma che il centro di altezza della cellula antrale corrisponde ad un piano orizzontale che passa per la spina *supra meatum*, ne consegue che il fondo dell' antro è molto più basso dell' *aditus* e dell' epitimpano del quale esso è emanazione. In tal guisa nel feto a completo sviluppo il pavimento dell' antro ha minimo dislivello con l' attico. Tal' altra invece col progredire dell' età l' antro subisce un arresto di sviluppo ed una linea orizzontale, tirata a livello del margine inferiore dell' epitimpano, cioè all' angolo superiore posteriore del condotto uditivo esterno, passa o lungo il pavimento dell' antro o al disotto di esso.

Di un altro fattore anatomico importante dobbiamo tener conto e cioè che la parete esterna dell' attico, a somiglianza di quella analoga dell' antro, è costituita dalla porzione squammosa del temporale, la quale può spingersi tanto in basso da ridurre notevolmente la porzione timpanica della cassa e da rendere meno accessibile l' epitimpano. Forse questo avviene in quei casi nei quali il limite inferiore dell' antro sorpassa la sutura petro-squammosa. Interessa eziandio il nostro argomento la pneumatizzazione ossea della parete esterna dell' attico nel cui spessore sono state notate delle piccole cavità fino dal 7° mese di vita intrauterina.

Il maggiore o minore sviluppo dell' epitimpano contribuisce a favorire o no la chirurgia endotimpanica e nei casi in cui l' attico rimane piccolo ed è ristretta la cavità nel senso antero-posteriore può essere difficile e perfino impossibile aggredire l' incudine.

Salvo che nelle otorree croniche complicate da labirintiti, nei casi di lesioni ben limitate dell' adito e dell' antro si credette di aver fatto un passo decisivo nella cura con il vuotamento petro-mastoideo, col quale non solamente si tende a sottrarre il paziente a tutte le complicazioni possibili inerenti ad un processo suppurativo cronico dell' orecchio, ma a salvare l' udito. Quanto alla funzione i risultati di questo atto operativo sono discussi, sostenendo alcuni che con esso non si modifica quel residuo di udito ancora esistente, altri che l' udito migliora, altri infine che esso peggiora.

Già parecchi anni or sono Scheibe aveva consigliato di rispettare la catena degli ossicini nella cosidetta operazione radicale. Anche Panse e Jansen poco dopo la volgarizzazione dell' operazione radicale consigliarono di risparmiare gli ossicini. Lo stesso Stacke aveva raccomandato di non toccare gli ossicini in certi casi e dello stesso parere fu più tardi Siebenmann.

Questo concetto conservativo in una operazione pur tanto demolitrice disorientò i partigiani della chirurgia endotimpanica che mira sovratutto alla avulsione degli ossicini. In conclusione si disse che allora quando la lesione è limitata all' adito ed all' antro, mentre si mantiene buona l' audizione *per aere* e la prova di Gellé è positiva, con l' asportazione degli ossicini si rischia di abbassare il potere acustico, fatto che assume grande importanza se esiste sordità dell' altro orecchio.

Ma è qui appunto che noi vogliamo entrare a discutere l' argomento ponendo la questione in chiari termini nel senso che bisogna non confondere le suppurazioni croniche che hanno loro sede nell' antro con quelle localizzate alla cassa ed all' epitimpano. Nelle prime essendo meno compromessa la funzionalità acustica, è forse ammissibile la conservazione degli ossicini ; nella seconda è assurdo sperare l' estinzione del processo suppurativo colla permanenza della catena che è sempre lesa nei suoi componenti.

Noi abbiamo sottoposto all' esame istologico numerosi ossicini e ci pare prezzo dell' opera riportarne i risultati.

Furono oggetto delle nostre osservazioni 11 casi, in 7 dei quali fu esaminato soltanto il martello, in 2 altri soltanto l' incudine e in 2 l' incudine e il martello.

Gli ossicini subito dopo asportati furono immersi in un decalcificante contenente il 5 % di formalina ed il 4 % di acido nitrico. Con tale liquido, per lunga esperienza fatta sull' intera capsula labirintica di animali diversi e dell' uomo, si raggiunge un' eccellente fissazione degli elementi. La decalcificazione completa richiede molto tempo (un mese ed oltre), presentandosi gli ossicini poco permeabili. Ottenutasi la decalcificazione, previo lavaggio di 24 ore in acqua corrente, furono colorati in massa con carminio (boracico o alluminoso) e qualcuno con ematossilina ed eosina, quando dalla colorazione in massa non si era ottenuto buon risultato ; e di ciò abbiamo poi compresa la ragione, come più avanti diremo.

Non fu adottata come decalcificante la fluoroglucina, perchè questa, mentre dà ottimi risultati nello studio delle ossa normali, non risponde ugualmente in quello delle ossa malate. Dopo colorazione gli ossicini furono inclusi in paraffina per ottenere tagli molto sottili.

In generale si osservò che la colorazione raramente riuscì completa e che per di più i preparati, scarsamente o punto colorati, nemmeno ricolorati con ematossilina, riuscirono migliori. Questo fatto crediamo dovuto all' avanzata necrosi del tessuto sottoposto ad esame.

Premesso ciò, va notato che quasi nessun ossicino ci dette preparati nei quali fosse possibile osservare la struttura normale dell' osso e che dalle lievi alle profonde alterazioni era una graduale successione di quasi tutte le fasi di iponutrizione, di atrofia, di rarefazione, di distruzione e necrosi della sostanza ossea, salvo un caso in cui si trovò un processo tubercolare del martello consistente in un voluminoso granuloma con cellule giganti, il quale sovrastava alla testa dell' osso ed era più voluminoso di essa.

Fra le alterazioni lievi abbiamo notato uno speciale aspetto che diremmo schistoso della sostanza ossea, come se il taglio risultasse da diversi pezzi accollati di una lastra rotta (Tav. I, figs. 2, 5, 6 ; Tav. II, fig. 9). Questo aspetto, reso evidente dall' ineguaglianza di tinta assunta dal taglio colorato, è dovuto al fatto che le lamelle di una trabecola ossea

vanno in senso obliquo o perpendicolare rispetto a quelle della lamella adiacente. Come si sa, questo aspetto è dovuto (Morpurgo) a pregresse riossificazioni avvenute in tempi diversi.

Spesso i vasi sanguigni anche in ossicini non ancora caduti in necrosi si presentano occlusi come da un tappo di ematoidina, nel quale osservansi zolle di emosiderina in formazione. Tale aspetto è perfettamente distinguibile da quello che presentano altri vasi normali, in cui elementi normali del sangue occupano tutto il lume. Inoltre abbiamo osservato zone di rarefazione abbastanza ampie nella testa del martello, o lungo il manico, o nel centro dell' incudine. Questa rarefazione procedeva dagli spazi midollari, o dalle guaine connettivali perivasali entro i canalini haversiani ; la sostanza midollare era ridotta al solo stroma a larghe maglie senza contenuto (Tav. I, fig. 3); il connettivo era atrofico e ridotto a stroma tenuissimo, con rari elementi cellulari.

I contorni degli ossicini in questo primo grado di alterazione erano non regolari, ma con infossamenti succedentisi l' uno all' altro, e il periostio in questi infossamenti o era lievemente proliferato oppure retratto, sottile ed atrofico (Tav. I, fig. 1ª).

Fra le alterazioni più avanzate e gravi abbiamo notato : una rarefazione molto più accentuata e non di una parte, ma di tutto l' ossicino, le cui trabecole erano distaccate fra loro ; soltanto alcune di esse presentavano vestigia di struttura lamellare e di corpuscoli ossei visibili (Tav. I, fig. 4). Questo aspetto può essere assomigliato a quello suddetto di una lastra rotta, i cui pezzi però siano stati un poco allontanati fra loro (Tav. I, fig. 8). Spesso in mezzo a questo grosso detrito lamellare era dato ancora osservare l' apertura di qualche residuale vaso sanguigno a pareti indecise, perchè prive di nuclei colorati e col lume perfettamente occluso da residui amorfi di coagulo sanguigno rimasti come *caput mortuum*, senza presentare le varie fasi di organizzazione trombotica. Infine in qualche ossicino abbiamo notato che accanto al residuale tessuto osseo caduto in necrosi era del tessuto fibroso che in parte lo sostituiva (Tav. I, fig. 7ª).

Osserviamo incidentalmente che nell' orecchio medio in genere e nella catena degli ossicini in ispecie i processi di organizzazione ed i processi regressivi si svolgono con molta lentezza e spesso si arrestano in una fase intermedia, cui segue uno stato speciale che diremmo di mummificazione. A questo stato secondo noi è dovuta la descritta occlusione dei vasi non per un trombo ordinario, ma per mezzo di un coagulo in cui è dato solo distinguere l' aspetto ematoidinico e raramente quello emosiderinico, che, com' è noto, si ha in presenza dell' ossigeno, che probabilmente nei casi in esame faceva difetto. È ragionevole indurre che l' arresto del circolo per tal modo determinato sia stata la causa precipua della cattiva nutrizione e poi della necrosi del tessuto osseo.

Ad uno stadio più avanzato, quando nessuna colorazione dei tagli è riuscita, abbiamo osservato un assieme di pezzetti staccati, che, mentre disegnavano la sagoma dell' ossicino, nessuna struttura più presentavano, risultando di sostanza amorfa uniformemente ma incompletamente tinta

dalla protratta colorazione con ematossilina. Fra questo grosso detrito ancora era dato riconoscere qualche forma di canalino haversiano con relitti del vaso decorrentevi.

La struttura lamellare era perfettamente scomparsa e nessuna vestigia di corpuscoli ossei era più dimostrabile.

Le descritte alterazioni, colpite a diverso grado così nel martello come nell' incudine, furono riscontrate in 9 degli 11 casi esaminati. In uno solo di questi abbiamo notato nella fase attiva un processo di riorganizzazione della sostanza ossea, appena accennato nel martello e molto manifesto nell' incudine, e in un altro, che è quello colpito da tubercolosi, i processi di riossificazione del martello sono appena iniziati.

Nel martello del 1° di questi due casi soltanto verso un margine si scorgeva la presenza di osteoclasti in fila di fronte a tessuto osteoide. Nell' incudine invece si notava fra il periostio in proliferazione e l' osso sottostante un versamento ematico a forma di sottile striscia che in un punto si affondava in una escavazione ossea relativamente ampia, entro la quale, nel più chiaro aspetto, erano visibili le varie fasi del processo di organizzazione trombotica (Tav. II, fig. 11). Ebbene in corrispondenza della sottile striscia di versamento ematico, l' osso a contatto si presentava fortemente tinto e ricco di osteoblasti, sulle pareti della stessa escavazione erano molti osteofiti allineati come digitazioni sorgenti dall' osso e penetranti nel trombo ematico. Questa riproduzione del tessuto osseo in immediato contatto col sangue effuso sotto il periostio o organizzantesi nel trombo ora descritto era del tutto evidente e costituisce, secondo noi, uno speciale processo di reintegrazione di un osso malato che merita di essere ulteriormente studiato.

Questo che nella serie degli 11 casi esaminati costituisce, per il momento in cui il processo fu sorpreso, una eccezione alla regola della graduale denutrizione, atrofia e necrosi degli ossicini, molto probabilmente non rappresenta che una fase transitoria della malattia degli ossicini. Ma siccome anche in ossicini caduti in atrofia e necrosi abbiamo trovato indizii di pregressa riossificazione, così è lecito indurre che anche nel caso ora descritto questa reintegrazione non fosse definitiva e potesse anche essere seguita da fasi regressive.

Abbiamo infine il caso suaccennato di un martello colpito da processo tubercolare (Tav. II, fig. 10). Si tratta, come abbiamo detto, di un granuloma tubercolare di volume relativamente enorme che supera per due volte quello della testa e sorge dall' osso come una produzione connettivale fibrosa, la quale però subito si rigonfia per costituire un bottone carnoso nel cui centro sono lacune filamentose contornate da grosse cellule giganti ed altri nidi di cellule giganti contornate da connettivo. Alla periferia questo granuloma presenta una vasta infiltrazione ematica ed elementi cellulari caduchi con nuclei in picnosi. Orbene anche in questo caso la sostanza ossea del sottostante martello ha struttura simile a quella normale per disposizione delle trabecole, delle lamelle e dei corpuscoli ossei ; ma vi si notano due alterazioni di cui una consiste nella debolissima colorazione

dei corpuscoli, che spesso appariscono vuoti, l' altra nella rarefazione delle trabecole per penetrazione di propaggini connettivali.

Queste propaggini connettivali solo in punti limitati hanno i noti caratteri del connettivo osteogeno, per la presenza di rari osteoblasti nella linea di confine con la sostanza ossea, e tutta la sostanza ossea del martello è colpita da denutrizione ed atrofia che s'avvicinano quasi alla necrosi. Anche ammettendo in questo caso una graduale sostituzione dell' osso nuovo al malato, dobbiamo osservare che il connettivo osteogeno è diretta promanazione di un granuloma tubercolare e che perciò dovrà fatalmente andar soggetto alle fasi progressive del processo tubercolare.

Possiamo quindi concludere, dal punto di vista anatomo-patologico, che qualsiasi processo flogistico degli ossicini, malgrado simili tentativi di reintegrazione, finisce in definitiva con la necrosi totale dell' osso.

Tutti gli autori che si sono dati la cura di conservare nella cura radicale la catena degli ossicini convengono che per ottenere i migliori successi acustici è necessario: 1° — che la catena rimanga integra nella sua continuità; 2° — che la membrana del timpano sia il più possibile conservata per la sua tendenza a riformarsi ed a sostituire perfino la catena, quando va ad accollarsi alla staffa; 3° — che sia conservata una parte del cercine timpanico e della parete esterna dell' attico, come punto di appoggio e sorgente nutritizia alla membrana (Heath, Bondy, Schwenemann, Botey, Mahu).

Stacke crede al successo della radicale colla conservazione degli ossicini in quei casi nei quali vi sia assoluta immunità della cassa propriamente detta, allorchè insomma si tratti di colesteatoma dell' attico, dell' antro e dell' adito. Egli si assicura di questo mediante un catetere introdotto nella tromba ed attraverso il quale una corrente d'aria non giunga nell' attico. Stacke sostiene in complesso che con la epidermizzazione dell' attico, adito ed antro, essendo la cassa immune e la membrana integra, si ovvia alle reinfezioni tubo-timpaniche e si giunge alla guarigione nel 50 per cento dei casi, d' accordo in questo con Bondy, Bárány, Urbantschitsch ed altri. Stacke nei suoi operati potè conservare la funzione uditiva del tutto normale o di poco abbassata. Si credette perciò sempre più autorizzato ad operare radicalmente un orecchio affetto da colesteotoma con buon potere uditivo, anche quando era l' unico che udisse, purchè esistessero le condizioni da lui enunziate.

Coscienziosamente Stacke però dice che egli da questa cura radicale conservativa non ricavò miglioramenti notevoli nella funzione acustica, quando questa era già compromessa. Secondo tale autore però ci sarebbe questo vantaggio che, non avendo interrotta la catena, si resta liberi dopo guarita la suppurazione attico-antrale di procedere alla ossiculectomia per via endotimpanica a scopo acustico.

La pregiudiziale che io faccio è: se veramente in quei casi, relativamente rari, in cui la lesione colesteatomatosa è limitata all' antro ed all' *aditus* si possa ammettere che gli ossicini si sieno conservati sani nella

loro struttura, ancorchè l' apparenza otoscopica li faccia credere tali e la funzione sia più o meno buona. Io mi domando poi con quali criteri Stacke sia giunto a raccomandare come logico un intervento conservativo anche allorquando la catena sia interrotta nell' apofisi lunga dell' incudine, lasciando così una cavità timpanica chiusa con residui di osso certamente malato. La teoria conservativa di Stacke va più oltre ; egli raccommanda il rispetto del martello e della membrana eziandio se l' incudine sia stata già espulsa e vi sia però speranza che il timpano si riformi.

Ci si permetta per la ormai lunga esperienza che abbiamo della chirurgia dell' orecchio medio di non mostrarci troppo entusiasti di questo metodo cosidetto radicale conservativo, che mirando unicamente a tutelare la funzione corre rischio di lasciare, secondo noi, dei focolai suppurativi che un giorno o l'altro saranno causa di recidiva.

D' altra parte noi abbiamo numerosi operati di radicale classica nei quali il potere acustico, se non ha migliorato dopo l' intervento, non ha certo subito diminuzione. A questo proposito raccomandiamo di sperimentare, anzichè con i diapason ed altri apparecchi sonori, mediante il microtelefono. Con questo strumento avendo proceduto, prima e dopo l'intervento operativo sulla catena, all' esame a determinata distanza della voce conversata ed afona, si riscontra dopo un certo periodo, nella maggioranza dei casi, un miglioramento e ben di rado una diminuzione nella percezione uditiva. Preferiamo in queste ricerche il microtelefono agli altri audiometri perchè esso più facilmente ci svela i difetti nella percezione della voce che è quello che più importa nella vita di relazione, difetti che alle volte sfuggono all' indagine con altri strumenti di esame, per la ragione che molti individui, mentre ancora sentono i suoni musicali, sono incapaci di percepire la voce.

Nella cura radicale conservativa si comprende che torna a galla come un' ossessione il medesimo quesito che si facevano gli otoiatri favorevoli o contrari all' ossiculectomia, giudicandola ora dannosa ed ora utile dal punto di vista acustico. Lasciando da parte ogni apprezzamento sul meccanismo di trasmissione del suono al labirinto attraverso la catena, sta di fatto che di qualunque teoria fisiologica si sia partigiani, tutti ammettono che la *exenteratio cavi tympani* reca dei miglioramenti positivi della percezione acustica ogni qualvolta restituisca la mobilità alla staffa ; tanto vero che in pazienti da lungo tempo operati di ossiculectomia per suppurazione cronica e guariti con miglioramento della percezione della voce potemmo dimostrare mediante una pressione sulla staffa o indirettamente con la chiusura del condotto o direttamente con la sonda di Lucae la diminuzione e persino l'abolizione della percezione ossea del diapason.

Ci siamo indotti a discutere queste vessate questioni in quanto che la chirurgia otoiatrica, come tutti gli altri rami della chirurgia, in questi ultimi anni ci è sembrata troppo aggressiva e poco preoccupata dell'importanza dell' operazione radicale non sempre perfettamente giustificata. Essa costituisce invero un intervento non scevro da pericoli gravi e che sottrae

spesso per lungo tempo al lavoro individui che con traumi chirurgici molto minori potrebbero guarire. Nè agli inconvenienti ovvia la cosidetta radicale conservativa, la quale, pur non accorciando il tempo di guarigione, non dá, secondo crediamo di aver dimostrato, risultati funzionali superiori alla chirurgia endotimpanica.

Come conseguenza di quanto abbiamo detto emerge che non in tutte le suppurazioni croniche è leso l'antro e l'*aditus*, per cui il far sacrificio di osso sano secondo noi non è giusto. Una lunga esperienza del resto ci ha dimostrato che moltissime otorree croniche guariscono stabilmente con la ossiculectomia accompagnata o no dalla demolizione della parete esterna dell'attico ed abbiamo visto anche che la funzione acustica o non è compromessa da questo intervento o è migliorata.

Lo studio anatomo-patologico sistematico degli ossicini asportati o per via endotimpanica o durante l'operazione radicale ci ha rivelato costantemente che nei processi suppurativi cronici gli elementi della catena sono sempre malati anche allorquando macroscopicamente sembrano normali. A questo proposito ci sia lecito affermare che la operazione radicale classica è da preferirsi alla cosidetta radicale conservativa in quanto questa, mentre è un trauma chirurgico sempre grave e laborioso, lascia in sito ossicini di dubbia integrità e messi, per il dato e fatto dell' intervento, in condizioni di minorata resistenza ; quindi noi crediamo che tale decantato intervento non possa sostituire nella indicazione quella veramente radicale e non costituisce un passo avanti nella chirurgia conservativa da renderlo preferibile alla più semplice e meno demolitrice chirurgia endotimpanica.

Infine a noi sembra più rigoroso e logico metodo chirurgico incominciare il trattamento operativo delle suppurazioni auricolari croniche da quello più lieve endotimpanico per giungere in caso d'insuccesso alla radicale, piuttosto che procedere in senso inverso.

Sarebbe vano contestare i reali definitivi risultati raggiunti dalla medicina operatoria intratimpanica nella cura delle suppurazioni croniche limitate alla cassa ed all'epitimpano che prima, nelle strette di una terapia medica, di rado giungevano a guarigione definitiva e per lo più, se non davano luogo a peggioramenti, non portavano che a risultati dubbi.

Questa grande rivoluzione nella cura delle suppurazioni delle otiti purulente croniche è opera sopratutto del Sexton e Ludewig. Si domandarono questi autori perchè non ammettere come metodo radicale di cura la escissione della membrana e degli ossicini quando è noto che, laddove la suppurazione distrusse la base della staffa, avviene qualche volta una guarigione spontanea, pur conservandosi un udito sufficientemente buono?

Ma passato il periodo dei facili entusiasmi, si dovette pure ammettere che non sempre l'andamento clinico era così lusinghiero come si sarebbe lasciato sperare agli infermi da un intervento per la via intratimpanica. Se ne vollero allora studiare le cause, giungendo alla conclusione che erano assai più numerosi che non si pensasse i fattori della mancata guarigione, escluso, bene inteso, qualunque errore di tecnica operativa.

Postisi su questa via di ricerche si vide che a far mancare la guarigione di queste otorree croniche, sottoposte alla escissione della membrana, degli ossicini, della parete esterna dell' attico, delle briglie ed aderenze cicatriziali, delle granulazioni e delle fungosità dell' epitimpano ecc. ecc., per la via del condotto uditivo, avevano concorso ora la natura del microrganismo specifico che aveva determinato la suppurazione (tubercolosi, sifilide, ozena), ora la speciale discrasia del paziente (rachitismo, osteomalacia, diabete, gotta, arteriosclerosi, fosfaturia, alterazioni delle ghiandole endocrine ecc.), ora la conformazione anatomica anormale delle cavità annesse del timpano, ora infine una deficiente funzionalità della tromba di Eustachio dipendente da speciali alterazioni delle prime vie respiratorie.

L'esperienza clinica giunse però a stabilire che alcuni di questi ostacoli alla guarigione delle suppurazioni croniche potevano essere riconosciuti prima di sottoporre il paziente all' operazione per la via naturale del condotto uditivo. Basterà ricordare le otorree croniche nei tubercolosi e negli ozenatosi nei quali, dato che il caso richiede un atto chirurgico, ricorreremo senz' altro all' ampia apertura dell' antro, dell' adito e dell'epitimpano e non ci limiteremo mai alla semplice *exenteratio cavi tympani.*

In tali casi la diagnosi è relativamente facile. Per la tubercolosi: l'anamnesi e l'esame generale ci riveleranno una predisposizione ereditaria, una speciale costituzione individuale, la presenza di altre alterazioni in atto o pregresse. L'otoscopio ci mostrerà poi un abbondante scolo purulento fetido e sanguinante con quasi assenza di dolore e contemporaneamente vasta e precoce distruzione ossea dell'orecchio con paresi e paralisi del facciale e grave diminuzione dell' udito.

Avrà inoltre valore per la diagnosi la rapida distruzione degli ossicini, la facile riproduzione delle fungosità appena tolte e la precoce manifestazione del processo di carie rilevabile con l'applicazione locale di una striscia di garza bagnata in una soluzione di glicerina e dermatolo. Questa si annerisce perchè nei processi di carie il dermatolo (sottogallato di bismuto) acquista colore nero per lo sviluppo di acido solfidrico. Mancando parecchi di questi elementi clinici ad ammettere un' otite tubercolare a prima indagine e l'esame batteriologico del pus, come per solito avviene, essendo negativo anche per la ricerca dei granuli acido-resistenti, avremo ancora altri mezzi onde stabilire con sicurezza la natura della lesione e cioè la reazione organica della tubercolina (oftalmo- e cutireazione). Nell' incertezza ancora di questi segni non riconosciuti patognomonici, l'inoculazione peritoneale del ganglio linfatico premastoideo nei conigli sarà un procedimento infallibile (De Rossi).

Ugualmente ci asterremo dal compiere l'*exenteratio cavi tympani* attraverso la via naturale del condotto negli ozenatosi con otorrea, quando questa sia diretta conseguenza del processo morboso nasale e contenga il *bacillus uucosus ozenae.*

In precedenti miei lavori ho dimostrato che nei casi tipici di ozena il

processo suppurativo dell' orecchio medio è limitato allo strato mucoso e non invade mai le pareti ossee della cassa e la catena degli ossicini, salvo nei casi di vecchia data nei quali, come per i turbinati, si osserverebbe negli ossicini una rarefazione delle lamelle ossee periferiche, con riassorbimento osseo canalicolare. Orbene la suppurazione cronica degli ozenatosi per la pertinace resistenza del *bacillus mucosus* ben di rado si estingue con la semplice *exenteratio cavi tympani*, come purtroppo alle volte nemmeno si domina il processo con l'operazione radicale classica, se non si riesce a chiudere artificialmente la tromba.

Molto più difficile sarà prevedere gli insuccessi imputabili ad una reinfezione tubaria, sia che il processo suppurativo persista, nonostante la *exenteratio cavi tympani*, per la via del condotto, sia che sorgano delle vere recidive anche dopo la cutizzazione della cassa.

Nei primi periodi della chirurgia intratimpanica molti insuccessi operativi che non si sapevano spiegare erano da mettersi a carico della tuba di Eustachio, tramite fisiologico e patologico di comunicazione fra le prime vie respiratorie e l'orecchio medio, zona per lo più inesplorata prima di accingersi ai vari interventi operativi. Per lo più le affezioni della tromba non si riconoscono che al momento del cateterismo o quando all' esame otoscopico si riscontra una esagerata retrazione timpanica, dipendente da stenosi tubaria di origine naso-faringea.

In due successive mie pubblicazioni del 1904 e 1908 ho appunto trattato della necessità della obliterazione definitiva della tromba di Eustachio negli interventi auricolari allo scopo di guarire radicalmente l'otite media purulenta cronica, specie quando la persistenza della suppurazione non sia legata ad anomalie anatomiche del temporale, compresa la parte intrapetrosa della tromba, ma a lesioni latero-faringee che ostacolano l'azione fisiologica della tromba.

Qualsiasi alterazione del cavo naso-faringeo ed ispecie delle pareti laterali e del palato, compromettendo l'azione dei muscoli *elevator-palati*, *dilatator tubae* e *tensor-palati*, provocherà la chiusura dello sbocco faringeo della tromba, che in condizioni fisiologiche è sempre pervio, nonostante il parere contrario di alcuni. Trasformatosi in cavità chiusa il canale tubarico, sarà aumentata la virulenza dei microrganismi che per caso vi siano giunti.

De Rossi, avendo compiuto ricerche sulla flora batterica della tromba d' individui sani, riuscì a dimostrare sperimentalmente che la sua secrezione normale non contiene microrganismi patogeni. È perciò evidente che la penetrazione microbica è dovuta alla deficiente mobilità del padiglione tubario dipendente da lesioni del cavo naso-faringeo. La pervietà della tromba è così necessaria da far dire a Soyer Bryant che le flogosi dell' orecchio medio, eccetto quelle di origine ematica, di rado dánno luogo a complicazioni mastoidee o endocraniche quando la tromba è libera.

Sapendo che la tuba si divide in due sezioni, di cui l'ossea è un fornice accessorio della cassa e la fibro-cartilaginea un diverticolo della faringe,

SPIEGAZIONE DELLE TAVOLE

Tavola I

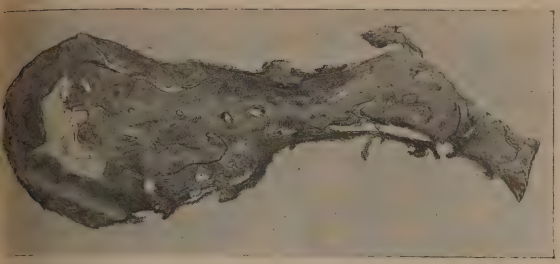


Fig. 1. Martello con alterazioni lievi. Cavità midollare ıpia. Alcuni vasi occlusi. Bordi dell' ossicino irregolari.

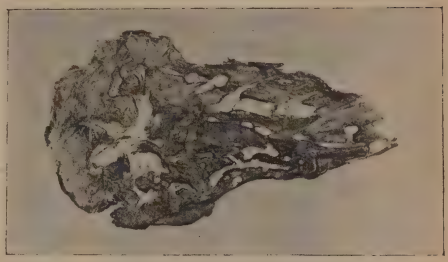


Fig. 2. Martello a contorni irregolari con periostio alterato e sostanza ossea di aspetto schistoso.

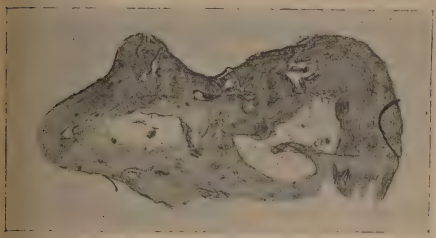


Fig. 3. Martello non uniformemente colorato nei tagli, con tessuto midollare molto rarefatto.



Fig. 4. Martello con alterazioni di grado più accentuato. Tessuto osseo pochissimo colorato nei tagli, con corpuscoli ossei appena visibili.

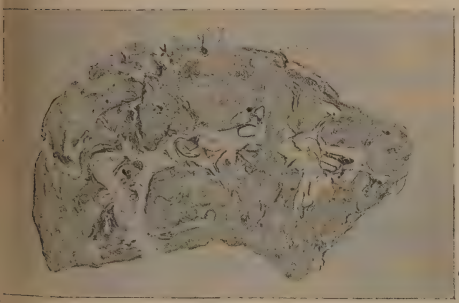


Fig. 5. Incudine in necrosi. Aspetto di ıstra rotta.

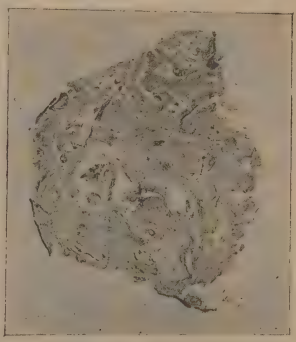


Fig. 6. Incudine. Trabecole e corpuscoli ossei appena visibili. Aspetto di lastra rotta, i cui pezzi sieno scostati fra loro.



Fig. 7. Martello con residui ossei necrotici, sostituito in parte da tessuto fibroso.

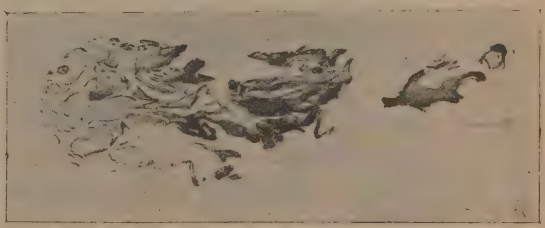


Fig. 8. Martello costituito da un assieme di pezzetti necrotici senza struttura, disposti secondo la sagoma del martello.

aggiungerò che l'infezione della cassa dal cavo naso-faringeo avviene per il lume tubario e sovratutto per la parte superiore ossea detta da De Rossi ' spazio di sicurezza ', che rimane costantemente più o meno aperto anche nei gravi casi d'infiltrazione della mucosa di rivestimento. Che ciò sia vero è dimostrato dal fatto che pure nelle otorree di vecchia data con compartecipazione della tromba i liquidi iniettati nella cassa raggiungono più o meno tardi la gola. Sopra questa costatazione basavasi il metodo di cura da me adottato prima del periodo chirurgico che consisteva appunto nella introduzione per la via della cassa di liquidi fortemente caustici, facendo inclinare la testa del paziente dalla parte opposta all' orecchio malato, mentre si cercava d'impedire la caduta dei liquidi nella faringe per mezzo di un catetere ad estremo chiuso e fortemente occludente l'orificio faringeo della tuba.

Fondandomi su queste nozioni anatomo-fisiologiche fin dal 1904, allo scopo di far cessare le otorree a ripetizione non dipendenti da lesioni dirette dell' orecchio medio e delle cavità annesse, ma da materiali settici che vi affluivano dalla tromba nella posizione di riposo, pensai di tentare il metodo operativo proposto da Segura.

Ne fui però trattenuto sia dalla difficoltà pratica d'introdurre un' ansa di platino nella tromba e renderla incandescente, sia dai dolori fierissimi e dai fenomeni riflessi che ne seguivano, sia dalle suppurazioni acute dell' orecchio e cavità accessorie che si possono presentare in seguito a questo intervento. Credetti piuttosto migliore consiglio troncare la via di passaggio dalla cassa timpanica alla volta naso-faringea, ostruendo mediante cauterizzazione galvanica l'orifizio timpanico della tuba.

In una seconda comunicazione del 1909, ribadii il concetto che era preferibile chiudere questo orifizio timpanico in base alle stesse nozioni anatomo-patologiche che spiegano all' evidenza essere la porzione ossea della tromba più compromessa nelle affezioni primitive della cassa, mentre la fibro-cartilaginea è più colpita nelle affezioni del cavo naso-faringeo.

Nè si obbietti che, nonostante la chiusura dell' orifizio timpanico della tromba, l'infezione può, per il tessuto sottomucoso di rivestimento della tuba, invadere la cassa arrestandosi questo presso l'istmo ; l'infezione potrebbe ancora giungere nell' orecchio medio per la circolazione linfatica, la quale però è povera nella porzione della tromba fibro-cartilaginea, e ciò dá il massimo valore alla nostra preferenza di chiudere l'orifizio timpanico anzichè quello faringeo.

Non è stata poca la mia sorpresa scorrendo i resoconti della XX[a]. Riunione della Società tedesca di Otologia tenuta ad Hannover dal 22 al 24 maggio 1912, di leggere una comunicazione di Laurowitsch di Jena in cui fra i tanti mezzi di chiusura dell' orifizio timpanico della tromba mediante plastiche non si fa parola dei precedenti metodi da me consigliati. Alla discussione partecipò Urbantschitsch ricordando un metodo proposto da suo figlio, consistente nell' introduzione di una sonda intrisa in una soluzione di nitrato d'argento con cui si fanno frizioni energiche delle pareti tubarie. Egli crede che in tal modo si possano guarire le

TAVOLA II

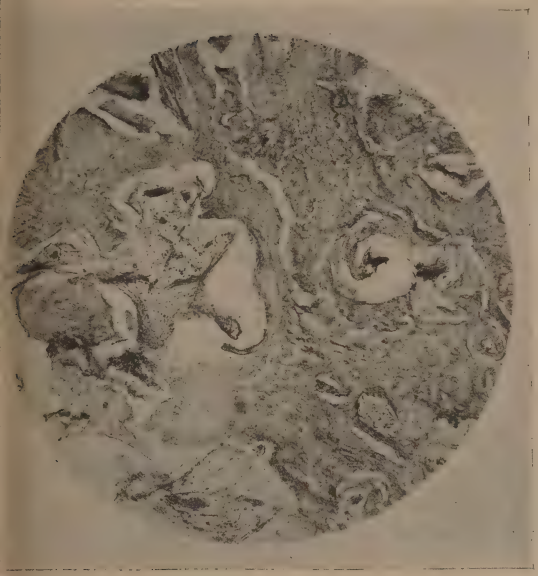


FIG. 9. Aspetto schistoso di un' incudine veduta a :te ingrandimento. Non sono più visibili i corpuscoli sei.



FIG. 11. Incudine con ematoma sottoperiostale e processo di riossificazione.



FIG. 10. Granuloma tubercolare con cellule giganti sormontante il martello.

suppurazioni tubarie, evitando una vera chiusura della tromba di Eustachio, che, secondo lui, danneggerebbe la funzione uditiva.

Concludendo riporteremo le indicazioni nelle quali è giustificato, a nostro avviso, l'intervento operativo per la via del condotto.

1°. Nelle suppurazioni croniche riacutizzate, allorchè si possa fondatamente credere che i fenomeni reattivi dipendano da ritenzione di pus nell' attico.

2°. Quando la suppurazione persistente della cassa non è in rapporto con la tubercolosi o con l'ozena e sia da sperare che con l'asportazione degli ossicini si determini il drenaggio anche di possibili suppurazioni estese all' antro.

3°. Quando sia accertata la carie degli ossicini e della parete esterna dell' attico.

4°. Nelle otorree croniche non dovute a lesioni ossee, ma localizzate ai tessuti molli (otiti medie purulente croniche degli adenoidei, perforazioni della membrana deficienti ed improprie, stenosi tubarie in genere).

5°. In tutte le lesioni cicatriziali successive a suppurazioni pregresse che abbiano determinato disturbi funzionali per retrazione, fatto che si può avverare eziandio nelle suppurazioni in atto.

6°. In tutti quei casi nei quali sia possibile escludere una lesione ossea profonda della mastoide e si possa sperare che, avendo resa libera la cassa dai fisiologici ostacoli, con mezzi medici si giunga a modificare i tessuti, compresi i rimedi fisici che oggi ci offre il radium e i suoi derivati.

Non mi dilungo a parlare della tecnica in quanto essa è delle più semplici e rimane tal quale la insegnarono i primi fautori della chirurgia intratimpanica. Insisto nella necessità di chiudere l'ostio timpanico della tuba secondo il mio metodo del quale parlai diffusamente in precedenza.

Così pure raccomando ai colleghi di voler provare nella demolizione della parete esterna dell' attico gli scalpelli a taglio retrogrado già da noi raccomandati fin dal 1908. Io credo questi strumenti preferibili anche alle pinze mordenti in quanto il taglio con essi riesce netto e si evita il pericolo di ledere la parete interna della cassa. Le pinze, secondo alcuni autori, hanno l'inconveniente di produrre lesioni ossee a distanza per chiusura dei canali Haversiani, compromettendo la nutrizione di una zona più o meno estesa di osso.

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DISCUSSION

The PRESIDENT. Professor Ferreri has alluded to a report of mine on the question of ossiculectomy in chronic middle-ear suppuration, published many years ago. I wish to say that I have abandoned the operation for at least ten years and perform the radical operation.

Dr. DENCH (New York) said that he now never did an ossiculectomy for chronic suppuration. Undoubtedly in some of these cases the simple operation was sufficient, and he had cases of this kind in his own practice. The trouble was that he could never tell in what cases this operation would suffice, and that a second operation might be necessary.

He advised the radical operation in all cases with the application of the primary skin-graft, and in his experience the hearing was only very rarely impaired as the result of the operation—in cases when there was no labyrinthine involvement.

In many of his cases the hearing has greatly improved as the result of the operation.

Professor MYGIND (Copenhagen). In many cases the hearing power of a patient with chronic suppuration of the middle ear is at its very

worst just before the operation is performed, because there is, besides, an acute inflammation present which has caused symptoms forming the indication for the operation (headache, giddiness, &c.). If we compare the hearing power after the operation with that just before the operation, we often form a too good opinion of the effect of the operation on the hearing power. It is, I believe, necessary for a surgeon to be optimistic, but I think, on the other hand, that we should not be too optimistic as to the improvement of the hearing power after the radical operation, and that our indications for the operation, therefore, should not be too wide. Besides, every one of us has seen a series of cases where the hearing power has been totally destroyed through a post-operative labyrinthitis.

Dr. JANSEN (Berlin) empfiehlt die Radikaloperation mit Erhaltung von Trommelfell und Gehörknöchelchen als ein wertvolles Mittel zur Erhaltung und Wiederkehr des Gehörs und zum Abschluss der Paukenhöhle. Er kann seit 21 Jahren derartige Fälle mit vorzüglichem Gehör verfolgen und empfiehlt in geeigneten Fällen das Verfahren.

Mr. C. E. WEST (London). Ossiculectomy rarely results in permanent cure of chronic suppuration except such as would recover without operation. Caution is necessary in estimates of improvement of hearing after the radical operation. The speaker agreed with Professor Mygind as to the depression of hearing which frequently precedes the performance of a radical operation. Especially in young children were hearing results apt to be disappointing.

In one class of case only is the conservative operation of value, that of limited suppuration in the attic and antrum with preservation of the membrane and ossicles and with really good hearing. Here the speaker was convinced that it was worth while to modify the radical operation by preserving the ossicles and membrane while treating the rest of the wound on the lines of a typical operation.

Professor ALEXANDER (Vienna) recommends the typical radical operation for all cases of unilateral disease and for all cases of bilateral suppuration, if we have to operate on the deafer side.

In cases in which the operation is necessary on the better hearing side we can hope for good results by the so-called incomplete or conservative radical operation, supposing the middle-ear suppuration is limited towards the upper region of the middle-ear spaces and the meso- and hypotympanum are free and normal.

Mr. G. J. JENKINS (London) believes that many cases of chronic suppuration of the middle-ear tract can be cured by removal of the disease in the outer wall of the antrum and attic without removal of the ossicles.

The obvious suggestion is that in all cases of chronic suppuration it is

important to know the condition of the ossicles. The state of the ossicles may be arrived at clinically in some cases. The ossicular chain may be on sight obviously imperfect.

The ossicles are probably in good condition if the hearing is extremely good—say twenty-four feet for the whisper. Also after thoroughly cleansing the middle-ear tract, if there is a marked improvement on inflation and more especially if there is further improvement with repeated inflations.

Lastly, let the ear be thoroughly cleansed by syringing, &c., and in two hours time the hearing be tested and once more syringed with lotion. If the hearing is improved after the second syringing then possibly the incudo-stapedial joint is imperfect, as it is in these cases that ' artificial drum ' improves the hearing. If an ' artificial drum ' improves the hearing then probably the incudo-stapedial joint is gone.

If clinical evidence is in favour of an intact ossicular chain he proceeds as in the Schwartze operation, but removes the outer attic wall more extensively to get rid of all the disease and at same time to get a better view of the ossicles.

This modified Schwartze operation gives much satisfaction in many cases, and should be tried in those cases in which there is doubt as to the condition of the ossicles.

SECTION XVI

OTOLOGY

INDEPENDENT PAPER

DE QUELQUES REMARQUES SUR LES PANSEMENTS DES ÉVIDÉS

Par M. le Dr. E. V. SEGURA, Professeur d'Oto-Rhino-Laryngologie à la Faculté de Médecine de Buenos-Ayres, Chef du Service Oto-Rhino-Laryngologique à l'Hôpital Saint-Roch.

L'évidement pétro-mastoïdien, communément appelé opération radicale par les spécialistes, est une de ces opérations dont la technique a été souvent modifiée. Même à l'heure actuelle où cette intervention est devenue de pratique courante, l'accord n'est pas encore fait sur la meilleure manière d'aborder l'aditus, afin d'assurer une large communication de l'antre avec la caisse ; les uns utilisent la technique de Stacke, d'autres recourent au procédé de Wolff, mais la plupart adoptent la méthode préconisée par Schwartze : ils commencent par ouvrir l'antre en trépanant l'apophyse au lieu d'élection, puis se portent vers la caisse en supprimant la paroi du conduit. Je ne m'arrêterai pas à décrire dans leur détail ces différents procédés ; leurs particularités sont trop connues ainsi d'ailleurs que les indications générales et les contre-indications de l'intervention radicale.

La valeur de cette opération est en effet admise par tous. Il est actuellement reconnu, qu'en présence d'une otite chronique suppurée, le traitement doit d'abord consister à recourir aux pansements méthodiques, mais que dans les cas où l'usage de ceux-ci n'est suivi d'aucune amélioration et à plus forte raison s'il existe des symptômes inquiétants c'est à l'évidement pétro-mastoïdien qu'il faut s'adresser ; lui seul peut amener la guérison.

L'opération terminée, une autre tâche s'impose, non moins importante pour la guérison du malade et exigeant de la part du chirurgien une grande patience et une extrême minutie : je veux parler des pansements consécutifs qui ont pour but d'assurer *une épidermisation complète de la cavité artificielle creusée dans le rocher en conservant intégralement la forme primitive de cette cavité.*

Les nombreuses discussions auxquelles se livrent encore à l'heure actuelle les otologistes, les divergences absolues qui séparent les plus

notables d'entre eux, montrent que la question n'est pas encore résolue ou plutôt qu'elle ne comporte pas une solution univoque.

Comme dans tous les actes chirurgicaux dans lesquels la guérison ne doit se faire que par deuxième intention ou plus exactement d'une manière tardive, le traitement post-opératoire joue un rôle des plus importants, dans la réparation des grands délabrements causés par l'otite chronique ou par l'acte opératoire destiné à la combattre.

Cette formule est si vraie dans l'évidement pétro-mastoïdien que je n'hésite pas à poser les deux principes suivants :

1° *De mauvais pansements compromettent le résultat d'un bon acte opératoire.*

2° *Des pansements bien faits peuvent permettre d'obtenir un bon résultat, même quand l'acte opératoire fut défectueux ou incomplet.*

Je n'hésite même pas à être plus catégorique : il suffit qu'un pansement ne soit pas fait avec les soins nécessaires ou qu'on ait laissé trop d'intervalle entre deux pansements pour s'exposer à perdre le bénéfice de quelques semaines de travail et retarder d'autant la date de la guérison.

Les pansements doivent être faits en observant l'asepsie la plus rigoureuse et avec l'attention la plus soutenue : tout coin ou recoin de la cavité opératoire doit être visible et inspecté presque journellement si on ne veut pas s'exposer à des surprises désagréables.

Ils doivent être faits, du commencement à la fin, par une même personne, qui sera autant que possible l'opérateur lui-même ; par ce moyen seulement il peut exister une suite suffisante dans les idées indispensable à la conduite à bonne fin de la guérison et ainsi le panseur est mis en garde d'avance contre les écueils découverts au cours de l'intervention.

Souvent, les différences qui existent entre les statistiques de divers opérateurs ou même dans celles d'un même chirurgien résultent uniquement de la façon dont on a assuré les pansements.

J'ai pu ainsi observer qu'il existait une différence très notable entre les cas opérés à l'hôpital et ceux de ma clientèle particulière. Les premiers ont été pansés alternativement par moi et par mes distingués collaborateurs dont la compétence ne peut être méconnue ; les seconds au contraire ont toujours été pansés par moi : or, de la comparaison de ces deux statistiques, il résulte que le temps employé pour obtenir la guérison des premiers a été le double ou le triple que celui employé dans le second cas. On pourrait objecter que ce retard dans la guérison des malades hospitalisés tient à certains facteurs : alimentation insuffisante, alcoolisme, hygiène défectueuse ; ces causes en réalité ne peuvent être invoquées car si on prend soin de toujours faire panser le même malade par le même panseur, on arrive à obtenir des résultats très satisfaisants : c'est ainsi que dans mon service de l'hôpital Saint-Roch et dans celui de l'hôpital des cliniques que j'ai dirigé ces dernières années, bon nombre de mes opérés, que j'ai confiés aux soins de mes distingués collaborateurs, les docteurs Zambrini et Chiodi, ont été guéris dans un délai inférieur à 45 jours.

Bien traiter un opéré d'évidement pétro-mastoïdien n'est pas chose facile, car il est non seulement nécessaire de posséder des connaissances de spécialité, mais surtout et avant tout, il faut avoir une bonne et longue pratique du traitement de cette variété de malades.

Si les chirurgiens généraux qui pratiquent cette intervention n'ont pas des résultats aussi favorables qu'ils pourraient l'espérer, ce n'est certes pas faute d'opérer brillamment, mais c'est uniquement dû à ce qu'ils n'ont pas une pratique suffisante de la manière d'effectuer les pansements consécutifs. Je connais plus d'un cas qui, opéré par le plus habile de nos chirurgiens généraux, ne fut pas suivi d'un résultat aussi satisfaisant qu'on pouvait l'espérer, c'est-à-dire d'une bonne épidermisation de la cavité opératoire ; celle-ci fut en effet interrompue par la formation de brides cicatricielles, de diverticules qui entretenaient la suppuration. D'ailleurs ces ennuis furent aussi le fait de tous les spécialistes lorsqu'ils furent à leur début dans la carrière. Ce n'est donc qu'à la suite de multiples interventions, et après avoir suivi nombre de malades, que l'on peut arriver à obtenir des résultats toujours satisfaisants.

Si nous considérons les différentes statistiques publiées par le même opérateur, nous pouvons voir que tous ont été en améliorant leurs résultats à mesure qu'ils corrigeaient les petits défauts de leur technique, sans toutefois rien changer à leur méthode opératoire ni à leur manière de panser leurs opérés.

Dans ma pratique personnelle, il en fut de même ; les résultats opératoires ont été en s'améliorant progressivement et à l'heure actuelle je suis arrivé aux beaux résultats dont on peut se rendre compte en lisant la statistique qui accompagne ce travail.

Le terme moyen de durée du traitement post-opératoire est en moyenne de 41 jours ; durée certes assez courte, et qui peut soutenir avec avantage la comparaison avec les différentes statistiques de tous les opérateurs les plus fameux du monde.

Cette comparaison est non seulement flatteuse pour moi, mais aussi pour notre École de Médecine de Buenos-Ayres, c'est d'ailleurs ce qui m'a décidé à faire la présente publication, désirant apporter mon humble tribut à la question de l'opération radicale de l'oreille moyenne, opération qui est, certes, une des plus importantes de toute la chirurgie.

Dans ce travail, il ne faut pas compter trouver de grandes nouveautés, car je désire seulement attirer l'attention sur certains détails auxquels j'attache une importance toute spéciale ; ce sont : la largeur du conduit, la manière de préparer la gaze qui sert à pratiquer le tamponnement, la forme sous laquelle la gaze doit être appliquée, ainsi que la durée pendant laquelle elle doit être laissée ; enfin, je terminerai en disant quelques mots de l'oblitération de la trompe d'Eustache.

1° La largeur post-opératoire du conduit auditif présente selon moi une importance de tout premier ordre ; alors que les conduits étroits ne

permettent pas de se rendre compte des suites opératoires, et risquent de compromettre l'issue de l'opération ; au contraire, un conduit large permet de s'assurer régulièrement et sûrement de l'état de toutes les parois de la cavité, ce qui est indispensable pour parer aux difficultés qui peuvent toujours survenir. J'irai même plus loin : dès le moment où la cavité opératoire ne communique plus avec l'extérieur que par un conduit étroit, la marche de l'épidermisation se trouve notablement retardée ; en effet, la peau a besoin d'air et de lumière pour vivre, et à plus forte raison pour se développer ; nous pourrions donc comparer la cavité opératoire à une habitation qui, pour réunir les conditions hygiéniques nécessaires, doit être bien aérée et bien éclairée.

Il est donc nécessaire que la cavité opératoire puisse se trouver dans les meilleures conditions pour que la marche vers la guérison ne soit interrompue par une mauvaise épidermisation. Quand par une faute lors de l'exécution de la plastique, ou pour toute autre cause quelconque, le conduit n'a pas conservé l'amplitude nécessaire, presque invariablement un certain retard dans la marche de l'épidermisation a été constaté. Par contre, les cas les plus favorables dans ma statistique sont ceux dans lesquels l'amplitude du conduit a permis l'exploration très facile de toute la cavité, permettant dans certains cas de se rendre compte de l'état de toutes les parois de la cavité et de la caisse du tympan sans avoir recours à l'emploi du spéculum, car il doit être suffisant de projeter la lumière à l'aide du miroir ou du photophore frontal.

Le fait d'obtenir un conduit suffisamment ouvert dépend en grande partie de la méthode de plastique que l'on a adoptée, sans méconnaître toutefois une certaine importance à la façon d'exécuter cette plastique.

Après avoir employé, dans ma pratique, toutes les différentes techniques jusqu'ici connues, je me suis arrêté à deux de celles-ci qui me paraissent réaliser mieux que toutes les autres l'objectif que nous recherchons, ce sont : *les méthodes de Siebemann et de Körner.*

Dans la méthode de Siebemann, je résèque le lambeau cartilagineux triangulaire qui résulte de la bifurcation de l'incision au niveau de la conque, le plus loin possible, afin de pouvoir très facilement déprimer ce dit lambeau par le tamponnement, et par le fait même, assurer l'amplitude maxima du conduit. Je suis aussi d'avis qu'il est bon de réséquer avec une pince coupante les bords du cartilage qui restent visibles à la partie antérieure et à la partie inférieure de l'incision de la conque ; ainsi on éloigne ces dits bords cartilagineux de la surface cruentée, et en les protégeant à l'aide des parties molles, on évite, autant qu'il est possible, le danger de la périchondrite. Comme on peut le constater par la lecture de mes observations cliniques, je n'ai jamais eu à me plaindre de cette complication.

2° La manière de préparer la gaze qui sert à pratiquer le tamponnement a aussi une très grande importance, et je vais même jusqu'à affirmer qu'elle a un rôle capital dans la rapidité avec laquelle j'assure la guérison de mes opérés.

Dans les différents services d'oto-rhino-laryngologie, on emploie pour pratiquer les pansements des opérés de radicale, de la gaze que l'on coupe au moment même de s'en servir ; on est amené ainsi à recourir soit à de grandes pièces de gaze stérilisée, soit à des bandes de 2 ou plusieurs centimètres de large, à bords tissés, vendues au public toutes stérilisées par certains établissements industriels. Dans le premier cas, comme les ciseaux coupent la trame de la gaze dans différentes directions, il s'ensuit comme conséquence la formation d'une quantité de bouts de fils qui, se détachant facilement, deviennent adhérents et restent perdus sur la paroi de la cavité opératoire chaque fois que l'on change le pansement. Dans le second cas, lorsqu'on emploie de la gaze à bords tissés, il est naturel que le nombre des fils tirés et détachés est beaucoup moins important que dans le cas précédent, mais, néanmoins, un certain nombre adhèrent et se perdent sur les parois de la cavité opératoire.

Ce sont ces bouts de fil qui s'incrustent pour ainsi dire entre les bourgeons charnus formés sur toute la superficie de la cavité, véritables corps étrangers, qui maintiennent une irritation ayant comme conséquence un développement exubérant et, permettez-moi l'expression, pathologique, desdits bourgeons charnus qui empêchent presque complètement l'épidermisation. Celle-ci en effet ne se fait pas avant que les tissus de nouvelle formation aient obtenu un certain degré de condensation, leur permettant de former un plan uniforme, à superficie lisse, sur lequel se développera la peau neuve. Que de fois il est arrivé, alors que je pensais obtenir rapidement une guérison, de voir celle-ci interrompue par une prolifération exagérée des bourgeons charnus, survenue en un point quelconque des parois de la cavité. En présence de ces bourgeons, devant la ténacité de leur production, on emploie les caustiques les plus différents parmi lesquels nitrate d'argent, acide chromique, acide trichloro-acétique, etc. ; d'autres opérateurs ont recours au galvano-cautère ; parfois même on pratique un curettage des parties molles et de la superficie osseuse parce qu'on pense à l'existence d'une ostéite, alors qu'au fond tout était dû à la présence de pareils débris de gaze qui étaient restés cantonnés entre les bourgeons et maintenaient une irritation permanente qui était la cause de cette prolifération. Heureusement que le plus souvent il suffit d'extraire ces débris de gaze pour voir rétrocéder spontanément les granulations exubérantes, et arriver à obtenir que le tissu de nouvelle formation présente une surface plane et une condensation indispensable pour la bonne marche de l'épidermisation.

Pour éviter ces inconvénients, j'ai fait préparer la gaze de la manière suivante : sur une bande de gaze à bords tissés et de deux centimètres de large se coupent des mèches de 5 à 6 centimètres de long ; on prend soin d'arracher au début les extrémités des fils qui pourraient ultérieurement se détacher, et on place ces mèches dans de petites boîtes métalliques où elles supporteront la stérilisation.

De cette manière, j'ai la certitude qu'il ne restera pas sur la paroi opératoire des bouts de fil pouvant faire effet de corps étrangers, et

déterminer les accidents antérieurement signalés ; les pansements peuvent se faire avec la plus grande facilité et avec toutes les garanties de la plus complète asepsie.

On pourrait certes éviter qu'il reste des fils dans la cavité opératoire en employant une seule mèche de gaze à bords tissés aussi large qu'il serait nécessaire, et en prenant soin de pratiquer les pansements en laissant à l'extérieur les deux extrémités de la gaze, mais j'objecterai que de cette manière il est impossible de faire un tamponnement aussi complet, aussi uniforme, qu'avec la gaze telle que je l'emploie.

3° La manière de pratiquer le tamponnement a aussi une importance très grande ; si on ne le fait pas avec la régularité nécessaire, certaines parties de la cavité se trouvent plus comprimées que d'autres, et il en résulte que les tissus de nouvelle formation se développent d'une façon inégale, ce qui se trouve avoir toujours une répercussion sur la marche de l'épidermisation.

Par contre, s'il existe une certaine inégalité dans le développement des bourgeons charnus, avec un bon tamponnement effectué avec soin il est facile de remédier à cet inconvénient, et en procédant de cette manière, nous sommes pour ainsi dire maîtres de la cavité.

4° Un autre point important, se rapportant au tamponnement, est la fréquence avec laquelle on doit renouveler les pansements et la durée pendant laquelle ils doivent être employés, chez chaque opéré. En somme, ma manière de procéder est à peu de chose près la suivante : 4 à 5 jours après l'opération, je fais enlever seulement le pansement superficiel afin de constater d'une part l'état de la suture rétro-auriculaire, d'autre part l'entrée du conduit ; en un mot, je jette un coup d'œil sur l'état de toute la région opératoire. De cette manière, s'il se produit la plus petite complication, telle que eczéma du pavillon auriculaire, infection d'un point de suture, etc., je puis immédiatement intervenir pour tout modifier favorablement.

Au contraire, si ce pansement avait été fait tardivement, l'eczéma aurait pu prendre de grandes proportions ; le point de suture aurait pu infecter par contiguité toute la plaie rétro-auriculaire, et finalement amener l'infection de toute la cavité opératoire ; en outre, de ces différents avantages, j'estime que le changement assez précoce du pansement superficiel est à recommander par simple règle d'hygiène, car il est toujours plus ou moins imprégné de sang, et par son odeur peut incommoder le malade, qui, ne souffrant nullement lors du changement du pansement, en éprouve au contraire un véritable soulagement.

Les pansements internes, c'est-à-dire les mèches qui ont servi à tamponner la cavité, ne sont changés pour la première fois que le septième ou le huitième jour après l'opération, et ultérieurement tous les deux jours ou même tous les jours selon que je le crois nécessaire. Si la formation du tissu de nouvelle formation qui va recouvrir toute la superficie

de la cavité opératoire se fait dans de bonnes conditions, et si l'épidermisation a commencé franchement, je suspends le plus vite possible tout tamponnement, car on sait que celui-ci agit comme un corps étranger, et, souvent, contribue à retarder la marche de l'épidermisation.

Je continue à surveiller de très près mes opérés, et une fois que je me suis décidé à ne plus faire de tamponnements, je me contente de nettoyer la cavité des sécrétions qui toujours se produisent ; ce nettoyage doit être fait toujours avec de la gaze stérilisée sans fils, et très soigneusement afin de ne pas faire détacher les fines lames épidermiques qui sont en formation. Je pratique enfin, selon que je le crois nécessaire, ou bien des insufflations à l'aide d'acide borique, ou de stéarate de zinc, ou bien des applications de pommade au scarlate R ayant pour but de protéger l'épiderme en formation et si possible d'accélérer sa marche.

Dans certains cas cependant, j'ai dû avoir recours aux greffes épidermiques selon la méthode de Thiersch pour accélérer une épidermisation qui se faisait trop lentement, ce qui peut s'observer chez des malades à mauvais état général.

5° La trompe d'Eustache est un des pires ennemis avec lesquels il faut compter au cours des pansements d'un opéré de radicale et même pendant l'avenir de celle-ci. Comme dans la majeure partie des cas de suppurations chroniques de l'oreille moyenne il s'est déjà produit aussi une infection plus ou moins intense de la trompe (salpingite), ayant, comme caractéristique, la production d'une petite quantité de sécrétions muco-purulentes qui apparaît sous forme d'une goutte au niveau de l'embouchure de la trompe dans la caisse. C'est cette petite goutte qui en macérant l'épiderme de nouvelle formation le détruit, et avec ses débris forme un magma qui finit par infecter toute la cavité. Parfois, alors qu'il n'existe qu'une simple salpingite, si la trompe est restée perméable, tous les états inflammatoires à point de départ nasal ou naso-pharyngien se répercutent sur la cavité opératoire ; c'est à peu près la même chose qui se passe dans les cas de perte totale ou presque totale de la membrane du tympan au cours de processus anciens ; dans ce cas, sans avoir pour ainsi dire un état pathologique permanent de la caisse du tympan et de ses annexes, les malades sont sujets à de fréquentes poussées suppuratives chaque fois qu'il se produit une infection aiguë nasale ou naso-pharyngienne. Pour remédier à ces inconvénients, j'ai pratiqué plusieurs fois, comme je l'ai communiqué au Congrès Universel de Madrid en 1903, l'oblitération de la trompe par une méthode que j'ai alors décrite.

On sait d'ailleurs la grande rapidité avec laquelle se guérissent les évidés chez lesquels l'oblitération de la trompe se produit spontanément ou consécutivement à l'acte opératoire.

Dans ma pratique, en cherchant systématiquement la réalisation de ce principe, je fais toujours, au cours du temps opératoire dans lequel je pratique l'évidement pétro-mastoïdien, le fraisage de l'embouchure de la trompe ; je cherche à détruire la muqueuse, à provoquer une

cicatrisation qui oblitère le conduit ; en outre, en régularisant cette entrée de la trompe, je facilite l'épidermisation, ce qui est aussi un moyen d'obtenir son oblitération.

Grâce à cette façon de procéder dans le traitement post-opératoire des évidements pétro-mastoïdiens, je me trouve très satisfait des résultats puisque, non seulement, j'arrive à obtenir la guérison dans un temps aussi court que les meilleures statistiques publiées jusqu'à ce jour, mais aussi parce que, ainsi qu'on a pu le constater sur mes opérés, mes résultats sont particulièrement bons au point de vue de la conservation de l'audition.

[Thursday Afternoon, August 7]

SECTION XVI

OTOLOGY

INDEPENDENT PAPER

THE MECHANISM OF LABYRINTHINE NYSTAGMUS AND ITS MODIFICATION BY LESIONS IN THE CEREBELLUM AND CEREBRUM. AN EXPERIMENTAL INVESTIGATION

By J. GORDON WILSON, Northwestern University, Chicago, and F. H. PIKE, Columbia University, New York

Labyrinthine nystagmus, both in clinical practice and in discussions on experimental results, is designated in accordance with the quick movement. This is an anomaly, since it reduces to a position of secondary importance the most essential factor in the phenomenon—the cause of the deviation of the eyes, from the primary position of equilibrium (Sherrington, *Integrative Action of the Nervous System*, p. 279, New York, 1906), or from the primary position of the line of fixation of Listing. It must not be forgotten that the eyes of normal subjects have a very definite position when at rest or during fixation. Any involuntary departure from this position means that some unusual influence is operative to cause the deviation, and it is precisely this deviation and influence which the clinician is called upon to explain. Various influences, acting through the central nervous system, may cause such deviation of the eyes, and each one may act in its own peculiar way. The return to the primary position, if at all possible, is likely to be much more uniform than the manner of deviation from the normal. In labyrinthine nystagmus, as we have elsewhere described it (Wilson and Pike, *Phil. Trans. Royal Soc.*, pp. 127–60, 1912; also *Proceedings of the Society for Experimental Biology and Medicine*, New York, 1913, x, pp. 81, 82), the slow component is of labyrinthine origin, and hence is of most significance so far as its relation to the labyrinth is concerned. Variations in the manner of the quick return indicate disturbance in the oculomotor rather than in the labyrinthine or vestibular mechanism. In labyrinthine nystagmus both the slow deviation and quick return are a co-ordinated series of associated movements, involving contraction of certain groups of muscles, and at a certain stage of the process, relaxa-

tion of their antagonists, as Bartels's experiments demonstrate (Bartels, 'Ueber Regulierung der Augenstellung durch den Ohrapparat,' *Arch. f. Ophthal.*, vol. lxxvi, p. 1; vol. lxxvii, p. 531; vol. lviii, p. 129; vol. lxxx, p. 207). Without lesions in other parts of the central nervous system, there is no double vision at any period. Only when some intracranial complication or oculomotor paralysis exists does double vision arise.

Uhthoff (Uhthoff, *Augenerkrankungen bei Erkankung des Nervensystems*; Graefe-Saemische, *Handbuch d. ges. Augenheilkunde*, Abt. Rückenmark, 2. Ausg., S. 3, v. 1904) distinguishes two varieties: (1) Nystagmus (Bartels, *Pendelnystagmus*), a pendulum movement of the eyes in both directions from the central point of rest; (2) nystagmus twitching (Bartels, *Rucknystagmus*), when the movement is quicker in one direction. English writers have generally applied the term nystagmus to all oscillations of the eyes. Occasionally, the term nystagmic twitching or nystagmoid movement is used to distinguish the more irregular types.

Various mechanisms for nystagmus have been postulated from time to time. Wundt postulated a centre for the afferent impulses from the soft parts of the eyes in the cerebellum. As we have shown, however, typical labyrinthine nystagmus may be observed after complete removal of the cerebellum. Bárány has located a nystagmus centre in the angular gyrus, and at some point above the oculomotor nuclei, possibly in the pons (Marburg, *Neurologisches Centralbl.*, 1912, Bd. xxxi, p. 1366). Marburg (*loc. cit.*) has postulated a nystagmus centre in Deiter's nucleus, basing his conclusion on the fact that lesions of Deiter's nucleus, when the rest of the encephalon is supposedly intact, give rise to nystagmus.

Two general objections may be urged against the various hypotheses so far advanced. The first is that writers often assume that if nystagmus arises from a lesion of a particular region, that region is a centre for nystagmus. A lesion of Deiter's nucleus, for example, is merely a lesion of one part of the central portion of the vestibular tract, and nystagmus would naturally arise from such a lesion, just as it does from lesions of the membranous labyrinth of the ear or from intracranial section of the vestibular nerve. The second objection is that little regard has been paid to the whole complex motor mechanism of the eyes, which cannot be described in terms of circumscribed isolated centres, but which must be described in terms of an integrative mechanism or system.

In the consideration of nystagmus after unilateral labyrinthine destruction we are met at the outset with two series of facts. First, nystagmus does not occur in many of the lower vertebrates, but in all of these vertebrate types with movable eyes so far examined, a deviation of the eyes does occur, and this deviation is permanent. Second, nystagmus does occur in the higher vertebrates, but neither nystagmus nor deviation is permanent. In considering, therefore, the mechanism of nystagmus in the higher vertebrates, we have to explain why neither nystagmus nor deviation of the eyes is permanent in these forms.

In the vertebrates, nystagmus, after stimulation or unilateral labyrin-

thine removal, varies as a rule in intensity with the position of the animal in the vertebrate phylum. Its intensity is greatest in the mammals. In Petromyzon it is not present, nor does it appear in the frog and the turtle. Some, however, believe that small compensatory movements of the eyes may be observed in the frog. In the cartilaginous fishes it is absent in many forms, e.g. dog-fish. When present, careful observation is required to demonstrate its presence, both in the cartilaginous and in the bony fishes. Ewald (Bartels, *loc. cit.*, 1910, pp. 17–21, 51) showed its presence in pigeons, but so far as our observations go, it is slight and less pronounced than in mammals.

There are two very definite reasons which lead to the separation of the two phases in nystagmus with regard to cause and source.

1. The deviation alone occurs under an anæsthetic. The quick phase only appears as the effect of the anæsthetic begins to wear off, and the animal regains consciousness.

2. When the anæsthetic is pushed, the slow deviation is one of the last of the phenomena about the head to disappear, and persists nearly or quite as long as the corneal reflex (Wilson and Pike, *loc. cit.*, pp. 151–5).

3. Bartels has shown that the slow phase alone can be produced by rotation in sleeping children, and that in prematurely born children (seven months) the slow deviation alone can be elicited for some time after birth.

4. We have shown in a previous paper (Wilson and Pike, *Phil. Tran. Roy. Soc.*, 1912) that the quick phase can be eliminated by destruction of the cerebrum and thalamus while the slow persists. As there pointed out, it appears to us that the paths along which the slow deviation occurs are fairly definitely established. We have shown that the slow deviation persists when the animal is at least partly under the influence of a narcotic (Wilson and Pike, *loc. cit.*, p. 158). Where and how far in their course the vestibular fibres cross the middle line between the labyrinth and the oculomotor nuclei, has not been established. Loeb's experiments and Bartels's work would seem to indicate a crossing, while the anatomical work of Winkler appears to indicate that the crossing is only partial.

We have endeavoured to throw some light on the question by splitting the pons and medulla in the middle line in dogs. So far these experiments tend to substantiate Winkler and our already published hypothesis, since they show that there is no crossing of fibres from the vestibular nerve in the anterior two-thirds of the pons, and whatever crossing occurs is in the region of the nucleus of the sixth nerve.

Having determined that in labyrinthine nystagmus we are dealing with a double movement in which the slow deviation can be definitely separated by experiment from the return movement, we now look for the source of the quick backward jerk. There are several possibilities of its origin.

We may suppose that the quick backward jerk arises in response to

impulses which tend to pull the eye back so that the visual images will fall on a particular retinal field (Wundt). Two series of facts militate against such an hypothesis. Nystagmus occurs in blind subjects when the lesion is in the retina or some part peripheral to the terminations of the optic nerve in the central system (Mulder, *Archiv für Ophthalmologie*, 1875, Bd. xxi, p. 68), when placed on the turning table, or when the ears are irrigated with hot or cold water. And, as we have shown (Wilson and Pike, *loc. cit.*, p. 151) nystagmus quickly returns when one or both occipital lobes of the cerebrum are removed. Hitzig also noticed nystagmus in blind tabetics.

A second possibility is that the abnormal deviation of the eyes sets up sensory impulses in the afferent endings of the eye muscles themselves, which bring about a return, either reflex or voluntary, of the eyes to the normal position. Nystagmus would then be a response to kinæsthetic sensations. We consider this the more probable hypothesis, supported as it is by some observations we have made on third-nerve paralysis. It is an hypothesis very closely akin to that of Bartels. We have then to trace out the relation of this movement to the cerebrum or thalamus. Bartels's observation on prematurely born children indicates that it involves a path which becomes medullated relatively late.

In comparing the deviation of the eyes which occurs after cortical stimulation with the deviation of the eyes which results from labyrinthine stimulation, it would appear that the relaxation of the antagonistic muscles is not the same in the two cases. Thus from Sherrington's observations (Sherrington, *Integrative Action*, p. 279; *Proceedings of the Royal Society of London*, 1913, vol. lxxxvi, p. 225, and 1893, pp. 407–20) it would appear that in cortical stimulation the contraction of a muscle is equal to and balanced by the relaxation of its antagonist. That this is not the case in stimulation of the labyrinth, a consideration of Bartels's diagram appears to show. Here the amount of relaxation in the antagonist falls behind the amount of contraction in the prime mover. As a result, we believe that in the deviation of the eyes from labyrinthine stimulation a strain is put upon the antagonistic eye muscles, which strain does not arise in movement from cortical sources. This strain produces a feeling of unusual or uncomfortable position which, when the sensory and motor systems are intact, results in a return to a more comfortable position. The return of the eyes to the median position would, on such an hypothesis, be of exactly the same nature as the return of a dog's hind foot from an uncomfortable position, when the sensory and motor systems are intact.

We have attempted to locate a region where a lesion consistently affected or abolished the quick component of nystagmus arising either as a result of destruction of, or stimulation of, the labyrinth. We have pointed out in a previous paper that such a region does not exist in the cerebellum. For as there shown, the removal of the lateral lobe or of

the vermis or of the whole cerebellum may affect, but does not abolish, the nystagmus which follows labyrinthine destruction. Since these results were published, we have repeated in detail these experiments, and with the same result. Accordingly we feel justified in saying that neither the slow deviation nor the quick return has any necessary pathway, nor any indispensable centre in the cerebellum.

We have previously pointed out that in cats and dogs complete decerebration, including the corpora striata and the thalamus, wholly abolishes the quick component after labyrinthine stimulation or removal, although the slow deviation of the eyes persists. We are unable to confirm Bauer and Leidler (Bauer and Leidler, *Arbeiten a. d. Wiener Neurologischen Institut*, 1911, xix. p. 155), who report the occurrence of nystagmus after removal of all parts of the cerebrum as far down as the corpora quadrigemina. A number of new experiments substantiate our former conclusions. The quick movement in nystagmus arises from some mechanism above the corpora quadrigemina—a point in which we confirm Bartels's view. The question becomes, then, whether the quick component of nystagmus arises from the thalamus or from the cerebrum. Anatomically, afferent tracts pass from the eye muscles back to the oculomotor nuclei, and then, in all probability, through the thalamus, where they are relayed, and then on up to the cerebral cortex. Efferent tracts arise in at least three different regions of the cerebral cortex, and pass down to the oculomotor nuclei. It appears to us that the mechanism of nystagmus involves cortical cells and areas, and that any return of nystagmus after injury to a part of the cortical mechanism may be explained by supposing that the shorter, more primitive paths through the subcortical regions become passable for nervous impulses.

In assuming that the mechanism of nystagmus involves thalamic paths and nuclei alone, we meet with certain difficulties in arriving at a satisfactory explanation. We must explain why, when every other part of the efferent oculomotor path is open, as shown by the presence of the corneal reflex, *and* the slow deviation of the eyes in response to labyrinthine stimulation, the path involved in the production of the quick movement should be the only one to be blocked. The explanation of shock does not explain anything until we tell what shock is. A hypothetical principle of explanation which in the last four decades of investigation has proved so confused and conflicting as shock, and which follows no laws so far discovered, and apparently no discoverable laws, is not likely to lead us far as a constructive principle. We may point out that shock, if present at all, follows the same general course with reference to nystagmus that it follows in the case of the skeletal muscles generally, that is, it is exerted on the afferent pathway, since, to all our tests, the efferent path is so obviously open (Pike, *Amer. Journ. of Physiology*, 1909, xxiv, p. 124; ibid., 1912, xxx, p. 436; *Quarterly Journal of Exp. Physiol.*, 1913, vii, p. 1).

We may assume then, as a working hypothesis, that some cerebral mechanism is involved in the production of the quick phase of nystagmus, and it remains to trace out as far as possible the regions and tracts concerned. There are two possibilities: (1) both cerebral hemispheres may be involved in the production of the quick phase of nystagmus when the slow component is in a given direction; (2) only one cerebral hemisphere may be involved. If only one is involved, what relation does it bear to the direction of the slow or labyrinthine phase? Experiment shows that the second possibility is more nearly the true one so far as the lateral movement is concerned. Space does not permit the recital of the details of the experiments at this time, and a brief presentation must suffice. This conclusion is based:

1. On the results of removal of one labyrinth and one cerebral hemisphere, noting the presence or absence of nystagmus.

2. On the results of electrical, caloric, and rotation tests in animals.

3. On the results of electrical, caloric, and rotation tests in animals in which both labyrinths are intact, but in which one cerebral hemisphere has been removed.

As a general result, we may state that complete removal of the cerebral hemisphere on the side of the slow deviation abolishes all except a small rotary component of the quick phase of nystagmus. Complete removal of the cerebral hemisphere of the side toward which the quick component is directed has but little effect on the quick movement.

Rotation, electrical, and caloric tests on such animals again show the dropping out of the quick phase of nystagmus when the slow deviation of the eyes is directed toward the side of the cerebral lesion, and little or no effect when the slow component is directed toward the uninjured side of the cerebrum.

We may here note in passing that injury to the anterior corpus quadrigeminum of the side to which the slow deviation of the eyes is directed does not abolish the nystagmus, either in its slow or quick phase, but it does bring about a dissociation of the eye movements.

These tests on animals with both labyrinths intact, but with removal of one cerebral hemisphere, lead to the same conclusion.

We have attempted in dogs, cats, and monkeys to find some region in a hemisphere where a lesion will modify or abolish the nystagmus which follows stimulation or destruction of the labyrinth. In the course of the experiments we have removed the various areas of the cortex, frontal, parietal, occipital, and temporal, and have also at times destroyed the subcortical ganglionic masses. Having destroyed these areas, we have then stimulated one or both labyrinths with the electrical current, with hot and with cold water, and by rotation. We have also in many of these cases destroyed one labyrinth either concomitantly with or subsequent to the cerebral lesion—a labyrinth either on the side of, or opposite to, the cerebral lesion. We cannot at this time enter into the

details of the results, which will be published in full later, but we desire to state here that the only region where we obtain consistent alteration of the labyrinthine stimulation, and that only so far as the quick component is concerned, is the region of, or adjacent to, the temporal lobe. In these cases we found that stimulation of the labyrinth on the side opposite the cerebral lesion with cold water or with the anode, gave typical labyrinthine nystagmus lateral in character, while stimulation with hot water or the cathode gave deviation, but no lateral nystagmus. We also found that destruction of the labyrinth on the side of the cerebral lesion gave no nystagmus. There was further noted in some of our cases that the eye on the side of the lesion showed a rotary movement around an antero-posterior axis, while this movement was wanting in the eye of the opposite side.

To sum up, our idea of the mechanism of nystagmus is first an agency somewhere which produces a deviation of the eyes from the primary position of equilibrium or the primary position of the line of fixation of Listing. In labyrinthine nystagmus this agency lies in the labyrinth. The internal rectus muscle on the side of the slow deviation and the external rectus on the opposite side are subjected to a strain greater than usual. This strain resulting in the stimulation of the sensory or afferent endings of the eye muscles, sets up afferent impulses, which are conveyed to the cerebrum along the afferent fibres in the third, fourth, and sixth cranial nerves (Tozer and Sherrington, *Proc. Roy. Soc.*, 1910, p. 450). These afferent impulses in their turn set up efferent impulses in the oculomotor cells of the cerebrum, which result in a quick, jerky contraction of the internal rectus on the side of the slow deviation, and of the external rectus of the opposite side, with relaxation of the antagonistic muscles. The effect is a restoration of the eyes to the primary position. The eyes do not go beyond this median position when the cerebrum is intact, since the afferent impulses to contraction of the external and internal rectus muscles concerned cease when the eyes reach this position of equilibrium.

Lesions of the cerebral hemisphere on the side of the slow deviation of the eyes abolish or reduce the quick component, since it is from the hemisphere of this side that the efferent impulses concerned in the pulling of the eyes back to the median line arise.

DISCUSSION

Dr. Bárány (Wien) beglückwünscht die Herren Wilson und Pike zu ihren Versuchen. Er freut sich, dass die Frage des Nystagmus jetzt solchem Interesse begegnet. Was das Detail der Versuche anbelangt, so möchte er bemerken, dass hier in der Tierreihe sehr wesentliche Unterschiede bestehen. Leidler und Bauer haben ihre Versuche am Kaninchen gemacht, Wilson und Pike am Hunde, der Katze und am Affen. Es ist also eine direkte Vergleichung nicht möglich. Ganz im allgemeinen

aber ist zu sagen: Leidler und Bauer haben ihre Operationsergebnisse mikroskopisch kontrolliert, und wenn sie ein positives Resultat angeben, d. h. wenn sie typischen Nystagmus auslösen konnten, so ist unbedingt dieser Angabe mehr Wert beizumessen, als einer negativen. Man muss bei den Tierversuchen enorm vorsichtig sein und nur, wenn ein Experiment wiederholt dasselbe Resultat ergibt, dieses Resultat verwerten. Denn in einzelnen Fällen ergaben sich oft ganz unerklärliche Folgen des Eingriffes, die offenbar auf Fernwirkung, auf Zerrungen oder Blutungen etc. beruhen. In einzelnen Details, so bezüglich der Nystagmuszentren, bezüglich der Rolle des Kleinhirns, haben Wilson und Pike Báránys Ausführungen missverstanden, doch würde es zu weit führen, darauf einzugehen.

Professor ALEXANDER (Wien) hat die histologischen Präparate der Versuchstiere seines Assistenten Dr. Leidler und Dr. Bauers genau durchgesehen, und auch eine Reihe der von Leidler und Bauer operierten Tiere beobachtet. Es kann kein Zweifel darüber sein, dass Leidler und Bauer noch Nystagmus an Versuchstieren beobachtet haben nach Entfernung des ganzen Grosshirns bis an die Corpora quadrigemina. Es ist nötig, dass Wilson und Pike diese Differenz aufklären. Referent fragt, wieviele Tiere Wilson und Pike operiert haben, welche Tiergattungen, welche Anästhesierung verwendet wurde, und ob die histologische Kontrolle der Versuche vorgenommen worden ist.

REPLY

Dr. J. GORDON WILSON (Chicago). In reply to Professor Alexander, I may say that our experiments were done on dogs, cats, and monkeys. An anæsthetic was always used, generally ether, though several of the animals had also a preliminary injection of morphia. In all cases a careful post-mortem examination was made, and in a few a microscopic examination is now being made. These experiments have not been done on rabbits, We found these animals less satisfactory for making observations on, and difficult to keep over a long period. The suggestion that the rabbit would yield so distinctly different results from those on carnivora and primates would be an interesting phenomenon in comparative physiology.

Replying to Professor Bárány, we do not expect to locate a nystagmus centre, for we believe we are dealing with an integrative mechanism.

There appears to be some misunderstanding in regard to the number of experiments on which this paper is based. This may have arisen from the printed copy which says, quote monkey, June 15, and again, dog, June 2. It was the original intention to report these experiments in full as typical results, but time did not permit. I would like to say that the experiments on which this paper is based includes records, with post-mortem findings, of seventeen experiments on dogs, six on cats, and one on a monkey. These figures are exclusive of the results published in a previous paper. The original records are here and can be seen by any one interested. They will be published in full at a later date.

SECTION XVI

OTOLOGY

INDEPENDENT PAPER

THE CLINICAL DEVELOPMENT OF MY SYMPTOM-COMPLEX

By Dr. ROBERT BÁRÁNY, Privatdozent, Vienna

If for a certain clinical image an anatomical or pathological explanation is given, it may be asked with full right that this explanation be proved by autopsies. But it may happen, that though the clinical image is observed frequently, no opportunities of making a post-mortem are obtained, because the disease does not lead to death, and also thorough examination during operation cannot be made. Then the explanation must remain a hypothesis, until this last direct proof is brought; but as in a case before the jury the barrister tries to prove his case by indirect evidence, the clinician must do likewise. I am myself in this last position, though I have five cases in three of which post-mortems have been made and the other two have been operated upon, as these five cases also give no absolute proof but only a certain probability for my assertion. In this position I think it best to show how the clinical picture has been developed and how the conclusions as to the anatomo-pathological cause have been formed.

At the end of the year 1907 a girl (S.), aged 20 years, came under my observation in the clinic with the following history. For one year and a half she had been suffering from tinnitus of the left ear. In August 1907 she went on a mountain excursion in the neighbourhood of Vienna. The mountain is 2,000 m. high, the place at the foot of the mountain is 500 m. high. When she came down from the mountain she suddenly heard a loud blow in the left ear and then got an attack of very severe dizziness with vomiting. The hearing of the left ear diminished immediately and considerably. Some minutes after the beginning of the attack a severe headache in the posterior part of the head developed, which lasted several hours. Since this time the attacks of severe dizziness have recurred frequently, and in the meantime she is not free from dizziness. Turning the head to the left side, looking upward, bending the head to the left shoulder, produces short attacks of dizziness. When

she gets dizzy, she immediately feels ill, and in the severe attacks she always vomits. She has lost several kilogrammes in weight, having no appetite, and often vomiting the little she eats. When I saw her first, I found a girl who looked emaciated. The investigation of the inner organs, of the central nervous system, of the fundus of the eyes was negative. No signs of hysteria. No syphilis, no nicotine, no alcohol, no rheumatism. The investigation of the ear gave the following result: Left drum retracted, no secretory catarrh, right drum normal. Hearing on the left: for middle whispering voice ½ m., ordinary conversation voice 3 m., right side normal. Weber to the left. Rinné on left side negative, bone conduction a little lengthened, air conduction very much shortened. High notes shortened. No spontaneous nystagmus. When the head is bent backward, an attack of rotary nystagmus to the left is elicited, which lasts about half a minute. Caloric reaction on the left side diminished; compression and aspiration of the air in the meatus produces exceedingly small slow movements of the eyes and dizziness. With compression the slow movement goes to the right, with aspiration to the left. Considering the retracted drum, the localization of the Weber to the left, the lengthened bone conduction, the long duration of the tinnitus, I made a diagnosis of chronic catarrh or adhesive process of the middle-ear involving the labyrinth. The patient first was treated with inflations, with catheter, and with massage without any improvement. Then her neck was massaged, which produced vomiting, but did not help her. She was given bromides, sulphate of quinine, aspirin, pyramidon, and other remedies without any influence upon her disease. She again was sent to the neurologist, who diagnosed hysteria and suggested a hypnotic treatment. This was effected, with the result that she slept very well during the hypnosis, but felt no effect on her disease. I did not believe in the hysteria, because I had observed very often the above-described attacks of nystagmus with rapid head-movements and the diminished caloric reaction. These two things hysteria cannot produce. Except her ear trouble no other sign of hysteria was present. Though all our treatment had not helped so far, the patient was still full of confidence and always came again begging us to help her. By chance I read at this time the paper of Babinski, published in 1904 in the *Annales de maladies de l'oreille*, in which he describes the marvellous effects of lumbar puncture on different ear symptoms, especially on the dizziness. Having detected by chance that dizzy attacks ceased after lumbar puncture, Babinski had applied this method, which in the beginning was purely diagnostical, for a therapeutical purpose and had treated more than 100 cases in this way. A considerable number of cases showed recovery from improvement of hard hearing, tinnitus, and dizziness, especially if immediately after the lumbar puncture severe headache had developed. The cases of Babinski are not selected; there are cases with middle-ear disease, others with pure inner-ear disease; Babinski does not give any explanation of these facts. After Babinski, Lombard and Caboche made use of lumbar puncture in a few cases

with no striking effect, Lermoyez with a bad effect. Though I could not explain at all the effect of lumbar puncture, I still thought that this treatment should be tried. The patient accepted my proposal. The puncture at the beginning of March 1908 was made without narcosis, and 10 c.c. of clear liquid were evacuated. After this little operation the patient felt miserable. She got severe headache in the posterior fossa, the dizziness was increased, she vomited nearly continually. This stage lasted 10 days, then the headache slowly disappeared, the dizziness ceased, the hearing improved a little, the tinnitus remained. A functional test several months later showed middle whispering voice for 1 m., therefore no remarkable effect upon the hearing. Caloric reaction seems to give stronger nystagmus than before, but less dizziness. She rarely has mild attacks of dizziness, but no illness, no vomiting. Until October 1908 she felt pretty well, then the severe attacks of dizziness returned. In November 1908, on one occasion she fell down in an attack and became unconscious for a moment. She had a severe headache in the occiput after the attack, but no vomiting. As she felt very bad, a second lumbar puncture was made. But no liquid escaped. This puncture was also done without narcosis, and I tried suggestion. I showed the patient after the puncture a test-tube filled with water as if this liquid had been withdrawn. The suggestion remained without effect. This time the lumbar puncture did not produce headache like the first time, but the dizzy attacks came on without change. Three weeks later (November 26, 1908) I therefore made a third lumbar puncture under narcosis. This time 5 c.c. of liquor were again evacuated; after this puncture the same symptoms came on as after the first, but four days later she again was almost free from headache and dizziness. At this stage I presented her to the Otological Society of Vienna (November 30, 1908). Four months later she came again, and told me that for several days the tinnitus in the left ear had ceased, but that she was now completely deaf on this ear. The functional test showed the truth of this observation. It was very curious that the caloric reaction in opposition to the hearing had become more active. She did not wish any treatment, as she preferred to be deaf on one side and have no noises as during the previous stage. In May 1909 she came again with a double-sided acute otitis. She was almost totally deaf at this time, but soon recovered. The otitis resolved without paracentesis. In August 1909 she again presented herself and said that, to her great surprise, the hearing on the left ear had slowly come back. The functional test showed, indeed, that with the noise apparatus in the other ear she heard on the right ear conversation voice at 4 m., whispering voice at 1 m.; on the left ear conversation voice at 1 m., whispering voice on the ear. A month later she came again and told me that in the meantime the hearing had slowly improved and was now almost normal. I tried it with the noise apparatus in the other ear. She heard conversation voice at 7 m., whispering voice 2 m. with the left ear. A month later (October 29, 1909) the hearing had become almost normal, whispering

voice could be heard at more than 7 m., with noise apparatus at 4 m. Rinné positive ; bone conduction a little shortened. No dizziness. On bending the head backward I observed an attack of nystagmus to the left of a duration of 10 seconds. But she hardly felt dizzy. Compression and aspiration of the air in the meatus gave very small movements, but she felt no dizziness. Her general aspect was excellent and she felt perfectly healthy.

If we consider this history, we can only say that the diagnosis of a chronic adhesive process of the left middle-ear cannot be maintained, as without any treatment the hearing returned to the normal. This I remarked also at the time, but without knowing how to explain the case.

In the beginning of 1910, Dr. Breuer of Vienna, the father of the Breuer-Mach theory of the function of the semicircular canals, came to me with a patient and told me the curious history of the case.

It was a lady, aged 30 years (Schi.), married, who was very musical and had excellent hearing in both ears. For some years she had suffered from attacks of headache, which usually continued for several days, but on some occasions extended into weeks. The headache was always localized to the left posterior fossa ; at this time she heard worse in her left ear and was dizzy. But the most disagreeable symptom which she complained of was the double hearing during this time. In the left ear she heard every note higher than in the right ; it was, therefore, impossible for her, to hear music or to play the piano herself. The examination showed a normal drum on both sides. The hearing was very little diminished for C^4 and for the watch on the left side. Also a slight conduction trouble seemed to be present. Bending the head to the left produced an attack of rotary nystagmus to the left. Spontaneous nystagmus was absent. I could not understand the case, but Dr. Breuer gave me a very interesting explanation. He told me that this stage lasts usually for several days or weeks, but suddenly the patient feels as if something in the back of her head had ruptured and immediately she feels well again. The headache and dizziness disappear and the hearing becomes normal again. Dr. Breuer said : ' I think there is a cyst in the cerebellopontine angle, which fills with liquid ; then the wall ruptures, the liquid escapes, and the patient is well again.' The reasoning of Dr. Breuer seemed to me very striking, but I was obliged to say that until now I had never seen a case analogous to this. I thought that in the future I would pay special attention to such cases. I had no idea at this time that the first case, related in this paper and which I had had already for such a long time under my observation, was analogous to this one. The history of a discovery is sometimes very complicated and it is really in this respect. I must now beg you to follow me in quite another way ; the connexion between this and the first will only be apparent later.

At the meeting of the British Medical Association in July 1910, by

the kindness of Sir Victor Horsley, I saw four cases of cerebellar tumours, which had been operated upon and were already healed. I was able to make the diagnosis (only by testing the vestibular reactions, the pointing and falling reaction) that three of the cases had been operated on the left hemisphere, the fourth on the right side of the vermis. My diagnosis proved to be correct. This good result gave me much hope and energy to overcome the difficulties which I should still find. On my return to Vienna I was fortunate enough to diagnose, operate, and heal a cerebellar abscess in a case where I could observe the symptoms from the very beginning. It was a young man, who suffered from cholesteatoma, sinus thrombosis, and extradural abscess of the posterior fossa on the right side. I first performed a radical operation, ligated the jugular, and opened the sinus. The patient behaved quite well for two days. Then he got a cerebral aspect, he slept very much and when awake suffered from headache. I examined him and found a little nystagmus to the diseased side—the labyrinth had been normal—no signs of temporal lobe abscess, no papillitis optica, no ataxia of the limbs, no dysdiadokokinesis. The only symptom I found was that the right arm continually pointed to the right 15 cm. I made a functional test, syringed the left ear with cold water. The nystagmus to the right was quite normal, the left arm pointed normally to the left, the right arm did not point to the left, but only pointed correctly or a little to the right. There was evidently no reaction of the right arm to the left. I drew the conclusion that I had to do with a cerebellar abscess of the right hemisphere. I operated and found the abscess immediately. It was induced by the extradural abscess, was small and superficial. I made some incisions, went in with the forceps and drained the abscess with an india-rubber tube. The first day after this operation the pointing error to the right was still as before, but already on the second day the pointing error had disappeared. The patient pointed quite normally spontaneously. But when I syringed the left ear with cold water, the right arm was not influenced and pointed correctly as before, the left arm showing a big error to the left. This stage persisted for about eight weeks. I could examine the boy nearly every day, he being a very good patient, and not sensitive at all. After eight weeks, when I once again tried the reaction, I had to my great surprise and anxiety the impression that the reaction in the right arm was present again. I did not believe that the first time. But on the next day the reaction was clearer, and several days later it was perfectly normal. The wound had made good progress in the meantime, the abscess was nearly healed. This reappearance of the reaction gave me great trouble. I thought and thought how to explain it. Regeneration of nerve-cells was impossible, other centres, in my opinion, could not take the function of the part which I had destroyed. At last I came to the following conclusion. It is not at all certain that my incision in the cerebellum has really destroyed the centre for the arm. It is possible that this centre only lies very near the incision, so that the inflammation

and injury to the neighbouring structure had caused a temporary loss of function. Until now I had only examined the reactions of the arm and of the hip. But it is very probable also that the other muscles are under the influence of the vestibular apparatus. I must examine the other joints, the elbow, the wrist, in order to see if another centre has not been really destroyed by the incision, where the reaction did not return. I therefore developed methods to examine the elbow- and wrist-joint. The elbow gave no clear result. But the examination of the wrist was striking. In the left wrist the reactions to the left and right were absolutely normal and very strong, in the right wrist the reaction to the right was normal, the reaction to the left was completely absent. The reaction was absent to the left as well, when the palm of the hand was directed upward, as if it was directed downward. This result was constant, and remained also when the abscess was completely healed. I therefore was obliged to conclude that I had really destroyed by my incision the centre for the wrist for the movement to the left. From this case I drew quite a number of important conclusions, which have since turned out correct. I thought: the centre for the arm to the left must be quite near to the centre for the wrist to the left; so it is probable that all the centres for the movement to one side are near each other on one spot. As the centre for the arm may be quite normal, when the centre for the wrist is completely paralysed, it is clear that the localization in the cerebellum must be arranged after other joints. In the right wrist, as I have said already, the reaction to the right was present in the position of the wrist with the palm downward and upward; that means with the forearm pronated and supinated. In the position with the palm downward this reaction was produced from the cerebellar innervation of the ulnar muscles of the wrist. But these same muscles are paralysed when the palm is held upward should a reaction to the left take place. The same is true for the radial muscles. We therefore see that the same muscles must be represented at least twice, once in the centre for movement to the right and again in the centre for movement to the left. The centre for the left must be quite separated from the centre for the right. If two centres for the direction to the right and left are present, it is highly probable that two other centres also exist for the movement up and down. The centre for the wrist must be immediately behind the labyrinth, the centre for the arm in front or behind this centre. It has since been found out that it is behind the centre for the wrist. Since this case I have paid great attention to all the joints, especially to the arm, wrist, and hip. I examined also the reactions up and down. Just at this time it happened that the first-mentioned case (S.) came again to the hospital to see me. She was free from dizziness since the last lumbar puncture. Only the hearing had not always remained so well as when I saw her last. It often changed, got quickly worse and slowly better again. At this time I suddenly got the idea that perhaps a cyst in the cerebellopontine angle might be the cause of her disease, which

from time to time was more or less filled. I remembered at this moment the patient of Dr. Breuer with the sudden change of symptoms, and therefore I tested the reaction movements also in this case. The arms reacted normally, the wrist of the left side seemed to give no reaction to the inside, and the reaction to the other side was also not very strong. I did not come to a definite conclusion, and then the new idea seemed to me a very keen hypothesis, which therefore was again rejected.

At this time I had another very interesting case (E.) under my observation which in several respects taught me new things. It was a man of 42 years of age with a double-sided chronic otitis media. On both sides the lateral wall of the attic was destroyed, there were remains of cholesteatoma, but little suppuration, and the drainage seemed to be free. For several months the man had suffered from severe headache, especially in the posterior fossa, from dizzy attacks and vomiting. The investigation gave the following result : The head was mostly held bent forward, as in this position the pain was not so severe. There was strong spontaneous nystagmus of an intracranial character (to the right and left and upward). There was a tendency to fall backward. Both arms were slightly ataxic. No papillitis optica. The examination of the pointing test in the arms showed that in the left arm there was no reaction to the right, the right arm was normal. I thought that a cerebellar abscess or tumour was present in the left hemisphere of the cerebellum, but wished to examine the case again. When several days later I again made this examination a great change had taken place with the patient. He felt much better, had no headache, could walk quite well, and when the reactions of the arms were examined, I found a normal reaction in both arms to both sides. At this time I had not yet examined the other joints. The patient felt so well that he wished to leave the hospital and went home to Galicia. It was only six weeks later, in December 1910, that he returned again with the following history. At the time when he arrived home all the severe symptoms had come back, and he felt so miserable that he was quite unable to travel. As soon as he was better, he came again to Vienna and to the hospital. It was just at the time that I had detected that the above-described case of cerebellar abscess (V.) had normal reactions in the arms, but no reaction in the right wrist. When I examined the man again, and found that his arm reactions were normal, I therefore examined his reactions in the wrist. Then I found the curious fact that in the left wrist no reaction to the right was present when the forearm was pronated and the palm directed downward, but a normal reaction to the right could be obtained when the forearm was supinated and the palm directed upward. The reactions to the left of the left wrist were both normal, and in the right wrist all the reactions were normal. You remember that in the cerebellar abscess (V.) in both positions of the forearm the reactions to the inside were not present. This case, therefore, was different from the cerebellar abscess case, and I was compelled to diagnose that the cerebellar lesion must be still

smaller than in the abscess case. I could observe the above-described finding for a fortnight in repeated examinations, and could easily demonstrate to different men, as Professor Urbantschitsch, Professor Schlesinger, &c. In this case I still found a new symptom, which later became very important for the diagnosis of my symptom-complex. The spontaneous pointing of E. in the wrist had always given a normal result. I had seen already several cases where a reaction in a certain direction was absent, but no spontaneous error was present. Theoretically a spontaneous error opposite to the loss of reactions would be expected; for example, if the reaction to the inside is not present, there should be a spontaneous error to the outside. The absence of the spontaneous error I explained to myself by a correcting innervation which the cerebrum had learned to add to the cerebellar innervation, thus replacing the absent cerebellar tonus by an increased cerebral tonus. This cerebral correction seemed to me quite easy to understand, when in all positions of the limb the reaction in one direction was absent. But in the present case the reaction to the inside only was absent in the pronated position of the forearm and not in the supinated position. I therefore was astonished that no trace of spontaneous error was to be detected. Then it occurred to me that the position with the forearm pronated is the usual position, whereas the position with the forearm supinated is quite unusual. I understood that the cerebrum could have learned to correct the cerebellar defect in the usual position. In the other unusual position it had nothing to correct, and therefore also in this position no error was found. But now an idea occurred to me. I thought, when I let the man point to my finger several times in the unusual position with the supinated forearm, and then suddenly pronate the forearm to the position with the palm downward, and now let him quickly point to my finger again, it might be, that at first the cerebrum would forget to correct the cerebellar defect, and therefore a spontaneous error to the outside would appear. When I came to the clinic (December 19, 1910) again I made this examination with the result which I had theoretically expected. There was a very big spontaneous error to the outside when the left forearm suddenly was pronated; on the right side no effect was obtained by the same manœuvre. Though naturally only a very small clinical fact had been brought out by this reasoning, I still was very contented, because it showed that I was on the right way to understand the cerebellar mechanism.

For diagnosis I had not gained anything by this discovery. I thought that perhaps a small encapsulated abscess might still be present, which did not destroy much, but only pressed the fibres aside. I therefore proposed to the patient to free the dura mater of the cerebellum of the left side behind the petrous bone and to look if the dura was changed. If the appearance indicated an abscess an incision should be made; if the dura should be found intact, nothing more would be done. This operation was performed. The dura was found completely normal. No incision was made. After the operation the patient felt excellently.

At first I was astonished, but then understood it. It was the effect of decompression and of the loss of blood, which in increased intracranial pressure always acts beneficially. Five days after the operation I was enabled to examine the man on the turning chair. To my great surprise all the reactions were completely normal: no spontaneous error in the wrist, normal reaction to the inside. I was obliged to conclude, that if an abscess or tumour was present, this could not have destroyed anything at all, but only acted by pressure. Some days later the old troubles commenced suddenly again, the headache, the dizziness, the vomiting, and at this moment the error in the left wrist was again present, the reaction to the inside was missing in the position with the palm downward. Several days later the patient died suddenly. The autopsy showed a big cysticercus of the fourth ventricle, which probably from time to time had occluded the foramen of Magendie. For the cerebellar symptoms of the wrist no explanation could be found. A microscopical examination could not be obtained.

Though the clinical facts had not been explained by the autopsy, I still learned very much from this case. I saw that complete paralysis of certain reaction movements can persist for weeks and suddenly give place to normal function; I saw that pressure can produce all the symptoms of destruction. This new notion had become important in the elucidation of a case, which was under my observation at the same time. It was a girl (Sche.), aged 20 years, who on account of chronic otitis and labyrinthitis had been operated upon. A radical and labyrinth operation had been performed. After this operation she did not recover as usual. She still had headache, she vomited from time to time and felt dizzy. The examination showed at first that nystagmus, rotary and horizontal, of moderate degree was present towards the diseased side. The fundus of the eye was normal. No other neurological findings were present. When I examined the pointing tests I found that the left arm and wrist acted normally; the right arm was also normal, but the wrist made a big pointing error to the outside, which was present without any artificial help. In the position with the palm upward no pointing error was found. I tried the reactions by syringing the left ear with cold water and got a normal reaction to the left, in the right wrist, with the palm upward, but no reaction with the palm downward. The left wrist and both arms reacted normally. The next day a very interesting and important change had taken place. The temperature on the previous evening was 38°. Severe headache in the parietal region was present. Testing the pointing I found the same error in the right wrist as the day before, but this day also the right arm pointed to the right spontaneously. Also this error only was present in the position with the palm downward and no error was present when the palm was directed upward and the arm therefore was supinated and rotated outward. Syringing the left ear with cold water gave no reaction in the right wrist to the left, the right arm is not mentioned in my notes; the left side reacted normally. Syringing with

hot water gave completely normal reactions to the right in both arms and wrists. Severe dizziness and nausea were produced. A third examination was made on the next day. This day the pointing error in the right arm, elbow, and wrist was present to the right in all positions of the joints. No dysdiadokokinesis was to be perceived ; also she did not point correctly to her own nose with the right fore-finger. From the progress of all the symptoms we thought that a cerebellar abscess in the posterior fossa might still be present. The patient was therefore again immediately operated upon by Dr. Ruttin (January 2, 1911). After incision of the dura in the posterior fossa in front of the sinus, near the place, where the labyrinth had been before, a large quantity of clear liquid came out. Several incisions in the cerebellum produced no pus. When I saw this large quantity of liquid escape, I immediately thought that a circumscribed serous meningitis or encephalo-meningitis had been the cause of the patient's symptoms. After the operation the patient felt much better, and as the pointing error slowly disappeared, recovered. But also after the complete healing the pointing reaction to the inside was absent in the right wrist. I thought that the incisions had injured the centre for the wrist. The observation of this case was not completed when another case (W., January 3, 1911), whose labyrinth had been operated upon, came to me, suffering from dizziness and headache on the diseased side. The functional test showed little spontaneous nystagmus, the right arm pointed to the right in the position with the palm downward (in the arm, elbow, and wrist), whereas in the position with the palm upward normal pointing was present. There was no reaction of the right arm and wrist to the left in the position with the palm down. The other position was not examined. In this case nothing was present to indicate a cerebellar abscess. I thought that in this case also there was probably an accumulation of cerebro-spinal fluid behind the dura, which in the labyrinth operation had been set free, and that this accumulation of fluid by its pressure produced the symptoms. A second quite analogous case (K.), like the above described, I saw a month later (February 16, 1911). It was a man whose left labyrinth had been operated upon and completely healed and epidermised. This man came and complained of headache in the left posterior fossa and slight dizziness. The examination showed no spontaneous nystagmus, but a big pointing error of the left arm to the left. The wrist made no error. Here I only thought I had to do with a circumscribed accumulation of liquid, pressing on the cerebellum. The next important and interesting case—regarding my symptom-complex—was observed first on May 15, 1911. It was a boy (Z.), aged 12 years, upon whom a radical operation had been done. The dura of the left posterior fossa had been exposed, because an extradural abscess was present. The labyrinth was normal. This boy, after the operation, did not feel well, he had attacks of dizziness and headache on the left side of the skull. The examination showed a mild degree of nystagmus to the diseased side, the pointing test gave a constant error

to the left in the left arm, elbow, and wrist. Cold irrigation of the right ear produced a strong nystagmus to the left, the right arm showed a normal reaction to the right, but the left arm was not influenced. As the headache and vomiting had already persisted several days, the boy was operated upon on the 15th. I did not expect a cerebellar abscess, because the whole aspect was not so severe as it usually is in any abscess-case. The boy was not sleepy. When he temporarily had no headache he laughed and played with the other children. At the operation (Dr. Bondy) after incision of the dura in front of the sinus, but behind the labyrinth, a great quantity of clear liquid escaped, but no pus was found. This case showed that, without preceding labyrinthitis or labyrinth operation, a circumscribed accumulation of liquid may take place in the posterior fossa and produce severe symptoms. After the operation the boy soon recovered. Also in this case first the pointing error disappeared for the position, ' palm upward,' in the arm and wrist, and later on, in the arm for the position ' palm downward '. In the wrist the reaction in the position ' palm downward ' never came back even after complete healing. This boy several weeks later had a relapse with the same symptoms. After lumbar puncture and withdrawing 5 c.c. of fluid all the symptoms disappeared again.

When we consider the long list of cases here related these cases have taught me the following facts :

1. Case S. Severe attacks of dizziness with nystagmus, impaired caloric reaction, complete deafness, very severe headache in the posterior fossa of the diseased side behind the ear, recover after lumbar puncture completely. The hearing and the caloric reaction may become normal again. Hysteria cannot be the cause of this disease, as hysteria cannot make an impaired caloric reaction and cannot produce attacks of nystagmus.

2. Case Schi. Attacks of dizziness, headache in the posterior fossa of the diseased side, hard hearing may suddenly recover with a feeling that something in the posterior fossa behind the ear has burst. Dr. Breuer believes a cyst in the cerebellopontine angle might be the cause of the disease.

3. Horsley's cases showed me that a certain localization is present in the cerebellum, which may be detected by the vestibular tests, especially by the falling and pointing test.

4. Case V. A cerebellar abscess is diagnosed only by the pointing test. From this case is learned that in the cortex of the hemisphere of the cerebellum four centres are present for the pointing in the different directions, to the right and left, up and down. In every one of these centres all the muscles and joints are represented. The centre for the movement of the wrist to the inside is situated immediately behind the labyrinth. When this centre is paralysed, there exists no reaction to the inside in the position ' palm upward ' and ' palm downward '.

5. Case S. (1) again examined, reminded me suddenly of case Schi (2).

The examination of the movements of the wrists gave no clear result. The idea that in this case a cyst filled with liquid produced the severe symptoms, which disappeared again when the cyst was evacuated, is very striking in the first moment, but is rejected, being too keen a hypothesis. Still, the idea that a process not in but behind the labyrinth in this and other similar cases might be present has taken ground and sharpens my attention for every suspected case.

6. Case E. shows that complete paralysis of reaction movements may be temporarily present, produced by pressure of cerebro-spinal fluid. It shows that, if in the wrist no reaction to the inside is present in the position with the palm downward, and a normal reaction in the position with the palm upward, a spontaneous error can be demonstrated by suddenly turning the wrist from the supinated to the pronated position. The case showed that, after the decompression operation, this symptom may completely disappear, but with the return of pressure appear suddenly again.

7. Case Sche. showed that all the symptoms of a cerebellar abscess may be produced by an accumulation of liquid in the posterior fossa behind the labyrinth. In the beginning only the error in the wrist in the position with the palm downward is present and the reaction to the inside is absent in this position. With the progress of the symptoms this error is to be seen in all the joints and all the positions of the upper extremity, and all the reactions to the inside are lacking. After healing only the reaction in the wrist does not come back. Very probably the incision had destroyed this centre.

8 and 9 are two cases of healed labyrinth operation where the same symptoms, that is, the error in the upper extremity, are present. Only the other symptoms are so meagre that the question of cerebellar abscess does not arise and the explanation is given that an accumulation of liquid in the posterior fossa is the cause of these troubles.

10. The case Z. shows that also, without labyrinth disease, the same symptoms may occur. The operative examination reveals the accumulation of liquid.

In addition to these personal experiences, I was influenced by a paper of Dozent Dr. Robert Bing, of Basle, which was kindly sent to me by the author in March 1911, entitled ' Die Meningitis cystica serosa der hinteren Schädelgrube ', *Medizinische Klinik*, 1911, Nr. 6. In this paper the author first gives a short *résumé* of the literature of this disease. ' In the monograph of Friedrich Schultze, on disease of the meninges, 1901, no analogous finding is reported. It was first mentioned by Strœbe in his text-book on the pathological anatomy of the nervous system, 1904. Strœbe says : It is probable that accumulations of liquid can remain in the soft meninges, where inflammatory processes have previously been present. There adhesions between the arachnoid and the pia are formed, by which the communication of liquid is made impossible. Hence the liquid accumulates in this space and even in rare cases can

exert a pressure on the subjacent brain tissue.' The clinicians first described such cysts in the spinal cord. In 1907 Placzek and Krause[1] first published a case of circumscribed serous meningitis in the posterior fossa. Then a paper of E. Unger[2] appeared, describing a case of circumscribed syphilitic arachnitis in the region of the right cerebellopontine angle. The next is a case of Oppenheim and Borchardt,[3] 1910, where very severe symptoms were completely healed by opening a cyst on the inferior surface of the right cerebellum. Analogous cases of cysts of the cerebellopontine angle have been described by Frazier and Finkelstein, and Borchardt mentioned a second case upon which he had operated. After this review of the literature Bing describes his own case. It is a man who previously had suffered from an otitis media suppurativa, which healed perfectly. A year ago severe cerebellar symptoms developed. The patient was operated on and a cyst corresponding to the cisterna cerebello-medullaris was found, which afterwards healed.

The most important point for me in this paper was the knowledge that circumscribed adhesive meningitis of the cerebellopontine angle already had been found several times, causing severe symptoms by pressing on the nerves in the cisterna pontis and the cerebellum.

These were the stones from which the new building had to be formed. The courage and the mental energy which are necessary for such a building were given to me by the success which I had in diagnosing several difficult cases of cerebellar lesions and by the complete success of the cooling experiment on the cerebellum, which I had made several times in some cases in Vienna and in one case in Frankfurt. Especially by this last experiment I was encouraged very much in the belief that so far my reasonings concerning the cerebellar localization and the whole cerebellar mechanism were correct.

At this time a case came to the clinic with the following history. It was a girl, aged 20 years, who in December 1910, had suffered from an acute otitis media on the right side, which healed. After the healing of the otitis she got attacks of dizziness, headache in the right posterior fossa, especially behind the ear, hard hearing and tinnitus on the right ear. The investigation showed a normal drum, pain on pressure behind the ear in the region of the mastoid emissary vein, but no swelling. The hearing was diminished in a manner characteristic of an inner-ear lesion, the caloric irritability was also diminished on the right side. There was a big pointing error to the outside, when the wrist was turned from the supinated into the pronated position. A slight diminution of the right corneal reflex was present, no other neurological finding. This case naturally reminded me of Case 1 (S.) and also of the cases Sche., W., K., and Z. Especially the last case, which at this time was under my observation and in which, without a preceding labyrinthitis, the symptoms of

[1] Placzek und Krause, *Berl. klin. Woch.*, 1907, No. 29.
[2] Unger, E., *Berl. klin. Woch.*, 1909, No. 5.
[3] Oppenheim und Borchardt, *Deutsche med. Woch.*, 1910, No. 2.

circumscribed disease in the posterior fossa had developed, made me think that in the case of this girl a similar condition might be present. After having observed the girl more than a month, during which time the symptoms changed a little but not considerably, I presented her to the Neurological Society on June 13, 1911, and said: We have here a symptom-complex which I have already observed in more than twelve cases. (All the cases are not mentioned in this paper.) As to the pathological cause of this symptom-complex I must reserve my opinion. But I think it is probable that we have to do with an accumulation of liquid in the cisterna pontis, with a cystic serous meningitis, as Bing has recently described in fully developed cases. I had given my opinion about the causation of this symptom-complex more modestly than I really felt it, knowing that the greater part of my audience did not agree with me, and seeing the impossibility of showing them the way in which I had come to my conclusions. Some days later, in consequence of my diagnosis, I made a lumbar puncture. There was no increased pressure and only 10 c.c. were withdrawn. The effect of the lumbar puncture was enormous. The patient had a very severe headache in the right posterior fossa, continual vomiting, dizziness, and felt miserable. As these symptoms did not improve, after six days another operation was performed. With the idea that I had to do with an increased pressure in the right posterior fossa, I freed the dura of the posterior fossa, taking away a part of the mastoid wall in front of the sinus, but without opening the antrum. Then I gently massaged the dura by putting tampons between the dura and the bone. The wound was then closed. The effect of the operation was marvellous. Headache, vomiting, dizziness ceased immediately. Then I did not see the patient for almost two months, having gone for my vacation. When I came back the hearing had considerably improved and a fortnight later it was completely normal on the right side. Owing to this result, which after the history of the first case (S.) I had expected, I was much strengthened in my opinion about the diagnosis of this case. Paying very much attention to this symptom-complex, I detected several other cases in the rich material of the clinic, and also in my private practice four cases came under my observation with similar symptoms. In several cases only from the anamnesis, from the description of the attacks of dizziness, the hard hearing, tinnitus and headache over one side of the posterior fossa, I diagnosed that the pointing error in the wrist must be present. I made the first examination before different witnesses and my diagnosis turned out to be correct.

Though the clinical picture now seemed to me quite sure, the pathological cause was not yet clear to me. In the cases mentioned by Bing a complete cyst had been found. In my cases such a cyst could not be present, because the symptoms were often changing without treatment, but especially with lumbar puncture. It was necessary to assume that a circumscribed increased pressure was present. But how is it possible that the pressure remains circumscribed on one side of the posterior

fossa ? Thinking over this problem, I came to the conclusion that this circumscribed increased pressure could only be understood if an isolated secretion of cerebrospinal liquid could be found in the cerebellopontine angle. Now it came to my mind, that what I had to do was to study exactly the anatomy of the cerebellopontine angle. Here I soon found the classical description and images which Key and Retzius give in their book, *Studien in der Anatomie des Nervensystems und des Bindegewebes*, 1875, vol. i, and Retzius, *Das Menschenhirn*, Stockholm, 1896. There I found that immediately on the side of the glossopharyngeal and vagus nerve in the cerebellopontine angle the plexus choroideus lateralis comes out from the foramen Luschkæ. It is often adherent to the flocculus, and the opening of the foramen Luschkæ is often narrowed considerably by adhesions. Immediately in front of the plexus the facial and acoustic nerves enter into the inner meatus. The lateral choroid plexus lies with its villi in the subarachnoidal space of the cisterna pontis lateralis, as Key and Retzius call it. The borders of this cisterna, according to these authors, are as follows : 'Along the inferior margin of the pons a very thin membrane originates, from the bottom of the furrow, which separates medulla and pons, is continued laterally on the glossopharyngeal and vagus nerves and runs near the middle line to the basilar artery. This membrane forms the inferior border of the lateral cisterna pontis. From here a stronger membrane on both sides originates from the basilar artery, which separates the lateral cisterna from the median. The lateral cisterna reaches the upper margin of the pons, but very often is separated into an anterior and posterior half by a membrane, which runs from the trigeminal nerve to the median cisterna. Laterally and posteriorly this cisterna is in connexion with the cisterna magna cerebello-medullaris (in the region of the flocculus). In the lateral cisterna the vagus and glossopharyngeus are found (which also belong to the cisterna magna), then the facial, the acoustic, the abducens, and trigeminal nerve.' After this description of Key and Retzius it is clear that the lateral and posterior boundaries are very defective. Naturally, under normal circumstances the liquid can communicate also through the slits of the above-described thin membranes with the cisterna media and the other subarachnoidal spaces which lie on the base of the brain in front of the pons. The most important part of this description for me was, that, as I had theoretically expected, an autochthonous secretion of liquid really takes place in the lateral cisterna of the pons. To explain my findings I had only to suppose that in the cases which I had observed adhesions between the arachnoidea and the pia are present, which hinder the free circulation of the liquid. It was not difficult to imagine that in a still existing or preceding otitis a mild infection of the meninges of the posterior fossa had taken place, which led to the formation of such adhesions. Especially in the case of Sche., of K. and W., and of Z., this explanation was very probable, and in Sche. and Z. directly proved by the operation. Also in the case of cysts of the cisternæ mentioned by Bing such adhesions were

found at the operation. The cause of the cysts had been otitis media, syphilis and trauma. The cysts mentioned in Bing's paper were in my opinion only the last stage of a process, which must naturally be more frequent at its inception. This was the point of view when I held my demonstration in November 1911. In the winter 1911–12 quite a number of cases were added to my list, and at the German Otological Congress in May 1912 I could speak on my experience of 30 cases. The number of cases, which I have seen, amounts up to the present time to more than 40 cases. Since October 1912 I have had no ear material at my disposal and therefore the number of cases only grows slowly. Still I had the opportunity of observing some new things in this respect, and the following remarks are given from my present standpoint.

When in a completely closed vessel, which is filled with liquid, a secretion of liquid takes place, it is clear that this can only happen if exactly the same amount of liquid is absorbed and removed from the vessel, as is secreted in it, because the liquid is incompressible. If only the slightest disproportion between secretion and absorption is present, the pressure will rise in the vessel, because the liquid is incompressible. The effect of the rise of the pressure will be inhibition of the secretion, and in that manner the equilibrium between secretion and absorption will be controlled. The skull, together with the spinal canal and the eyeball, are such vessels but with a certain difference. Both are supplied with blood-vessels, which lead the blood to and from the vessel. If by any cause a disproportion between secretion of the liquid and its absorption should be established, the next effect of the rise of pressure will therefore be a compression of the veins and of the capillaries. In the eye the compression of the vertex veins makes a venous hyperæmia of the retina and the anterior ciliary veins. If the degree of compression is greater the arteries are compressed. By the stasis of blood an œdema is produced. Later the increased pressure leads to the excavation of that part of the eyeball least resistant to pressure, that is the lamina cribrosa of the optic nerve. In childhood the whole eyeball can give way, a hydrophthalmus being developed. In the skull quite analogous things must happen if from any cause a disproportion between secretion and absorption is established. First, the veins must be compressed, it is only a question where. As in the first stage of increased pressure the veins on the surface of the brain are more filled (Kocher, Cushing, Hauptmann [1]), it must be supposed that here also the first effect is that the veins which lead out of the skull are compressed. Later the veins on the surface are compressed, and then follow the capillaries and the small arteries. By the stasis in the veins, œdema of the cerebrum will be produced, followed by atrophy. If the skull is not yet closed, the sutures will be loosened and the skull will be enlarged. These reasonings for the rise of general pressure in the

[1] Kocher, 'Hirnerschütterung, Hirndruck,' *Nothnagels Path.*, Abt. 9; Hauptmann, 'Untersuchung über das Wesen des Hirndruckes,' *Zeitschr. f. d. ges. Neurolog.*, Bd. xiv, 1913, p. 313.

skull may also be applied to the cisterna pontis lateralis alone. Also this cisterna, together with the labyrinth, may be regarded as a closed vessel. In the normal this vessel communicates freely with the other subarachnoidal spaces. But if this communication by formation of adhesions is more or less reduced, the vessel represented by the cisterna plus the labyrinth becomes more or less closed. It is theoretically clear that even if a complete occlusion from the adjoining spaces is present, no increased pressure must be the consequence. This increased pressure will only arise at the moment when a disproportion between the secretion and absorption of the liquid in this vessel takes place. From the practical point of view it is very probable that by the formation of adhesions at the same time the disproportion between absorption and secretion is established, as this process will cause the occlusion of some of the capillaries which are necessary for absorption. But until microscopical evidence is adduced we can believe that this must not be the case. Surely the formation of adhesions is very dangerous for the establishment of this disproportion. Before these adhesions were present, if by any cause the plexus choroideus lateralis was secreting more liquid, this liquid could easily be distributed to a great surface and in that way absorbed. Now in every instance of secretion this absorption must take place at the place where the liquid is secreted. The analogy between glaucoma and this disease is clear. Every cause which leads to an attack of glaucoma will probably also be able to produce an attack of the symptoms which originate from an increased pressure in the cisterna pontis lateralis. Therefore every psychical excitement, excess in alcohol or constipation may lead to an attack. The causes which produce the adhesions also may be different. Otitis media, especially with labyrinthitis, extradural abscess of the posterior fossa, and sinus thrombosis, may very probably, by mild infection of the cisterna, lead to adhesions. Also without these complications in otitis media an acute serous meningitis may develop and cause the adhesions. In syphilis Ravant, Wechselmann, Knick and Zalociezky, Assmann,[1] have made it very probable that already in the secondary stage an infection of the cisternæ at the base of the brain is often present. From this serous syphilitic meningitis adhesions easily may develop. Trauma of the skull with hæmorrhages in the cortex of the cerebellum may easily lead to such adhesions. I have seen several cases where after a trauma of the skull these symptoms have developed. Influenza, angina, not infrequently lead to serous forms of meningitis. I saw two cases where these symptoms had developed in that way. The cause of hereditary migraine is not yet clearly found out. But it is highly probable that the attack of migraine is caused by the sudden establishment of a disproportion between the secretion and absorption of liquid. I saw several cases with my symptom-complex where previously

[1] Wechselmann, *Der gegenwärtige Stand der Salvarsantherapie*, ii, Berlin, 1912; Knick und Zalociezky, *Berl. klin. Woch.*, 1912, No. 14 u. 15; Ravant, *Présse méd.*, 1912, No. 18; Assmann, *Berl. klin. Woch.*, 1912, pp. 2346–2414.

typical migraine attacks had been present, or where one member of the family suffered from my symptom-complex, others from typical attacks of migraine. Schüller believes that the disproportion between the size of the skull and the size of the brain gives a predisposition to migraine. The brain is too big for the skull. It is clear that the bigger the brain is, the less room there is for liquid, and the easier an excess of secretion may lead to increased pressure, because the absorption cannot keep pace with it. In one of my cases Schüller, from the Röntgen-plate, without knowing my diagnosis, came to the conclusion that this man should suffer from migraine of the posterior fossa. In this case the posterior fossa was abnormally flat. Flatau in his book on Migraine, 1912, mentions that ear symptoms, dizziness, tinnitus, hard hearing, are not at all infrequent in migraine.

What will be the effect of an establishment of a disproportion between secretion and absorption of liquid in the cisterna pontis lateralis, when by adhesions the free circulation is made impossible ?

The effect will first be an increased pressure in the cisterna. By this pressure the communication between the fourth ventricle and the cisterna will be closed hermetically, the plexus choroideus lateralis closing the foramen Luschkæ like a valve. Then the brain will press all the movable liquid away from the neighbourhood of the cisterna, thus occluding the cisterna still more. In that manner a vicious circle is established, and it can be understood that once established, this condition may persist for a long time, for weeks and months, as I have observed. Healing will take place if the pressure is raised sufficiently to inhibit the secretion or if the adhesions are ruptured by the pressure.

The symptoms of this disease depend upon the organs which are included in the vessel. These are : the labyrinth, which communicates with the cisterna first by the fibres of the acoustic nerve ; if the aquæductus cochleæ also communicates with the cisterna, we do not know. The trigeminal nerve, especially its ramus recurrens meningeus, the facial, the glosso-pharyngeus and vagus are also included. From the side of the facial I have not seen any symptoms. It is known that this nerve is much more resistant than the acoustic. Also the abducens never was affected. It might be that the ear symptoms are not due to pressure on the nerves, but on the peripheral organ. Symptoms from the side of the glosso-pharyngeus in some of my cases seemed to me probable, but it is very difficult to make these findings sure. The vagus often is affected, but it may be that the action is not a direct one, but is due to a reflex from the vestibular nerve on the vagus nucleus. Pains from the trigeminal nerve are often present, the special kind of headache present in my cases is probably due to the tearing of the nervi recurrentes vagi, which leave the skull with the mastoid emissary vein. This is the place which is usually tender on pressure in my cases. The pointing error very probably is due to pressure upon the surface of the cerebellum. The centre for the wrist is very probably situated in the cisterna,

being very near the inner meatus. I do not know where the nerve-fibres to and from this part of the cortex run. Therefore, I will not exclude the possibility that perhaps the pressure on the brachium pontis, which is situated partly in the cisterna, is the cause of this symptom. Symptoms of compression of the pons in a very weak degree were present in several of my cases.

Frequently my symptom-complex is combined with hysterical or neurasthenic symptoms. I believe that, though the seat of hysteria and neurasthenia is not yet established, one will not assume that the vestibular nerve or the cerebellum gets hysterical. The attacks of nystagmus which I have often observed, the diminished caloric irritability, or the pointing error, cannot be of hysterical or neurasthenic origin. I consent, if one assumes that in cases with a hysterical or neurasthenic predisposition secretory disturbances may be elicited easier, but it is still questionable if the hysterical or neurasthenic symptoms are not provoked by the disease which leads to my symptom-complex, and therefore that hysteria and neurasthenia are not to be counted under the causes, but under the consequences, of this disease.

The treatment of the disease may be carried out in different ways. We may try to rupture the adhesions, we may try to inhibit the secretion, we may make room for the brain, so that even in increased pressure the effects cannot develop, because the brain can give way. To obtain the first result we may try different methods: (1) We can operate and tear the adhesions. In cysts, which were completely closed and therefore produced all the symptoms of a brain tumour, this was readily done. But in my cases we have not to do with a complete occlusion. Therefore, such a treatment would be a too heroic one. (2) We can try to increase the pressure in the cisterna and in that way cause the pressure to rupture the adhesions. Such an increase of pressure can probably be produced by alcohol. One of my patients, who during such an attack was obliged to drink alcohol, felt all the symptoms get worse, but suddenly he felt a sensation that something in the posterior part of the skull was rupturing and all the symptoms disappeared. Lumbar puncture, which was first recommended by Babinski against vertigo, tinnitus, and hard hearing, seems to act in the same way. Babinski already says that the best result is to be expected in the cases where severe headache appears after the lumbar puncture. In the cases with my symptom-complex extremely severe headache after the lumbar puncture appeared. How does the lumbar puncture act upon the cisterna? It is impossible that the liquid which is withdrawn comes directly from the cisterna. The liquid which is withdrawn must be replaced at the same moment by some other liquid, and as the cerebro-spinal liquid cannot be secreted so quickly, only an increase in the size of the veins can replace the liquid taken out. This swelling of the brain by the increased size of the blood-vessels must make the occlusion still more perfect and probably gives rise to an increased production of cerebro-spinal liquid.

Hence, the first effect will be an increase of the symptoms. If now by the increased pressure the adhesions are ruptured or if only the secretion is inhibited I cannot say. In some cases, where suddenly all the symptoms disappeared and did not come back for years, I am inclined to think that adhesions have been ruptured. The best remedy to inhibit the secretion of the liquid would be the extirpation of the plexus choroideus. In the cisterna lateralis this is impossible, but might it not be possible as a treatment of ventricular hydrocephalus to ligate and extirpate a part of the plexus choroideus of the cerebrum or of the fourth ventricle?

In our cases the inhibition of secretion might be tried by internal remedies. I have tried atropin and calcium lacticum, but until now without any definite result. The last way, and one which in my opinion should not be neglected, is to do a decompression operation by freeing the dura of the posterior fossa. I have done this in three cases with good results. In one case I even made an incision through the dura in the posterior fossa as the simple freeing had not a lasting effect. After the incision the case healed completely.

It is very important that in all cases not all the symptoms are completely developed. I have seen four rudimentary cases in which many of my symptoms were not present. In one case only tinnitus was present for two months, and then suddenly all the other symptoms developed; in another case only tinnitus and hard hearing were present. Here, after the lumbar puncture which was made for a diagnostic purpose, all the symptoms healed. In a third case no headache and no pointing error were present. The enormous change of the hearing in an inner-ear lesion without lues helped me to make the diagnosis. In this case the lumbar puncture revealed a pressure of 380 mm. in the horizontal position. It was a case of general increase of pressure; still no headache existed. In a fourth case also the enormous change in hearing in a non-hysterical man made the diagnosis. No headache and no pointing error were present. In two cases I saw all the symptoms of my complex occur in otosclerosis (fixation of the stapes). It is naturally possible that this symptom-complex also develops in cases with other ear diseases. Whether a connexion between the development of otosclerosis and this symptom-complex exists, I cannot say, but I do not believe it. The frequency of cases which very probably belong to this class is great. Therefore, and because by a harmless treatment we can often heal these cases perfectly, I found it necessary to draw your attention to this subject.

SECTION XVI

OTOLOGY

INDEPENDENT PAPER

LOCAL ANÆSTHESIA OF THE DRUM-HEAD

By N. Rh. BLEGVAD (Copenhagen)

Two years ago I brought before the notice of the First Scandinavian Congress for Oto-Laryngology a new solution for local anæsthesia of the drum-head. My starting-point was, that in order to influence the sensitive nerves of the membrana tympani it was necessary to destroy the stratified epithelium covering the membrane, which under normal conditions is impervious to fluids, as Lea Steinschneider has demonstrated. Such a destruction of the epithelium takes place also in the case of the anæsthetic solutions which have hitherto proved to be effectual. In the case of Gray's fluid it is the aniline oil, and in Bonain's solution the carbolic acid, which produces this effect. My reason for being dissatisfied with the solutions mentioned was, that their use was not unattended with risk. After the use of Gray's fluid aniline poisoning has been observed, which is not to be wondered at, as the solution contains a proportionately large quantity of highly toxic aniline oil, and in my experience, on more than one occasion, necrosis of the drum-head has supervened after the use of Bonain's fluid, whilst medical literature also records cases of unpleasant effects following the use of this solution, which would seem to be in the nature of things when one bears in mind that it contains 33 per cent. of carbolic acid, which possesses highly corrosive properties. So instead of carbolic acid I used salicylic acid, which is a powerful solvent of horn, and is therefore able to render the epithelium of the drum-head permeable to cocaine. The reason why I again beg to call attention to this solution is, that in the course of time I have modified it somewhat. Its composition is now as follows :

Hydrochloride of cocaine	āā	1 part
Salicylic acid		
Rectified spirit		2 parts

To this solution is added, just prior to use, a little 1 per cent. adrenalin. It has been evident that the solution would not keep when adrenalin was dissolved in it. The fluid is used in the following manner : a very

small pad of cotton wool is saturated thoroughly with the fluid, a drop of adrenalin is then poured over it and the pad is at once applied to the tympanic membrane at the place where the incision is to be made. Then about ten drops of the solution, plus a little adrenalin, is dropped into the external auditory canal; the latter is closed with cotton wool, and the patient is laid down with the ear which has to be treated uppermost. At once he feels a slight pain in his ear, but this quickly disappears. After twenty minutes the pad is removed, and the drum-head has become anæsthetized. Sometimes there is a white, purely superficial eschar at the place where the pad has lain, but no deep-seated necrosis of the membrane takes place, as the corrosive action of the salicylic acid is much less than that of carbolic acid. There can be no question of toxic action, even in children, in the case of the small doses of salicylic acid we have here referred to, nor is poisoning by cocaine at all probable, as the fluid only contains 25 per cent. of cocaine, and only a small quantity is used. One great advantage with this solution is its disinfecting action, salicylic acid being known to be a powerful disinfectant, stronger than carbolic acid. It has also been evident to me that cases of acute otitis media, where paracentesis of the drum-head has been made after the use of this fluid, have had a shorter course than is usually the case. I have performed a number of minor operations with this solution in children. The most successful is paracentesis of the tympanic membrane, which can be effected quite painlessly provided one (1) has a very sharp and pointed paracentesis knife, and (2) does not touch the medial wall of the tympanum. Moreover, the fluid may be used in the removal of polypi of the ear, as well as for incision of abscesses of the external auditory canal.

SECTION XVI

OTOLOGY

INDEPENDENT PAPER

OTOSCLEROSE (DEMONSTRATION MIT DEM PROJEKTIONSAPPARAT)

Von Prof. Dr. G. BRÜHL, Berlin

M. H.! An Hand des von mir selbst untersuchten anatomischen Materials möchte ich mir erlauben, Ihnen einen Ueberblick über die histologischen Veränderungen zu geben, welche wir bei der eigentümlichen Erkrankung des Felsenbeins finden, die in einem grossen Teile der Fälle zu einer Ankylose des Steigbügels und zu dem klinischen Bilde der *Otosclerose* führt. Wir werden kennen lernen :

1. Knochenalterationen im Felsenbein, die weit fort vom Vorhofsfenster sitzen und in keinerlei Beziehung zu dem Steigbügelgelenke treten.

2. Knochenalterationen, die in der Nachbarschaft des Steigbügels, und zwar regelmässig am *vorderen* Rande des Vorhofsfensters sitzen, ohne den Steigbügel zu überwuchern.

3. Knochenalterationen, die zur Ankylose des Steigbügels führen.

Die letzteren Knochenalterationen, die entweder isoliert im Vorhofsfenster sitzen oder gleichzeitig neben anderen multiplen Herden im Felsenbein vorkommen, sind allein diagnostizierbar und verlaufen unter dem klinischen Bilde der Starrheit der Paukenfenster, der *Otosclerose.*

Ich zeige Ihnen nun zunächst die Vorhofsfenstergegend des Menschen unter normalen Verhältnissen. Sie können zwei wichtige Tatsachen aus den Präparaten erkennen :

1. Die grössere Breite des Ringbandes des Steigbügels am vorderen Pol des Vorhofsfensters (Präparat 1).

2. Die unmittelbare Nachbarschaft des Musculus tensor tympani (Präparat 2) zum vorderen Umfang des Vorhofsfensters.

In der zwischen diesen beiden Punkten gelegenen Knochenpartie finden wir eine Prädispositionsstelle für Knochenumbildungen, die ihre Erklärung finden mag in den reizenden Momenten der unaufhörlichen Zerrung des vorderen Pols des Ringbandes am Fensterrand und in dem Scheuern der Sehne des Tensor tympani an derselben Stelle des Knochens. Die funktionelle Anpassung des Ringbandes an die Funktion des Hörens

zeigt ein Steigbügel des menschlichen Embryos (Präparat 3), bei dem das Ringband vorn und hinten die gleiche Länge hat. Erst während des Hörens nach der Geburt kommt infolge stärkerer Inanspruchnahme des vorderen Ringbandpoles auch die grössere Breitenentwicklung desselben zur Ausbildung.

Den normalen elfenbeinernen Charakter der Labyrinthkapsel und ihren Aufbau aus zwei Schichten zeigt Präparat 4.

Dagegen sehen wir im Präparat 5 bereits pathologische Verhältnisse: eine schwammartig durchlöcherte spongiöse Knochenpartie an der Schneckenspitze bei normal beweglichem Steigbügel, und einen ähnlich grossen Spongiosierungsbezirk an der Schneckenbasis, dem Felsenbein der anderen Seite, im Präparat 6.

Die Bildung einer spongiösen hyperostotischen Wucherung in der Nachbarschaft des Vorhofsfensters, und zwar an der typischen Stelle *vor* dem Vorhofsfenster, aber bei beweglichem Steigbügel, finden wir in den Präparaten 7, 8 und 9 als Zufälligkeitsbefunde.

Ich beginne jetzt mit der Demonstration der wichtigsten Fälle, in welchen die Knochenalterationen zur Steigbügel-Ankylose geführt haben. Es ist selbstverständlich, dass man die Diagnose ‘ Stapesankylose ’ zu Lebzeiten nur dann stellen kann, wenn der Patient noch hört. Ist infolge gleichzeitiger Labyrinthatrophie vollkommene Taubheit eingetreten, so kann man mit der Funktionsprüfung eine differentielle Diagnose nicht mehr stellen.

So stammt z. B. das nächste Präparat (10) von einer tauben Frau. Wir sehen den Steigbügel durch neu gebildeten spongiösen Knochen im Vorhofsfenster ankylosiert, und ebenso finden wir im Präparat 11, das ebenfalls von einer höchstgradig schwerhörigen Patientin stammt, den Steigbügel durch eine riesige Knochenwucherung in der Labyrinthwand fixiert und gleichzeitig einen zweiten hyperostotischen spongiösen Herd an der Basis der Schnecke.

Wir kommen jetzt dagegen zur Demonstration von *drei* Felsenbeinen, die von zwei jugendlichen Patienten herrühren, bei denen ich *zu Lebzeiten* lediglich durch die Funktionsprüfung imstande war, die Diagnose *Stapesankylose* (oder Otosclerose) zu stellen. Dass die Diagnose eine richtige war, werden Sie aus den Präparaten erkennen.

Das Präparat 12, welches vom linken Ohre einer 30 jährigen Patientin stammt, zeigt Ihnen, wie der Steigbügel durch eine Knochenbrücke fest fixiert mit dem unter der Tensorsehne gelegenen hyperostotischen Herde zusammenhängt. Besser erkennen Sie dies bei stärkerer Vergrösserung des Präparates (13), an dem Sie sich gleichzeitig von dem normalen Bau des Cortischen Organs und der Macula sacculi überzeugen können. Das Präparat 14, das von dem Ohre der anderen Seite stammt, zeigt Ihnen in ähnlicher Weise den völlig ankylosierten Steigbügel und den direkt unter dem Tensor tympani gelegenen Ausgangspunkt der Erkrankung (Präparat 15). Im Präparat 16 sehen wir die den Steigbügel fixierende Knochenbrücke in ausgezeichneter Weise. Das nächste

Präparat (17) zeigt Ihnen bei starker Vergrösserung eine Grenzpartie des kranken und gesunden Knochens. Wir erkennen, wie aus dem erkrankten Herde fingerartig Gefässe mit kolbig verdickten knöchernen Wandungen in die normale kompakte Knochensubstanz hineinziehen, und am nächsten Präparat (18), wie Marksprossen mit und ohne osteoider Kuppe und mit und ohne Ostoklasten in den gesunden kompakten Knochen vordringen. Dasselbe erkennen wir auch am nächsten Präparat (19), welches von demselben Falle stammt und einen neben dem Vorhofsfensterherd bestehenden Herd am Rande des Schneckenfensters zeigt. Die Verdrängung des alten Knochens erfolgt auf dem Wege zunächst periostaler Gefässe durch mechanische und chemische Einwirkung des in ihrer Umgebung sich neu bildenden Knochens auf den alten Knochen und durch Auflösung des alten Knochens durch Marksprossen, die mit und ohne Riesenzellbildung aus dem neugebildeten Knochengewebe herauswachsen und den kompakten Knochen geschwulstartig verdrängen. Der neugebildete Knochen macht sekundär mehrfache Veränderungen durch. Am Präparat 20 sehen wir z. B. ein grosses periostales Gefäss in die neugebildete Steigbügelbasis hineinziehen. In klarer Weise erkennen wir bei starker Vergrösserung die von seiner Wand ausgehende lakunäre Resorption des neu gebildeten Knochens. (Präparat 21).

Zum Schluss zeige ich Ihnen noch einen Fall von typischer Otosclerose, der von einem jugendlichen Manne herrührt, bei dem ebenfalls zu Lebzeiten die richtige Diagnose gestellt wurde. Sie erkennen im Präparat 22 den hyperostotischen Knochenkern, der das Vorhofsfenster verschliesst und die Steigbügelbasis ersetzt hat bei verhältnismässig geringen Veränderungen im Labyrinth, und schliesslich im letzten Präparat (23), das von demselben Falle stammt, die Stapesankylose als einzigen pathologischen Herd im knöchernen Felsenbein in typischster und ausgezeichnetster Weise.

Wenn es auch, wie Sie gesehen haben, Knochenalterationen gibt, die wegen ihrer Lage im Felsenbein der klinischen Wahrnehmung entgehen, so haben Sie doch Fälle kennen gelernt, — und das sind die praktisch wichtigsten, — in denen die Knochenerkrankung auf das Vorhofsfenster beschränkt bleibt, zur Stapesankylose führt und klinisch so charakteristische Symptome macht, dass das Zusammenfassen dieser Fälle in eine besondere Krankheitsgruppe, die typische Otosclerose (Politzer), gerechtfertigt ist. Ob für diese Fälle die von mir eingangs erwähnte ätiologische Hypothese, die eine auf Grundlage einer ererbten Disposition vorhandene Neigung zur Knochenum- und Neubildung, die *vor* dem Vorhofsfenster infolge der dort vorhandenen Reizmomente einsetzt, annimmt, zu Recht besteht, ist eine Frage, die neben dem hervorragenden klinischen Interesse der Erkrankung eine nur untergeordnete Bedeutung beansprucht.

DISCUSSION

Mr. J. F. O'Malley (London). Professor von Eicken has asked Professor Brühl how he explains the incidence of patches of otosclerosis in other parts of the temporal bone, than round the oval window, in connexion with the latter's statement that the junction of bone and fibrous tissue influences the incidence of the disease. I have recently put forward the view, when considering the analogy between osteo-arthritis and otosclerosis, that the anatomical site of election of the morbid changes in both seemed to be influenced by the juxtaposition of cartilage and bone. If this view is correct, it explains the incidence of patches of otosclerosis throughout the temporal bone, as areas of cartilage in normal cases are found elsewhere than around the oval window.

Reply

Professor Brühl (Berlin). Die ältesten Hyperoctosen sitzen vor dem Verhofsfenster und können auch den Anlass zur Knochenumbildung an anderen Stellen des Felsenbeins bilden. Jedoch erscheint mir die angeführte Hypothese nur wahrscheinlich für die Fälle von Otosclerose mit einem einzigen Herd im Felsenbein, und zwar im Vorhofsfenster. Dieselbe erscheint mir aber nicht wichtig im Verhältnis zu der praktisch wichtigen Tatsache, dass Otosclerose ein wohl fixierter, diagnostizierbarer Kranheitszustand ist.

[Friday Afternoon, August 8]

SECTION XVI

OTOLOGY

INDEPENDENT PAPER

LANTERN DEMONSTRATION ON THE PATHOLOGICAL CHANGES IN OTOSCLEROSIS

By Dr. ALBERT A. GRAY, Glasgow

Two early cases were described and illustrated.

Case 1. In the first case the patient had suffered from dulness of hearing of only eighteen months' duration, and the pathological changes were as follows:

On the right side a deeply staining focus of porous new-formed bone was found in the usual place, immediately in front of the oval window. The focus was sharply defined from the surrounding bone, and there were no signs of inflammatory activity. The focus reached the margin of the stapedio-vestibular synchondrosis, but did not extend across it into the stapes. The organ of Corti and ganglion spirale and the inner ear generally showed no evidence of pathological change. The middle ear was normal.

On the left side the changes were almost identical with those described on the right side. The bony focus extended up to, but not beyond the synchondrosis, and there was no inflammatory change. The nerve structures and other portions of the inner ear showed no pathological change. The middle ear was normal.

Case 2. The patient had been dull of hearing for two years preceding death.

On the right side the middle ear was quite normal in appearance. The diseased focus of bone was found in the usual place, just in front of the oval window. As in the first case the stapes was unaffected, the bony change not having extended across the synchondrosis. There was no sign of inflammatory activity. The curious feature about the pathological change in the bone was that the diseased focus did not stain more deeply than the normal surrounding bone, but distinctly less deeply. This condition has not, so far as the writer has been able to ascertain, been described before. The nerve-structures and other portions of the inner ear were quite normal in appearance, and the middle ear was also unaffected.

On the left side the conditions were found to be almost identical with those described on the right side. The synchondrosis was unaffected, there was no inflammatory change, and the diseased focus stained less deeply than the normal surrounding bone. The nerve-structures of the inner ear were normal in appearance. There was no abnormal pathological change in the middle ear.

SECTION XVI

OTOLOGY

DEMONSTRATION

ZUR PROGRESSIVEN FORTSCHREITENDEN SCHWERHÖRIGKEIT

Von Dr. H. NEUMANN, Wien

Aus der Reihe der demonstrierten Diapositive und mikroskopischen Präparate möchte ich zunächst diejenigen hervorheben, welche sich auf jene Form der chronisch-progressiven Schwerhörigkeit beziehen, deren pathologisch-anatomisches Substrat eine Erkrankung des Mittelohres bildet.

Zur Form der chronisch-progressiven Schwerhörigkeit gehören die Adhäsivprozesse und die mit Stapesankylose einhergehende Otosklerose.

Eine Form des chron. Adhäsivprozesses, die Lucae als postotitische bezeichnet, wird als Ausgang einer akuten oder chronischen Entzündung des Mittelohres aufgefasst, indem durch die Entzündung die ihres Epithels beraubten, gegenüberliegenden Schleimhautstellen miteinander verwachsen oder die im Laufe der Entzündung sich entwickelnden Granulationen sich zu Narben und Schwarten umwandeln, die entweder die Gehörknöchelgelenke oder die Labyrinthfenster in ihrer Beweglichkeit hindern. Diese Form des chronischen Adhäsivprozesses ist klinisch wohl charakterisiert, indem wir nicht bloss die Residuen einer stattgehabten Eiterung zu erkennen in der Lage sind, sondern der ganze Prozess trägt mehr den Charakter eines circumskript-lokalisierten an der Stelle der Veränderung selbst. Das Trommelfell ist mehr oder weniger zerstört, oder ist die einstige Perforationsöffnung vernarbt, so sind wir in der Lage aus Form und Sitz der resultierenden Trommelfellnarbe die einst stattgehabte Eiterung zu konstatieren. Auch der Katheterismus der Tube erleichtert unter Umständen die Diagnose, da dieselbe in der Regel wegsam und frei durchgängig und nur ausnahmsweise verengt ist, in welchen Fällen das Hinderniss am Ostium tympanicum tubae zu sitzen pflegt. Diese Fälle sind auch therapeutisch am besten beeinflussbar, u. zw. durch Anwendung eines künstlichen Trommelfelles oder durch die von Politzer mit Erfolg geübten intratympanalen Operationen. Dieselben bestehen in der Discission der Narben und Bindegewebsstränge und nachheriger Mobilisation. In jenen

Fällen, wo der intratympanale Eingriff nicht den gewünschten Erfolg gibt, wirkt die nachherige Applikation eines künstlichen Trommelfelles hörverbessernd. Im allgemeinen ist jedoch der Erfolg dieser Therapie nicht von langer Dauer und muss relativ häufig wiederholt werden.

In die zweite Gruppe der chronischen Adhäsivprozesse gehören jene Fälle, die nicht als Folge einer Eiterung, sondern als Folge eines Katarrhs sich entwickeln. Der sie veranlassende Katarrh kann entweder ein *akuter* sein, dann ist er in der Regel ein schleimiger, oder der schleichend verlaufende *sogenannte chronische Katarrh.* Die ätiologische Bedeutung des akuten schleimigen Katarrhes ist schon deshalb wichtig, weil er mit sehr geringer Belästigung des Patienten einhergeht und der durch ihn veranlasste chronische Adhäsivprozess Monate, ja sogar Jahre braucht, bis er das klinische Bild eines chronischen Adhäsivprozesses erzeugt. Diese Form des akuten Katarrhes hat ein ganz typisches klinisches Bild, indem er unter sehr geringen Schmerzen, in der Mehrzahl der Fälle fast ganz schmerzlos beginnt. Die durch die Schleimansammlung im Ohre bedingte Schwerhörigkeit ist im Anfange eine sehr geringe. Pathognomonisch für diese Form des akuten Katarrhes ist das milchglasähnlich getrübte Trommelfell mit sehr geringen Entzündungerscheinungen und ein kontinuierliches leicht zischendes subjektives Geräusch, welches in seinem Charakter an das Geräusch erinnert, das wir beim Ausströmen des Leuchtgases aus einer Gaslampe wahrzunehmen pflegen. Ist dieser akute schleimige Katarrh schon einige Tage älter, so ist die Schwerhörigkeit eine so hochgradige, wie wir sie in der Regel bei akut entzündlichen Prozessen des Mittelohres nur selten zu beobachten pflegen. Durch die Lufteintreibungen nach Politzer lässt sich diese Schwerhörigkeit nur wenig beeinflussen. In manchen Fällen stellt sich an die Politzer'schen Lufteintreibungen eine wesentliche Hörverbesserung ein, die jedoch schon nach einigen Stunden rückfällig wird. Der Patient hat stets das Gefühl des Verstopftseins im Ohre, sonst jedoch keine subjektiven Beschwerden. Wird in einem solchem Falle eine ausgiebige Parazentese des Trommelfelles gemacht, so sistiert sofort das subjektive Geräusch und das Hörvermögen ist auch wesentlich gebessert, bevor noch durch die Parazenteseöffnung sich Sekret entleert hat. Zur gründlichen Entleerung des gummiähnlich-fadenziehenden schleimigen Sekretes müssen ganz besondere Massnahmen getroffen werden, auf die ich jedoch hier nicht näher eingehen möchte.

Wird diese Form des akuten schleimigen Mittelohrkatarrhes in ihren allerersten Anfängen therapeutisch nicht beeinflusst, so ist nach einigen Wochen die Therapie erfolglos und der Anfang für einen langsam sich entwickelnden chronischen Adhäsivprozess gegeben.

Ob jeder nicht behandelte Fall von akutem schleimigem Katarrh als chronischer Adhäsivprozess endet oder nicht, die Frage lässt sich nicht entscheiden. Es wäre möglich, dass nur jene Fälle als chronischer Adhäsivprozess enden, bei welchen die sog. embryonalen Schleimhautfalten vorhanden sind. Sicher ist, dass eine grosse Anzahl der Patienten schon

mit dem fertigen chronischen Adhäsivprozess die Behandlung in Anspruch nimmt und in der Anamnese auf die einstige akute Erkrankung des Mittelohrs sich gar nicht mehr erinnert.

In die Gruppe der postkatarrhalischen Adhäsivprozesse gehören auch jene Fälle, die sich als Ausgang eines chronischen Mittelohrkatarrhes erweisen. Es ist schwer die Grenze zu bestimmen, bei welcher der chron. Katarrh, aufhört Katarrh zu sein, und bereits beginnt, Adhäsivprozess zu werden. In der Regel bezeichnen wir den chronischen Katarrh als chron. Adhäsivprozess, wenn die Hörstörung eine hochgradige ist und auch anderweitige Symptome, wie subjektive Geräusche, hinzugetreten sind.

Dass die meisten der chron. Katarrhe mit Veränderungen im Nasen-Rachenraume zusammenhängen, ist eine längst bekannte Tatsache. Ob jedoch in jedem Falle von chron. Katarrh ein chron. Adhäsivprozess sich entwickelt, die Frage ist noch nicht gelöst. Auf Grund meiner Erfahrungen wäre ich geneigt, die Frage dahin zu beantworten, dass zum Zustandekommen eines postkatarrhalischen Adhäsivprozesses eine anatomische Disposition des Mittelohres notwendig ist. Diese anatomisch begründete Disposition scheint mir durch jene zarte Fäden und Schleimhautduplikaturen gegeben zu sein, die schon normaler Weise im Mittelohre vorzukommen pflegen. Während Toynbee, v. Troeltsch, und Gruber sie für pathologische Bildungen hielten, betrachtet sie Politzer als Residuen des fötalen Schleimhautpolsters. Dieselben sind nicht geeignet, unter normalen Verhältnissen eine Hörstörung hervorzurufen. Wenn sie jedoch durch häufig rezidivierende Entzündungen oder durch katarrhalische Prozesse anfänglich bloss verdicken, später jedoch schrumpfen und sklerosieren, so können sie eine hochgradige Hörstörung verursachen, die umso grösser sein wird, je akustisch wichtigere Teile sie in ihr Bereich ziehen.

Ich habe Gelegenheit gehabt, Ihnen an meinen Diapositiven solche Schleimhautfalten von zarter Beschaffenheit im Bereiche des Pelvis ovalis bei einem Neugeborenen und solche bei einem 94 jährigem hochgradig Schwerhörigen von derselben Konfiguration und Lokalisation, jedoch sklerosiert und narbig verändert, zu zeigen. Wenn wir anderseits berücksichtigen, dass hochgradige Nasen-Rachenveränderungen durch Dezennien ohne wesentliche Beeinträchtigung des Hörvermögens vorhanden sein können und in einer anderen Gruppe von Fällen schon geringgradige Veränderungen des Nasen-Rachenraumes genügen, um chronische Adhäsivprozesse zur Folge zu haben, so ist es klar, dass hierzu eine bestimmte anatomisch begründete Disposition notwendig ist und dieselbe scheint mir in der Persistenz dieser embryonalen Schleimhautfalten gegeben zu sein.

Der postkatarrhalische chronische Adhäsivprozess repräsentiert jedoch nicht eine auf diese Schleimhautfalten lokalisierte Erkrankung, die ganze Schleimhaut der Tube und des Mittelohres partizipieren an diesem Prozesse. Die Folge davon ist, dass die Tube der ganzen Länge nach verengt und das Trommelfell diffus getrübt, verdickt oder atropisch, in manchen Fällen sich auch stellenweise verkalkt erweist.

Da der chronische Adhäsivprozess in der Regel eine binaurale Erkrankung ist und in manchen Familien gehäuft vorzukommen pflegt, so besitzt er eine so grosse Aehnlichkeit mit der sogenannten Otosklerose, dass nur eine genaue Funktionsprüfung und Berücksichtigung der Beschaffenheit der Tube sowie der des Trommelfellbildes die Differentialdiagnose ermöglicht.

Die Otosklerose mit Stapesankylose gehört ebenfalls zu dieser Form der chronisch progressiven Schwerhörigkeit, während die ohne Stapesfixation einhergehende Otosklerose mehr den Charakter einer Erkrankung des schallempfindenden Teiles trägt.

Denker charakterisiert in seiner im Jahre 1904 erscheinenen Monographie *Die Otosklerose* diese Form der Otosklerose folgenderweise : ' Wir verstehen unter Otosklerose eine Krankheitsgruppe, welche bei einer durchgängigen Tube und normalen Trommelfellbefunde klinisch das Bild einer progressiven, bei der funktionellen Prüfung bestimmte charakteristische Merkmale aufweisenden Schwerhörigkeit darbietet.' Als pathologisch-anatomisches Substrat für die Erkrankung hat die Untersuchung zur Bewegungslosigkeit führende, knöcherne Verwachsung des Steigbügels im Rahmen oder in der Nische des Vorhofsfensters und ferner in der Spongiosierung auslaufende Prozesse in der Labyrinthkapsel ergeben. Der Name ' Otosklerose ' wurde von Troeltsch in die Literatur eingeführt.

Der erste, der die Otosklerose als eigenartige klinische Erkrankungsform des Gehörorganes erklärte, war Politzer, auf Grund seiner im Jahre 1893 mitgeteilten histologischen Befunde, welche ihn veranlassten, als pathologisch-anatomisches Substrat dieser Form der progressiven Schwerhörigkeit eine primäre Erkrankung der Labyrinthkapsel anzunehmen. Gewiss existierten auch schon Mitteilungen vor dieser Zeit, jedoch die Statuierung dieses eigenartigen Krankheitsbildes mit dem oberwähnten pathologisch-anatomischen Substrate ist das Verdienst von Politzer, wenn auch, wie dies Denker sagt, schon im Jahre 1867 Moos die Ansicht aussprach, dass es sich um einen ' primär ostitischen Prozess des Felsenbeines ' handle und im Jahre 1890 Katz einen mikroskopisch untersuchten Fall einer während des Lebens diagnostizierten Otosklerose mitteilt. Ja Politzer selbst hat in der zweiten Auflage seines *Lehrbuches für Ohrenheilkunde*, die im Jahre 1887 erschien, eine Abbildung eines mikroskopischen Präparates von einer ankylosierten Stapediovestibularverbindung aufgenommen und kurz beschrieben, wenn auch ohne funktionellen Befund. Er selber beansprucht jedoch mit Recht die Priorität der Statuierung eines einheitlichen Krankheitsbildes erst auf Grund seiner im Jahre 1893 erschienenen Publikation. Vor dieser Periode hat auch Habermann im Jahre 1892 einen Fall mitgeteilt, bei welchem er jedoch über eine Prüfung mit Stimmgabel nichts berichtet.

Was die Pathogenese dieser Erkrankungen betrifft, so scheint vorläufig keine Aussicht auf eine Einigung der verschiedenen Ansichten vorhanden zu sein. Während Lucae, Stilling, Haimann sich noch der Ansicht Habermanns anschliessen, dass das Primäre die Erkrankung der

Trommelhöhlen-Schleimhaut und des Periostes sei, hat sich eine grosse Zahl der Autoren, wie Siebenmann, Hartmann und Manasse, der oben beschriebenen Ansicht Politzers angeschlossen. Wenn ich die diesbezüglichen pathologischen Veränderungen der von mir demonstrierten Präparate berücksichtige, so berechtigen sie mich nicht einen prinzipiellen Standpunkt einzunehmen, da in beiden der von mir demonstrierten Fälle die Schleimhaut auf das Vielfache verdickt und verändert im Bereiche des Knochenherdes ist, während die übrige Auskleidung des Mittelohres normale Verhältnisse aufweist. Ich bin nicht in der Lage zu entscheiden, ob der Knochenherd die Folge einer Schleimhauterkrankung ist, die sich bis auf diese eng umschriebene Stelle vollkommen zurückgebildet hat. Es ist jedoch auch nicht erwiesen, wenn auch höchst wahrscheinlich, dass die umschriebene Schleimhauterkrankung bloss sekundärer Natur ist. Noch schwieriger ist es, einen einheitlichen Standpunkt bei der Auffassung der Histogenese des Knochenherdes selbst zu erzielen.

Zur Illustrierung dieser Tatsache möchte ich bloss jene Autoren heranziehen, die in der Lage waren, auf Grund feinerer histologischer Untersuchungen sich einen Begriff über Art und Verlauf dieser eigentümlichen Erkrankung zu bilden. Manasse, der auf Grund histologischer Untersuchung von 17 Felsenbeinen (von 10 Patienten) die Histogenese des Prozesses folgenderweise erklärt: 'Die Alteration der Labyrinthkapsel beginnt mit Bildung von Osteoiden- und Granulationsgewebe innerhalb der präformierten Gefässräume. Der alte Knochen geht infolge dieses Prozesses durch einfache Verdrängung (nicht durch lakunäre Resorption) zugrunde, neugebildeter tritt an seine Stelle. Dieser hat nur einen intermediären Charakter, insofern als er wieder zerstört wird durch zwei verscheidene Arten der Einschmelzung, (1) durch siebartige Durchlöcherung des neugebildeten Knochens, (2) durch lakunäre Resorption mit Osteoklastenbildung. Dann wird von dem so entstandenen Markraume aus durch Osteoklastentätigkeit wieder Knochen (u. zw. spongiöser) gebildet, der nicht wieder verschwindet. Er wird derber und fester, aus dem zellreichen Mark wird fibröses, auch Fett- oder Gallertmark. Die Markräume werden immer kleiner, die Knochenbalken immer dichter, bekommen Lamellensysteme und stellen schliesslich wieder kompakten Knochen dar, der sich aber immer scharf von dem alten Labyrinthknochen abhebt.'

Manasse fasst den ganzen Prozess als einen entzündlichen auf, auf Grund des Auftretens von Granulations- und Osteoidengewebe, von Apposition und Resorption und erneuerter Apposition und Riesenzellenbildung, wie dies bei der chronisch entzündlichen Ostitis fibrosa in ähnlicher Weise vorzukommen pflegt. Da das Charakteristische des ganzen Prozesses eine Umwandlung der alten Labyrinthkapsel ist, so schlägt er den Namen Ostitis chronica metaplastica der Labyrinthkapsel vor.

Dieser von Manasse geschilderte Vorgang kann schon deshalb nicht als der allgemein gültige aufgefasst werden, weil es in der Osteopathologie ungewöhnlich ist, dass ein Knochen durch einen neugebildeten einfach

verdrängt wird, gleichgültig ob auf chemischem oder mechanischem Wege.

Das Primäre des Prozesses ist in der Regel der Abbau der Labyrinthkapsel und nicht, wie Manasse annimmt, die Bildung des neuen Knochens. Ein solcher Abbau ist nach dem gegenwärtigen Stande unserer histologischen Kenntnisse in der Osteopathologie nur durch Osteoklastentätigkeit möglich. Es würde mich hier zu weit führen, wollte ich die diesbezüglichen schönen Arbeiten von Axhausen ausführlicher zitieren. Hiermit entfällt auch die Ansicht Jörgen Möllers, der ebenfalls die Knochenneubildung für das Primäre hält.

Das Auftreten des Knochens mit intermediärem Charakter und der Abbau desselben durch Osteoklasten und durch siebförmige Durchlöcherung und dann erst das Auftreten des bleibenden Knochens ist ein Vorgang, dem wir sonst nirgends in der Osteopathologie begegnen.

Manasse hat anlässlich seines Wiener Aufenthaltes dem Hofrat Politzer ein histologisches Präparat von Otosklerose dediziert.

Ich bin in der Lage an diesem Präparate zu demonstrieren, dass die alte Labyrinthkapsel auch durch Osteoklastentätigkeit abgebaut wird, wenn auch nur an einer umschriebenen Stelle. Dass die soeben bezeichnete Stelle ein Teil der alten Labyrinthkapsel ist, erkennen wir mit Sicherheit an dem Nochvorhandensein von Interglobularräumen. Im jungen Labyrinthknochen ist eine sehr rege Osteoklastentätigkeit zu konstatieren, was ich jedoch nicht als Abbau des intermediären Knochens auffassen würde, sondern ganz einfach als regen Umbau in diesem Knochenherd.

Versuche ich die Auffassung Manasses an den histologischen Bildern meines zweiten Präparates zu prüfen, so bin ich auch nicht in der Lage dieselbe zu bestätigen. Wir finden im Bereiche des Knochenherdes zahlreiche in der Entstehung begriffene und etwas weiter vorgeschrittene Havers'sche Systeme. Wir finden im bunten Wirrwarr Knochen von altem und jugendlichem Charakter, Bildungen, die auch an Manasses siebförmige Durchlöcherung erinnern, die ich jedoch nicht als solche, sondern als geflechtartigen Knochen bezeichnen möchte.

Jedoch nicht ich allein, sondern auch Siebemann, der über eine viel grössere Anzahl von histologisch untersuchten Fällen verfügt als ich, sträubt sich gegen die Auffassung Manasses. Er bestreitet zunächst, dass der Prozess ein entzündlicher wäre, indem weder Granulationsgewebe, noch Rundzellen noch Plasmazellen vorhanden sind und bezeichnet als das wesentlichste des Prozesses die Spongiosierung. Er fasst die histologischen Veränderungen, mit Ausnahme dreier Fälle, welche jüngere Individuen betreffen und ein noch ganz junges Stadium repräsentieren, ungefähr folgenderweise zusammen :

‘ Lassen in meinen Präparaten stets neben der frischen Neuspongiosa auch die alten Umwandlungsprodukte derselben in Form von kompaktem, aber knorpelfreiem Knochen sich nachweisen. Die Markräume des spongiosierten Bezirkes entsprechen den ursprünglichen aber durch

lakunäre Arrosion erweiterten Havers'schen Kanälen. Sie sind bei jüngeren Individuen und frischen Prozessen sehr weit, werden aber durch allmähliche Apposition von neuer Knochensubstanz an die arrodierten Wandungen der Markräume mit zunehmendem Alter stets enger und spärlicher, so dass man von derartigen Präparaten oft mehrere Schnitte vergeblich durchsuchen kann, bis man an der Grenzzone der Neuspongiosa wieder eine frische lakunäre Arrosion mit ein- oder mehrzelligen Osteoklasten findet. Zeichen von Entzündung (Ostitis) fehlen.

Die Neuspongiosa ist in der Regel sofort nach ihrem Entstehen ziemlich kalkreich, so dass sie nicht als Osteoid bezeichnet werden darf.'

Er hält die Bezeichung *progressive Spongiosierung* als gerechtfertigt, und zwar 1. ' durch die anfängliche Erweiterung der Havers'schen Kanäle der betroffenen Knochenpartie, die — wenn auch zuweilen nur vorübergehend — ganz das Aussehen von neugebildeter Spongiosa annimmt,' und 2. ' dadurch, dass der Prozess nie zur Ruhe kommt ; auch in den ältesten Zonen der Neuspongiosa sieht man immer wieder Anbau und Abbau, teils nebeneinander teils nacheinander sich abspielen.'

Auf Grund meines histologischen Präparates bin ich nur dann in der Lage, den von Siebemann geschilderten Vorgang als den gewöhnlichen zu bezeichnen, wenn wir im Laufe der eben diskutierten Erkrankung ein *florides* und ein *latentes* Stadium annehmen, in welchem Falle der von mir demonstrierte Fall als latentes, das von Manasse hingegen uns überlassene Präparat als höchst florides Stadium der Erkrankung zu deuten wären.

Damit können wir auch vielleicht den insofern verschiedenartigen klinischen Verlauf der Hörstörung erklären, als wir die Fälle in die eine Gruppe, die von Politzer in ihrem Verlauf als gutartig bezeichnet wurde, und in die andere, mit rapider Verschlimmerung des Hörvermögens einhergehende, sogenannte maligne Form einreihen könnten.

Nicht unerwähnt möchte ich lassen, dass O. Mayer unter dem Titel ' Ein histologisch untersuchter Fall von Otosklerose ' in der *Monatsschrift f. O.* des Jahres 1911 über einen Fall von Otosklerose eingehend berichtet. Abgesehen davon, dass wir es hier, wie Manasse sagt, mit einem weit vorgeschrittenen Fall zu tun haben, der ausserdem an akuter lymphatischer Leukämie gelitten hat, unterscheidet sich sein Fall durch die Cystenbildung und Blutung so wesentlich von den bisher publizierten, dass, wenn man auch denselben als Otosklerose akzeptieren muss, man unter keinen Bedingungen berechtigt ist, aus seinen Befunden allgemein gültige Schlüsse über die Pathogenese zu ziehen.

So glaube ich für die ostitischen Veränderungen des von uns demonstrierten Präparates den Namen Ostitis fibrosa ablehnen zu müssen, denn bei dem Versuche, die in meinen Präparaten vorliegenden Veränderungen unter den von v. Recklinghausen aufgestellten Symptomenkomplex der Ostitis fibrosa (1891) (*Metaplastische Osteomalacie*, 1910) einzureihen, wie dies O. Mayer für seinen Fall getan hat, stosse ich auf folgende Schwierigkeiten :

I. Fehlen von Osteoklasten oder ausgesprochener Resorption im Sinne einer Rarefizierung.

II. Keine Umwandlung des Markes zu fibrösen Gewebslagen oder Granulationssprossen, daher auch keine Erweichungscysten, überhaupt keine ‘osteofibrösen Strukturen’ (Recklinghausen), wie sie Recklinghausen als Endprodukte der Umwandlungsvorgänge vorfand; ferner keine Zeichen von Blutstauungen, Blutgefässveränderungen oder stattgehabter Blutungen.

III. Der kompakte Charakter der Labyrinthkapsel bleibt trotz des gründlichen Umbaues und weitestgehenden Erneuerung der Elemente erhalten. Nur die jungen Havers'schen Räume sind etwas geräumiger als in der normalen Kompakta, so dass neben den zarten Blutgefässen noch einige spindelige Zellen Platz finden.

IV. Das Periost im Bereiche des Herdes ist wesentlich verdickt, ein Umstand, über dessen regelmässiges Vorkommen bei Otosklerose-Herden noch diskutiert wird. Recklinghausen betont, dass die Periostlage trotz stürmischer Erscheinungen in der Tela ossea solange normal bleibt, als normaler Knochen vorhanden ist. In meinem Falle muss die Entscheidung offen gelassen werden, ob die Verdickung etwas wesentlich Primäres oder nur die Reaktion der erkrankten Umgebung gegenüber darstellt.

V. Der junge, in den fibrösen Herden neugebildete Knochen bei der Ostitis fibrosa hat ausgesprochenen Bindegewebscharakter. In meinem Präparate befindet sich ein solcher geflechtartiger Knochen nur vereinzelt. Die Hauptmasse des neuen Knochens bilden zahlreiche Havers'sche Systeme mit mehr oder weniger vorgeschrittener Lamellenbildung. Wenn solche Recklingshausen auch nicht beschrieben hat, so will ich doch die Möglichkeit eines solchen Stadiums nicht bestreiten, wenn der Verlauf ein besonders protrahierter und der Umbau schon ein etwas weiter vorgeschrittener ist. Dies müsste jedoch erst bei der Ostitis fibrosa Recklinghausen nachgewiesen werden, um die in meinen Präparaten vorgefundenen Veränderungen mit der von Recklinghausen beschriebenen Krankheit zu identifizieren.

Dieser Widerstand wird nicht abgeschwächt durch die Tatsache, dass bei manchen alten Fällen von Otosklerose auch Fettmark gefunden wurde, wie dies Recklinghausen auch als für den Ausgang bei Ostitis fibrosa bezeichnend, angibt. Berücksichtigen wir das klinische Bild der Ostitis fibrosa und Otosklerose, so erhalten wir solch grosse Unterschiede, dass selbst, wenn das histologische Bild eine noch grössere Aehnlichkeit aufweisen würde, dies allein gegen eine Identifizierung beider Erkrankungsformen grosse Bedenken wachrufen müsste. Während nämlich die Ostitis fibrosa eine Erkrankung des gesammten Skelettes ist, unter hochgradiger Schmerzhaftigkeit nicht nur zu weitgehender Deformität, sondern auch meist zum Tode führt, haben wir es bei der Otosklerose mit einer in der Regel symmetrisch in beiden Ohren auftretenden, eng umschriebenen und genau lokalisierten Herderkrankung im Knochen zu tun.

Ueberblicken wir alle diese Momente, so erscheinen uns alle Versuche an Stelle des eingebürgerten und allgemein gebräuchlichen Namen eine zutreffendere Benennung zu setzen nicht nur verfrüht, sondern ganz geeignet, dieselbe Verwirrung hervorzurufen, wie sie die verschiedenartige Auslegung des histologischen Bildes bisher gezeitigt hat. Es wäre zweckentsprechender, den von Troeltsch allerdings für eine Erkrankung mit anderweitiger anatomischer Basis eingeführten Namen vorläufig bestehen zu lassen.

DISCUSSION

Professor Brühl (Berlin). Neumanns Ausführungen über die Pathogenese der Otosklerose veranlassen mich, zur Vorsicht bei der histologischen Deutung zu mahnen. Jeder Fall zeigt Neues; meine zehn Fälle erlauben mir *keine* definitive Stellungnahme, und wenn ich auch die Otosklerose nicht als Produkt einer Ostitis auffasse, so glaube ich doch sagen zu können, dass wir noch mit der Beurteilung abwarten müssen. Fest steht nur: Otosklerose ist knöcherne Stapesankylose, mit Sicherheit zu diagnostizieren, und schon von Troeltsch in ihrer Eigenart geahnt und von den Katarrhen abgesondert.

SECTION XVI

OTOLOGY

INDEPENDENT PAPER

OTOSCLEROSIS: CERTAIN CLINICAL FEATURES AND EXPERIMENTAL OPERATIVE PROCEDURES

By G. J. JENKINS, London

Before dealing with the main object of this paper it is necessary to touch on the physics of sound, but only so far as the transmission of the positive and negative phases of movement from the source to the end organs of the auditory nerve. With the pyschological interpretation of these impulses as sound, we are not at present concerned.

The transmission of these positive and negative phases of movement may be either by a mass or by a molecular movement.

The movement in the general atmosphere must be a molecular one under ordinary conditions. In the tympanic membrane and ossicular chain the transmission must be by a mass movement. The evidence in favour of the movement in this part of the mechanism of transmission being of the mass, is in the first place the mechanical arrangement of these ossicles. They are arranged as a system of levers so as to reduce the amplitude of movement. Also the presence of joints in this ossicular chain indicates that the elements are moving on one another in masses.

From the region of the foramen ovale to the nerve terminals the medium is a liquid, and the transmission of movement here is molecular. There cannot here be a mass movement, as the fluid would have to be enclosed in a rigid cavity, except at a remote part, otherwise the movement would be more or less dissipated. The labyrinth is not a rigidly enclosed space, as in places the walls are membranous, and in all parts there is a vascular lining membrane. These resilient structures could not be made sufficiently rigid except by a high tension of the fluid, but this tension is limited to the pressure in the veins. Also, it is most unlikely that a mass movement of such a fine degree could occur in the labyrinthine fluid, as the inertia of the fluid is probably too great. It cannot be accepted that the auditory terminal organs could be affected by a mass movement (even if possible) which could only be appreciated at a part that is resilient to phases of pressure.

In the above I have more or less assumed that impulses pass to the

internal ear by the foramen ovale, but whether by the foramen ovale or rotundum the construction and constitution of the labyrinth, I think, can admit only of the conclusion that the transmission of the impulses in this part is molecular.

Otosclerosis may be studied from many aspects, but clinically our attention is very strongly attracted to a comparison of the symptoms and signs of this condition with the signs and symptoms that constitute what is generally known as ' middle-ear deafness ', but better termed obstructive deafness.

These two groups of symptoms have many points of similarity, and more especially in the early stages of disease, but there are differences.

In this communication I intend to consider certain clinical differences that assist in the diagnosis of otosclerosis in its early stage, and have some bearing on a theory regarding the causation of the symptoms of this disease.

I propose in the first place to compare the results of the Rinné and Weber tests in otosclerosis and middle-ear or obstructive deafness. The results of these tests in the two conditions are similar, yet there are important differences.

It is necessary to make a further digression from the main object of the paper to consider the physics of the Rinné and Weber tests. The following is a summary of a study of these tests, which I shall deal with more fully elsewhere.

In the case of an individual with normal auditory apparatus, when a tuning-fork is applied as in the carrying out of the ordinary Rinné test, the fork is heard louder and longer at the external auditory meatus than when applied to the mastoid, but when the individual is suffering from a certain degree of middle-ear deafness or otosclerosis this appreciation is reversed, and the fork is heard louder and longer when applied to the mastoid than when at the meatus. This difference (negative Rinné), according to my observations and experiments, may arise from two causes:

Firstly.—In an ordinary room, and even in a noisy street, there are air-movements and vibrations which we have psychologically learned to eliminate, and of which we have become unconscious. If a normal individual passes from what might be described as a quiet room of a house into an underground cellar, or into a room specially prepared to ensure quietness, he will very readily appreciate the difference and recognize that the quiet room of the house is really comparatively noisy. These vibrations in a so-called quiet room do, however, cause confusion, and so we do not hear a tuning-fork so well when applied to the meatus in a somewhat noisy room as when in a quiet room, though whilst listening we are not conscious of the accessory sounds.

An analogous phenomenon occurring in everyday life may help to explain what is meant by this confusion. We all know how much more difficult it is to see the details of the tympanic membrane with reflected

artificial light in a room with a great deal of diffused daylight than in a darkened room. The extra light causes confusion. There are physical laws governing this confusion.

The accessory sounds above referred to as causing confusion are low tones.

Obviously when there is a defect in the meatal conduction apparatus both tuning-fork and accessory sounds are less distinctly heard by the meatal route than when the meatal conduction is normal.

Also where the same pathological condition exists, the fork applied to the mastoid is heard louder and longer than when the auditory apparatus is normal, because there is less confusion from accessory sounds reaching the auditory terminals by the meatus.

The Rinné test then becomes negative, not only because of defective conduction by the meatal route, but also because the bone conduction route is favourably affected since the meatal accessory sounds which cause confusion are diminished.

Secondly.—There may be an alteration in the media for bone conduction. If by some means one or more media be rendered more favourable to bone conduction, then obviously the bone-conduction route is placed at an advantage over the air-conduction route. The only alteration in the conducting media that seems possible or probable is in the labyrinthine fluid. This may be altered in the direction of increased density (specific gravity) in one of many ways, or there may possibly be an alteration in pressure. Of course these changes, if they occur, are incapable of direct proof, but indirectly there seems evidence of some change in this element.

I believe the difference between the results of the Rinné and Weber tests in ordinary middle-ear deafness and otosclerosis depend on the above. In middle-ear or obstructive deafness it is dependent on the first factor, but in otosclerous may be due probably to both factors.

Rinné test.—With middle-ear deafness it is necessary for the hearing distance for ordinary conversational voice to be reduced very considerably, in an ordinary room even to twelve feet, before the Rinné test becomes negative ; but in otosclerosis a patient may hear conversational voice at twenty-four feet and yet have a definite negative Rinné. The following is a summary of observations made with tuning-fork C^2 only.

The definite and doubtless answer of the otosclerotic patient to the Rinné test when negative is very characteristic, and the hesitative and doubtful answer of the middle-ear deaf patient to the Rinné test when negative is equally striking.

The Rinné test is negative with a lesser degree of deafness, and when negative is more definitely so in otosclerosis than in middle-ear deafness.

This test is important in otosclerosis, though not of great value in an ordinary case of middle-ear deafness. As a means of distinguishing between otosclerosis and middle-ear deafness, it is, in my opinion, of great value.

Indeed, when a patient is hearing conversational voice more than fifteen feet, and gives a certain and doubtless negative Rinné, one more than suspects that the patient has otosclerosis.

Weber test.—In very early unilateral otosclerosis, even with the Rinné test positive, one is even more struck by the definite and doubtless answer of the patient to the Weber test as compared with the doubtful answer of a patient with a similar degree of middle-ear deafness. With a very slight degree of unilateral deafness, due to otosclerosis, the patient without hesitation refers the sound of the fork to the defective side.

The difference between the two conditions in this respect is very marked and affords an important aid in the diagnosis of otosclerosis in the early stage.

The Rinné and Weber tests tend to prove that in otosclerosis the ' bone-conduction ' media have improved as well as meatal conduction has diminished ; for if in otosclerosis there is only obstruction to transmission of positive and negative phases by the ordinary meatal route, then the Rinné and Weber tests would be the same in otosclerosis as in middle-ear deafness. There must be, then, some additional factor.

It must be accepted, I think, that these differences between the results of the Rinné and Weber tests, and other clinical differences in otosclerosis and middle-ear deafness, indicate that the lesion in otosclerosis must be at the foramen ovale or in the labyrinth. In discussion on the clinical features and pathology of so-called otosclerosis most workers have confined their interest to the changes occurring in the neighbourhood of the foramen ovale, and most are inclined to attribute the symptoms of this condition to these changes causing fixation of the stapes. There seems to me difficulty in accepting this explanation. I believe they can be accounted for by a pathological change in the last link of the chain of conduction of sound waves—whether by meatal route or by bone conduction—the labyrinthine fluid. Suppose this fluid to have become heavier (increased specific gravity), then the inertia might be so great as to cause defective hearing of sounds through the middle ear, and at the same time the more dense fluid would favour bone conduction. Can this be the additional factor that would explain the above differences?

Another indication of the improvement in bone conduction is the common complaint of patients with advanced otosclerosis of the disturbance caused by personal movements, such as walking, eating, coughing, deep breathing, the annoyance to the patient by the noise produced often being considerable.

Two medical men who have been under my care said that walking on gravel was particularly annoying. They intentionally avoided gravel paths and the sea shore. This is sometimes complained of by patients with middle-ear deafness, but never to such a marked degree as in rapidly advancing otosclerosis.

Deafness.—As regards the hearing of the conversational voice, there are differences between patients with otosclerosis and middle-ear deafness.

It is a very common remark of a patient with otosclerosis, when being tested with conversational voice, that he hears the voice but cannot distinguish the words. The voice may be clearly audible some considerable distance, but the words distinguished only at a very short distance. This remark is occasionally made by a patient with middle-ear deafness, but not so definitely. An explanation of this phenomenon will probably be found in the same physical condition that is capable of producing paracusis.

One of the most important differences between middle-ear deafness and otosclerosis is the phenomenon called paracusis—a symptom of the latter.

What do we understand by ' paracusis ' ? The patient says he hears better in a noise. I have found it advisable to have the patient tested for this phenomenon under varying conditions. He should be tested by the same person in a very quiet room, in the open air, in a noisy street, a tramcar, and a railway train. In the early condition of otosclerosis the patient may hear better in the street than in the quiet room, but is at just as much disadvantage as an ordinary individual in a tram or a train. In later stages, the phenomenon of paracusis will not be noticed by the individual unless in a very noisy place, such as a railway train.

Does a patient with paracusis hear better in a noisy place than in a quiet room ? To say that he does, seems scientifically incorrect—though a question to this effect to the patient serves its purpose.

An individual with normal hearing always raises his voice when in a noise, though probably the otologist trained to testing with conversational voice, less so than others.

We are all familiar with the test for marked deafness by means of Bárány's noise apparatus. We know how constantly the normal individual when reading raises his voice when the apparatus is producing the noise in his ears, and that the very deaf individual scarcely speaks any louder than when the apparatus is still.

The paracusis patient tested by a normal individual in a quiet and in a noisy place is being tried with conversations of different degrees of loudness. If the observer, when testing, will cut off external sounds from himself by means of rubber plugs and test the otosclerotic in quiet and in a noise, he will find that the hearing of the patient is not improved in the noise, at any rate not obviously. In the noisy place, the otosclerotic patient with paracusis approaches the normal standard of hearing. If a normal person and otosclerotic patient be tested in the noise most suitable to the latter, the two will appear to approach one another in acuity of hearing.

The more advanced cases seem to hear even better than normal individuals in a very loud noise, such as that experienced in a railway train.

I am not sure that he actually hears better in the noisy place than the quiet place. I have experimented in this direction by plugging the

ears of the individual speaking, also by means of a gramophone record (to ensure constancy of intensity) and testing patients in the quiet and in various noises. The results of these experiments have not been convincing.

This is what might be expected, as there must be a proper ratio of degree of intensity between the voice and the noise, as otherwise the voice could not be differentiated.

There is another feature of paracusis that seems to me of great importance. Patients often say that they hear better in a noise than in the quiet, and add that that is what might be expected, as their friends naturally raise the voice in a noisy place. It seems we have paid too little attention to this common statement of patients. In order to investigate this feature, an otosclerotic and a normal individual were talked to in a quiet room by an untrained person with normal hearing, and suddenly a noise was produced in the room. The normal individual did not notice that the voice had been raised, but the patient said the voice was very much raised. In other words, the patient definitely appreciates the raising of the voice in a noisy place. It seems, then, that with otosclerosis the patient does not experience 'confusion' from the noise (if suitable). Also, the noise must in some way render the apparatus for transmission more efficient.

Without going further into a discussion on this matter we may certainly say that with the raised voice in a noisy place (more especially a low-toned continuous rumbling noise), a patient with paracusis approaches normal hearing; also, I think we may say that patients with advanced otosclerosis sometimes hear better than normal individuals, in a noise suitable to the former.

Can this phenomenon be explained? It is obvious that the lesion that could account for this phenomenon must be at the foramen ovale or in the labyrinth.

Paracusis has been said to be due to or at least associated with fixation of the stapes. There does not seem to be sufficient support for this hypothesis. If it be due to fixation of the stapes, then how is it that in the many cases of post-suppurative fixation that occur there is no paracusis? The occasional presence of this phenomenon in post-suppurative cases does not account for the absence of paracusis in the great majority. The occasional presence in such conditions may be explained by the presence of so-called otosclerosis as an additional factor in the deafness. If the stapes is restricted in its movement, the condition, in my opinion, would be indicated clinically by loss of acuity of hearing, as in middle-ear deafness.

Mr. Sydney Scott has given me what seems valuable information on this matter. He has found in two patients with the clinical condition described as otosclerosis that the stapes was freely movable. This seems to me a very important observation, as it indicates that it is possible for a patient to have the symptoms of otosclerosis without fixation of the stapes.

We are very apt to think that the striking pathological change in the

neighbourhood of the foramen ovale is the cause of the symptoms and signs of otosclerosis. It is possible, however, that some associated lesion—not so obvious—may be the real cause of the symptoms. Indeed, microscopic sections of early cases of otosclerosis show in some cases that the stapes is not fixed.

If paracusis cannot be explained by fixation of the stapes, then we must consider the only region left. I believe it could be explained by an increased density of labyrinthine fluid. This increased density (specific gravity) would mean an increased inertia that could not be satisfactorily overcome by the mechanism working so well with the normal fluid. A continuous noise would set this fluid in increased molecular motion, and so the fluid would be rendered more mobile and more suitable for the transmission of the voice vibrations, the inertia having been overcome.

The low rumbling tones must be the most suitable, as they would not cause confusion with the higher tone and overtones of the voice.

Is there any further evidence of a change in the labyrinthine fluid in this condition?

If water be run into the external auditory meatus, firstly at body temperature and gradually increased until the patient can just bear it, and continued for about five minutes, it will be found, in some cases of otosclerosis, that there is improvement of hearing immediately on ceasing, temporarily obtained.

The most marked improvement was obtained in those cases with hearing at 12 feet to 15 feet for conversational voice.

It is somewhat difficult to explain this effect, but is it not possible the result was attained by a change produced in the labyrinthine fluid? If the caloric test causes nystagmus by producing currents in the labyrinth, then it must do so by altering the density of the fluid. Hot water would, of course, diminish the density and increase molecular movement. This would be another way of overcoming the inertia of the fluid. These experiments were made with care to eliminate as far as possible any middle-ear defection—that is to say, they were inflated with catheter and Politzerised, on one occasion before syringing, and the same cases were at other times tested without inflation.

The results of these observations and experiments lead me to think that the lesion responsible for the clinical features of otosclerosis in the earlier stages is in the labyrinthine fluid, and that the changes at the foramen ovale and elsewhere are only associated or secondary.

It seems to me that though such a theory cannot be proved to be correct by direct evidence, yet, since it is so well supported by clinical evidence, it can be accepted as a working hypothesis.

Working on the above theory, I opened the perilymphatic space of the external semicircular canal with the view of altering the physical condition of the labyrinthine fluid in otosclerosis.

Case 1. A strong healthy female, 18 years of age, with all the classical symptoms and signs of otosclerosis.

Hearing. Conversational voice, $\frac{\text{R. 2 feet}}{\text{L. 1 foot}}$; whisper, $\frac{\text{R. 1}\frac{1}{2}\text{ feet}}{\text{L. 9 inches.}}$

Operation. The pinna was turned forward and the antrum opened as in the Schwartze operation.

The external semicircular canal was not obvious but working downwards and outwards from the floor of the antrum, the supero-internal surface of the bony canal was exposed, and eventually the perilymphatic space was opened for about 1·5 mm. No fluid was seen to escape.

The antrum was lightly plugged with ribbon gauze and the wound stitched up except at the lower angle.

The next day the patient was delighted with her condition, though suffering from vertigo and nausea, as she could hear conversational voice 6 to 8 feet, even through the bandage. On removing the bandage she could hear about 15 feet.

On the third day improvement in hearing was still maintained. The plug of gauze was removed.

It was a noteworthy feature that there was not at any time an obvious escape of labyrinthine fluid. This is in marked contrast with what occurred after a similar operation in another condition, when the gauze was soaked with fluid for two days after the operation.

On the fourth day the hearing was not so good, and from that time it gradually became worse.

Now, about two months after the operation, the hearing in the ear operated on is about 3 feet—somewhat better than it was before operation.

Although there is an improvement, yet the result is very disappointing, considering the immediate great improvement.

It is difficult to account for the immediate marked improvement with the subsequent return to almost the old condition. There are a few obvious suggestions.

Can it be that an alteration in the labyrinthine pressure had something to do with it? Can it be that after the escape of the labyrinthine fluid the secreting apparatus formed a lighter fluid? Can it be due to the formation of another window?

It seems to me more easy to explain it by alteration in fluid contents, as the patient was hearing so well with a plug in the antrum over the window, just as well as when the plug was removed.

If the opening in the canal had been acting as a window, then one would expect that the patient would hear better on removal of the plug. This was not so.

However, the experimental operation has so far only proved of this much interest, that the hearing can be improved by such a procedure, if only very temporarily.

Discussing the operation and its effects with my colleague, Mr. Arthur Cheatle, we arrived at the conclusion that it would be as well to try the effect of immediately skin-grafting the opening into the canal. This I have done in a second patient.

Case 2. The second patient was a strong healthy girl, aged 17 years, with the classical symptoms and signs of otosclerosis.

Hearing to conversational voice, $\frac{\text{R. 6 inches}}{\text{L. touching the pinna.}}$

The operation was done on the left ear.

The operation was similar in the first part to that on the first patient. A flap was then cut from the posterior wall of the meatus, and shaped so as to lie on the floor of the cavity of the mastoid. All the remaining raw

surface was covered with skin grafts and, of course, the opening in the external canal covered.

The immediate improvement in hearing was again very definite. She could hear conversational voice about 6 to 8 feet on the second day. The improvement did not last so long as in the first case, and in about two weeks from the time of operation the hearing was reduced to about 1 foot. This degree of hearing is still maintained about a month after the operation.

These operations were done with the view of altering the character of the labyrinthine fluid, but of course there are other physical disturbances to be taken into account when considering the effect of the operation. At present I do not wish to make any deduction. The subject is a complicated one, and I can only hope that these observations may be of some help towards the solution of this interesting problem of otosclerosis.

DISCUSSION

Dr. Bárány (Wien). Ich habe auch an Fällen von Otosklerose eine Bogengangseröffnung vorgenommen, jedoch von anderen Gesichtspunkten aus, als die, von denen Dr. Jenkins ausging, mit dessen Theorie des gesteigerten Labyrinthdrucks ich nicht übereinstimme. Ich ging von der Theorie des künstlichen Trommelfells aus. Um die Wirkung des künstlichen Trommelfells zu studieren, hatte ich bei einem Patienten mit epidermisierter Trommelhöhle nach Radikaloperation einen Tropfen Quecksilber ins Ohr getropft. Ich beobachtete, in dem Moment, wo das Quecksilber den Zugang zum runden Fenster verdeckte, eine sofortige Hörverbesserung. Unter Bezugnahme auf Experimente Machs erklärte ich mir dies folgendermassen: Stösst der Schall beim Radikaloperierten gleichzeitig an beide Labyrinthfenster, so kann er nicht eindringen, denn nach Mach muss das runde Fenster sich ausbauchen, wenn der Stapes hineinbewegt wird, und umgekehrt. Bedecke ich aber den Zugang zum runden Fenster, verhindere ich das Eindringen des Schalls an dieser Stelle, so kann nun beim ovalen Fenster der Schall eindringen und das runde Fenster fungiert als Ausweichstelle. Bei der Otosklerose ist das eine Fenster verknöchert, deshalb kann beim anderen der Schall nicht eindringen. Ich beschloss nun, eine Ausweichstelle für den Schall zu schaffen, an einer Stelle, an die der Schall direkt nicht gelangen kann. Deshalb eröffnete ich den hinteren Bogengang ohne das Antrum zu eröffnen. Es trat die erwartete Hörverbesserung auf, die auch nach Schluss der Hautwände bestehen blieb. Aber nach einigen Tagen war die Hörverbesserung wieder verschwunden. Die Hörverbesserung unmittelbar nach Eröffnung des horizontalen Bogenganges ist nach meiner Theorie ebenfalls verständlich, da jetzt der Schall, ohne durch eine Membran aufgehalten zu sein, direkt das Labyrinth erreichen kann. Sie muss aber nach Ausheilung, auch wenn sie ohne Knochenneubildung

erfolgt, wieder verschwinden. Die Offenhaltung des hinteren Bogenganges ist mir bisher auch nicht gelungen, trotzdem ich verschiedene Methoden, auch die Transplantation, versucht habe.

Reply

Mr. G. J. Jenkins (London) was interested to learn that Dr. Bárány had performed a similar operation, though his deduction was on very different lines. It was interesting that Dr. Bárány's patients also had a temporary improvement.

He did not know that the vestibular element had been so treated before in this condition.

He knew of the various methods of forming new windows described by Dr. Gray, but thought it probable that these procedures owed the improvement to changes in the labyrinthine fluid, although admitting the possibility of the improvement being due to the artificial window.

SECTION XVI

OTOLOGY

INDEPENDENT PAPER

NOUVEAU MODE DE TRAITEMENT DE L'ANKYLOSE DES OSSELETS

Par M. le Dr. J. MOLINIE, de Marseille

Des diverses lésions de l'oreille moyenne entraînant la surdité, l'ankylose des osselets est une des mieux établie et des plus anciennement connues.

Aussi n'est-il pas étonnant que depuis une époque déjà lointaine, les otologistes se soient efforcés d'en enrayer la marche ou d'en combattre les effets.

Quoique parmi les divers traitements institués, une place assez large ait été laissée à la médication générale, la thérapeutique locale l'a de beaucoup emporté sur la précédente, et pour restituer à la chaîne sa mobilité perdue c'est aux moyens mécaniques que l'on a eu plus particulièrement recours.

Les diverses pratiques tour à tour préconisées peuvent être classées en deux groupes :

1° Les mouvements indirects agissant sur les osselets par l'intermédiaire du tympan et qui sont : *Le Valsalva, la douche de Politzer, les insufflations* par la sonde *Itard*, le *massage pneumatique*.

A cette liste peuvent s'ajouter les tractions tympaniques exercées au moyen du *tympano-moteur* de Bonnier ou de *l'emplâtre* adhésif de Shambaug.

2° Les manœuvres directes s'exerçant sur la chaîne elle-même. De ce nombre sont : Le massage avec la sonde de Luçœ, *la mobilisation des osselets, leur extraction.*

Si nous envisageons la valeur de ces moyens thérapeutiques, nous dirons que les insufflations ou les massages tympaniques, dont il est fait dans l'otologie contemporaine un si large usage, sont vraiment utiles à titre préventif ou dans les formes légères d'ankylose mais dans les formes tant soit peu graves, ils sont à peu près complètement frappés d'impuissance.

[1] Commun. au Congrès international de Méd., Section d'Otologie, Londres, 6 août 1913.

En effet, si l'immobilisation de la chaîne coexiste avec un tympan cicatriciel, fibreux, adhérent, ce dernier, impossible à mobiliser, ne saurait exercer aucune action sur les osselets.

Si, d'autre part, le tympan est assez souple pour obéir aux variations de pressions auquel les on le soumet, il ne peut entraîner avec lui l'osselet auquel il adhère lorsque ce dernier offre un degré de résistance tant soit peu accentué. La continuation du traitement par les insufflations et les massages n'a donc pour effet que de distendre encore la membrane tympanique sans influencer favorablement l'ankylose. On superpose à la lésion ancienne une lésion nouvelle, l'inefficacité se double de la nocivité.

A ce degré d'ankylose on peut encore tirer quelque profit de l'application des procédés de Bonnier et de Shambaug, car leur action s'exerce non seulement sur le tympan, mais encore sur la chaîne par l'intermédiaire du marteau. Mais le champ d'action de ces procédés est assez rapidement parcouru et comme en outre ils tiennent le milieu entre les manœuvres indirectes et les manœuvres directes ils sont partiellement passibles des reproches que nous adresserons à celles-ci.

Parmi les manœuvres directes, nous avons cité en premier lieu le massage avec la sonde à ressort de Luçœ. Nous savons que si cet instrument est susceptible de mobiliser quelque peu la chaîne, il a le grave inconvénient de lui imprimer des mouvements exclusivement dirigés dans le sens de la pression et par conséquent de ne pouvoir agir utilement dans le cas d'enfoncement du manche du marteau.

La *mobilisation des osselets* a été la grande entreprise thérapeutique de la fin du siècle dernier. Conçue et exécutée par Kessel, Sexton, Miot, Boucheron et quelques autres, elle a éveillé des espoirs fort légitimes en apparence.

En effet, la relation de causalité entre la surdité et l'ankylose étant dans la plupart des cas dûment établie, il est logique d'attendre de la disparition de la lésion, le retour de la fonction.

Mais si la période d'essais a enregistré quelques succès retentissants, un silence significatif a succédé à la faveur passagère, et la mobilisation des osselets après une carrière plus brillante que durable est tombée dans un abandon dont il est peut-être téméraire de la tirer.

Cependant, il n'est pas sans intérêt de rechercher après coup et dans la tranquillité d'esprit que permet le recul du temps les véritables causes de ces insuccès.

Déjà au cours de sa carrière des objections très judicieuses s'étaient élevées — on faisait valoir en particulier la nature dyscrasique des lésions de l'oto-sclérose qui les vouait fatalement à la récidive.

Certes à l'heure actuelle cette objection n'a rien perdu de sa valeur, mais elle ne saurait expliquer à elle seule l'absence si fréquente des succès temporaires dans l'oto-sclérose, et la rareté également très grande d'amélioration dans les cas d'ankylose à la pathogénie desquels l'oto-sclérose reste complètement étrangère.

D'autres raisons sont nécessaires pour expliquer ces échecs. A notre

avis il y a lieu d'incriminer la technique opératoire qui nous paraît s'être trouvée en cette circonstance fort au-dessous de sa tâche. C'est ainsi que certains auteurs commencent la mobilisation par la section préalable de la chaîne des osselets au niveau de l'articulation incédo-stapédale.

Ils mobilisent ensuite l'étrier en lui imprimant des mouvements de latéralité, or, c'est là une manœuvre antiphysiologique, car l'étrier se mouvant dans la fenêtre ovale à la façon d'un piston, il est par conséquent illogique de chercher à lui faire exécuter des mouvements de bascule.

La façon même d'opérer ces déplacements n'est pas moins malheureuse. Après avoir incisé le tympan on va armé d'un stylet, véritable bâton d'aveugle, à la rencontre de la tête de l'étrier. Là utilisant cet instrument à la façon d'un levier on lui fait prendre un point d'appui sur le bord du spéculum et on opère sur l'étrier une pression plus ou moins vigoureuse en vue d'obtenir le déplacement désiré.

Mais le résultat ne répond pas à l'attente. Parfois on n'obtient rien du tout, si non en augmentant les efforts la fracture des branches de l'étrier, d'autres fois au contraire on dépasse le but et c'est l'avulsion de l'osselet qui couronne ces tentatives.

Le résultat n'est guère meilleur dans un cas que dans l'autre, car l'impotence fonctionnelle est sensiblement la même avec la luxation qu'avec l'ankylose.

L'extraction des osselets ne présentant aucune supériorité sur leur mobilisation, nous ne nous attarderons pas à la discuter et, en présence du résultat de ces manœuvres, on peut dire que si à l'heure actuelle on doit éprouver quelque surprise, c'est moins de l'échec habituel de ces tentatives que de leur réussite partielle ou intermittente.

Aussi de l'examen critique auquel nous venons de nous livrer, nous croyons avoir le droit de tirer cette conclusion : que la mobilisation des osselets ne peut, en tant que méthode thérapeutique, être appréciée d'après les résultats qu'ont donnés les précédents essais. Tant qu'elle ne sera pas exécutée dans des conditions qui la mettent à l'abri des reproches encourus jusqu'ici on devra suspendre tout jugement à son égard.

Encore moins les tentatives antérieures comportent-elles des raisons péremptoires de renonciation définitive à cette méthode qui repose malgré tout sur la logique la plus rigoureuse, et nous dirons même que notre pénurie actuelle de moyens thérapeutiques vis-à-vis de la surdité, nous impose le devoir de nous engager à nouveau dans cette voie qui peut-être sera plus féconde, si nous la poursuivons à l'abri des écueils que l'expérience du passé nous permet d'éviter.

Parmi les difficultés inhérentes à cette tâche, il en est une qui paraît presque insurmontable, parce qu'elle poursuit des desiderata en apparence inconciliables. Comment exercer une action dynamique suffisante pour amener la mobilisation, sur des organes n'offrant pas une résistance suffisante pour la subir ?

En particulier comment triompher d'une ankylose stapédo-vestibulaire très serrée quand on ne peut agir sur elle que par l'intermédiaire des branches de l'étrier essentiellement minces et fragiles ?

Certes, il est des cas où l'existence d'une véritable synostose de cette articulation interdit toute tentative, mais dans les cas d'ossification partielle ou en voie d'évolution, on doit chercher à triompher de la résistance opposée par le tissu fibreux sans s'exposer à provoquer la fracture des branches de l'étrier. Il nous paraît indiqué d'utiliser dans ce but les *tractions continues* si courantes dans quelques branches de la chirurgie.

Là où un effort considérable exercé brusquement est impuissant ou funeste, une action dynamique d'intensité, bien moindre, mais suffisamment prolongée atteindra sûrement le but.

Si la chirurgie orthopédique nous offre par ses redressements de membres et de la colonne vertébrale une preuve éclatante de cette assertion, non moins saisissants sont les exemples fournis par l'orthodontie, qui non seulement opère des déplacements dentaires d'une étendue invraisemblable, mais obtient encore, par des efforts continus, de véritables transformations du squelette de la face, alors qu'une intervention brusque ou sanglante n'eut produit que mutilations.

Nous devons donc nous inspirer de ces exemples, pour agir sur la chaîne des osselets et transporter dans le domaine de l'otologie le principe des tractions continues qui a donné par ailleurs de si heureux effets.

Nous lui adjoindrons aussi le massage des osselets, car nous savons que cette pratique favorise singulièrement le bon fonctionnement des articulations et nous verrons plus loin que l'application de ce moyen mécanique est le complément aisé, facile et naturel des tractions continues.

Mais ce n'est pas tout, ces actions dynamiques ne doivent pas s'exercer aveuglément, ou simplement au jugé, elles doivent être l'objet de mensurations précises, connues et parfaitement réglables.

De plus, il faut que nous soyons toujours à même d'apprécier les effets du traitement et de nous assurer de l'existence du déplacement ossiculaire et d'en déterminer rigoureusement le degré.

C'est à la réalisation de ces conditions opératoires de mobilisation ossiculaire que nous nous sommes employés ; nous allons faire connaître les moyens qui nous ont paru les plus propices à ce but.

Instrumentation

L'instrumentation à laquelle nous avons recours pour la mobilisation des osselets comprend :

1° Un *petit crochet* en or de 6 à 7 mm. de longueur totale et dont la partie prenante de $1\frac{1}{2}$ mm. de long est simplement formée par une coudure de la tige principale. La portion coudée réalise avec l'axe un angle un peu inférieur à 45°. La partie de la tige opposée au crochet est légèrement évasée en entonnoir. Elle est creusée en forme de douille.

Elle porte sur un de ses bords un trou dans lequel passe un fil d'acier très fin de $2\frac{1}{2}$ à 3 cm. de longueur. Ce fil est terminé par un anneau. A l'extrémité de l'anneau s'attache un bracelet en caoutchouc. A l'extrémité de ce bracelet se trouve fixé un fil de soie de 15 à 20 cm. de longueur.

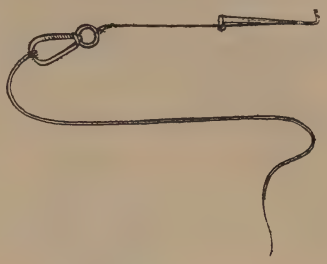


Fig. 1

2° Un *stylet monté* sur un manche à instruments otologiques et dont l'extrémité vient s'emboîter exactement dans la douille du crochet.

3° Un *coussinet pneumatique* en forme de couronne ayant 5 cm. de diamètre. Sur un point de la circonférence extérieure se branche un tube de gonflage terminé par un robinet d'arrêt et un ajutage.

4° *Un manomètre* indicateur de pression, gradué en centimètres de mercure.

5° Une *poire de gonflage* munie à son orifice de sortie d'un tube en Y dont chaque branche est munie d'un ajutage s'adaptant l'un au tube du coussinet pneumatique l'autre au tube du manomètre.

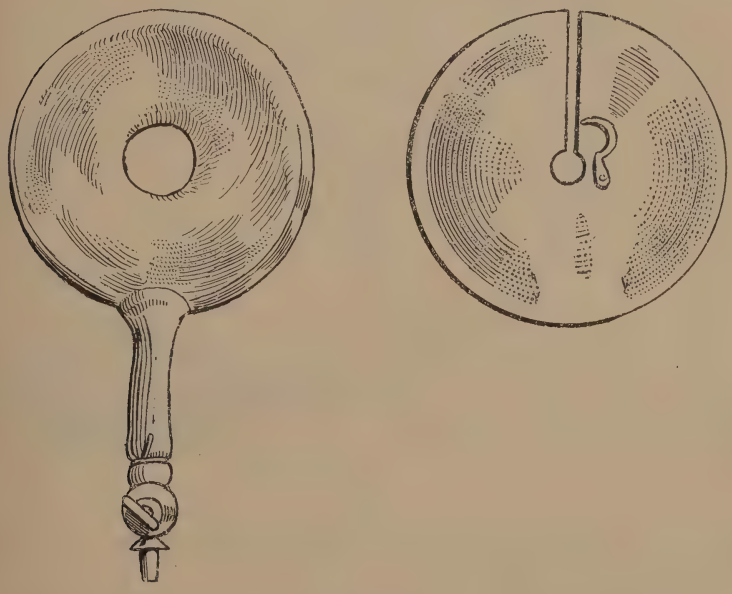


Fig. 2

6° Un *disque de métal* légèrement convexe portant une fente de 1 mm. de large disposée en un rayon, c'est-à-dire allant de la circonférence du disque à son centre. Près du centre se trouve un crochet de fermeture.

7° Un *appareil de contrôle* composé d'une vis micrométrique montée sur un trépied.

Application

Pour opérer la traction continue ou le massage des osselets, on procède de la façon suivante :

On fait une incision au tympan variable suivant l'osselet ou la portion d'osselet sur laquelle on désire faire porter l'action mécanique.

On monte le crochet sur le stylet, en introduisant l'extrémité du stylet dans la douille du crochet. Le bracelet de caoutchouc passé à un ergot

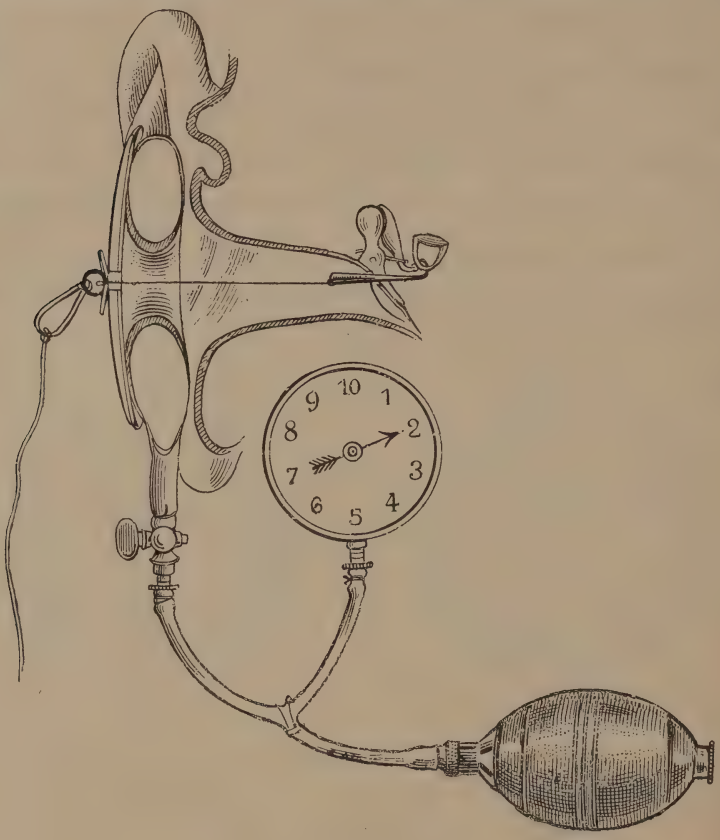


Fig. 3

siégeant au voisinage du manche, maintient le crochet sur le stylet dans un état de fixité parfaite.

Le crochet est insinué à travers l'ouverture tympanique et porté au contact de la portion de l'osselet déterminée d'avance.

Saisissant le fil de soie vers son milieu on exerce sur lui une traction de façon à le tendre ; on dégage le bracelet de caoutchouc de son point de fixation, et on retire le stylet. Seul le crochet reste en place du seul fait de la traction sur le fil que l'on continue toujours.

Après avoir mis un peu de gaze stérilisée dans le conduit, on fait

passer l'extrémité libre du fil dans l'ouverture médiane du coussinet et on amène ce dernier au contact de la main qui tient le fil, reprenant ce dernier en arrière du coussinet et en continuant toujours de maintenir le fil tendu on amène le coussinet au contact de l'oreille.

On tasse de la gaze au-dessous de lui, on le gonfle de telle façon que sa face supérieure affleure à peu près ou dépasse légèrement le niveau de l'anneau.

Dans l'anneau on passe une clavette qui vient prendre point d'appui sur le coussin ou bien on insinue le fil d'acier dans la fente du disque et lorsque le fil occupe le centre du disque on le maintient dans cette position par la fermeture du crochet. Si la pression exercée par la face extérieure du coussinet est suffisante pour tendre le fil d'acier, l'appareil tient en place de lui-même.

Pour augmenter la pression on met le robinet du coussinet en rapport avec la poire de gonflage ; comme celle-ci est également en communication avec le manomètre, ce dernier indique à tout instant le degré de la pression de l'air du coussinet et par là même l'intensité de la traction supportée par l'osselet. Lorsque l'on estime la pression suffisante on ferme le robinet du coussinet et on laisse l'appareil en place. On le protège par une feuille d'ouate et on maintient le tout par une bande de crêpe Velpeau très légèrement serrée ; ainsi s'exerce la traction continue à laquelle moyennant une surveillance quotidienne on peut donner une durée plus ou moins prolongée.

Pour faire le *massage des osselets*, on met le robinet du coussinet en communication avec une pompe pneumatique dont on a déterminé au préalable, avec le manomètre, la puissance d'action correspondant aux divers degrés de course du piston et aux divers degrés de vitesse du moteur. Les variations de pression du coussinet sont transmises à l'osselet qui subit ainsi un massage dont on peut à volonté faire varier l'intensité ou l'étendue.

Ainsi qu'on le voit dans cette série de manœuvres, on ne livre rien au hasard. Aussi bien la traction que le massage s'opèrent avec une précision parfaite, exempte de risque et d'aléa ; on ne peut produire, si l'on opère avec prudence, ni désordre de la caisse, ni désarticulation des osselets, tout en conservant la possibilité d'atteindre l'intensité d'effort nécessaire au résultat cherché.

Contrôle des Mouvements des Osselets

Si l'on veut bien réfléchir sur la mise en place du dispositif ci-dessus on se rendra compte que les déplacements subis par l'anneau fixé à l'extrémité du fil d'acier et qui prend place au centre du disque, sont la représentation fidèle des mouvements d'oscillations subis par l'osselet. En effet le crochet, le fil d'acier et l'anneau étant inextensibles si sous l'influence de la pression du coussinet il se produit un déplacement vers l'extérieur ; il ne peut résulter que d'un déplacement de même sens et de même étendue subi par l'osselet.

La mensuration du mouvement apparent nous renseigne sur l'amplitude du mouvement de l'osselet. Pour mesurer ce déplacement du disque on procède de la façon suivante : on applique le trépied que nous avons montré au-dessus de l'oreille et après avoir placé la vis bien au centre du disque chacun des pieds vient prendre contact avec un plan osseux résistant du crâne ou de la paroi externe du visage.

Le coussinet étant gonflé à une faible pression de 0,01 cm. de mercure par exemple, pression suffisante pour opérer une tension du fil, mais pas une véritable traction, on amène la vis micrométrique au contact du centre du disque et on lit le degré de pénétration de la vis. On la fait ensuite reculer de la quantité correspondant à l'étendue du mouvement que l'on désire faire acquérir à l'osselet. On gonfle alors le coussinet et on voit si on peut amener le disque au contact de la vis micrométrique et quelle pression est nécessaire pour obtenir ce déplacement.

Selon que ce résultat sera ou ne sera pas atteint on jugera s'il y a lieu d'interrompre ou de continuer le traitement.

Indications

En principe, ce mode de traitement de l'ankylose convient à tous les cas où existe cette lésion, mais il y a quelques différences dans son mode d'application suivant la forme et la nature de l'affection.

Conformément à la règle établie, nous ramènerons les multiples variétés de lésions otiques chroniques non suppurées à trois types bien connus, quoique dans la pratique cette systématisation soit rarement réalisée ; ce sont :

1° L'otite adhésive.

2° L'oto-sclérose.

3° L'otite cicatricielle post-suppurative.

Dans les phénomènes de la première catégorie on observe comme lésions caractéristiques la rétraction en masse du tympan qui est quelquefois épaissi, mais le plus souvent mince, plissé, ondulé et vallonné avec des enfoncements plus particulièrement limités, à l'extrémité du manche. Ce dernier présente assez souvent un déplacement marqué, il a une tendance à l'horizontalité, souvent il est adhérent au promontoire.

Dans ce cas, il faut ouvrir le tympan en arrière du manche par une incision qui lui est perpendiculaire et venant tomber au voisinage de son extrémité inférieure.

Cette incision n'a pas besoin d'être très longue, 2 mm. suffisent, mais si on a un tympan fortement rétracté on peut la prolonger en arrière de façon à pratiquer du même coup une plicotomie postérieure.

Cette incision perpendiculaire à l'extrémité du manche présente en dehors de l'avantage de permettre la plicotomie celui d'empêcher le glissement du crochet vers l'extrémité supérieure du manche.

Le crochet étant passé derrière le manche on exerce sur le fil de soie une traction dirigée un peu en avant et en bas. On continue par la

mise en place de l'ensemble du dispositif que nous avons indiqué tout à l'heure. On fait monter graduellement la pression de 0,01 à 0,03 ou 0,05 cm. de mercure et on observe les modifications qui apparaissent à l'extrémité de l'anneau.

Il est rare qu'on n'observe pas un mouvement vers l'extérieur assez accentué, car la résistance du manche est généralement assez vite vaincue.

Lorsqu'en passant d'une pression de 1 à 5 cm. de mercure on obtient un déplacement de l'osselet de 3 à 4 mm. on peut considérer que le but est atteint.

Généralement après cette mobilisation on observe une amélioration très grande de l'audition, mais il ne faut pas perdre de vue que cette otite adhésive est souvent provoquée et entretenue par des lésions tubaires et que si celles-ci ne sont pas traitées, la récidive de la lésion de l'oreille pour se produire à une époque plus ou moins lointaine n'en sera pas moins fatale.

Oto-sclérose. — Ce qui caractérise objectivement cette lésion, c'est l'intégrité apparente du tympan qui n'est nullement enfoncé ni déformé. Le manche du marteau est vertical, blanchâtre, souvent spatuliforme à son extrémité. La transparence du tympan permet souvent d'apercevoir le fond de la caisse et souvent aussi la branche descendante de l'enclume et une portion de l'étrier.

Ici bien que l'ankylose stapédo-vestibulaire soit la lésion prédominante, elle s'accompagne souvent aussi d'ankylose incudo-malléale, car non seulement le spéculum de Seigle n'entraîne pas de mouvement du manche du marteau, mais encore une traction exercée directement sur l'osselet ne permet pas le moindre déplacement.

La chaîne immobilisée sur place forme un bloc solidement fixé à la paroi labyrinthique. Veut-on obtenir une mobilisation de force on est à peu près certain d'amener une fracture de l'osselet qui se traduit par un petit claquement sec.

Il ne faut donc pas vouloir faire de la mobilisation brusque et c'est ici que les tractions continues et les massages doivent l'emporter sur les manœuvres entemporanées.

L'ouverture du tympan doit se faire dans le segment postéro-supérieur, par une incision partant de la courte apophyse et suivant le bord supérieur du tympan pour venir rejoindre le cadre tympanal en arrière ; on taille ainsi un lambeau que l'on rabat en bas, ce qui donne une vue et un accès assez larges sur la portion postérieure de la caisse.

L'ouverture ainsi pratiquée permet d'agir à volonté sur le manche ou sur l'étrier. La solidarité qui unit les osselets permet de commencer le traitement par une traction sur le manche, mais il faut ensuite agir sur l'étrier lui-même.

Ici on accrochera l'osselet en-dessous. Souvent l'accès de l'étrier est facilité par sa position apparente, mais d'autres fois il échappe à la vue et alors on est obligé de se guider sur les sensations fournies par le toucher.

On fait le traitement comme précédemment, mais il ne faut pas chercher à atteindre des déplacements considérables de l'étrier. A l'état normal les mouvements qu'il exécute sont de l'ordre du dizième de millimètre, on devra donc se guider sur les sensations éprouvées par le malade, car les mouvements d'amplitude si infime ne peuvent être contrôlés expérimentalement.

Il arrive assez souvent que l'étrier soit bloqué à fond et que même avec cette pression continuée plusieurs jours on n'observe pas la moindre modification dans l'état du malade.

Nous n'avons jamais dépassé une durée de traction de six jours, peut-être faudrait-il continuer le traitement pendant plusieurs semaines, pour triompher de la résistance de l'ankylose, mais l'expérience nous manque sur ce point.

Otite cicatricielle post-suppurative. Ici il faut distinguer deux cas, tantôt il n'y a pas de trace de perforation et le tympan vient adhérer au fond de la caisse et alors il faut non seulement inciser le tympan, mais se frayer une voie au milieu du tissu cicatriciel pour passer derrière l'osselet et le traitement peut être assez laborieux.

Par contre, dans d'autres cas il existe une perforation du tympan assez vaste qui permet d'opérer très librement dans la caisse.

On observe assez souvent la désarticulation des gros osselets et de l'étrier. Les premiers sont d'un côté et le second présente sa tête et ses branches parfaitement accessibles. On peut opérer les tractions sur les gros osselets, mais quand l'étrier apparaît c'est sur lui que l'on doit diriger les manœuvres.

On peut ici faire les manœuvres courtes et répétées, car la perforation du tympan permet de les interrompre ou de les reprendre à volonté, mais il faut agir aussi avec délicatesse et les interrompre ou même les arrêter si l'on constate une réaction inflammatoire tant soit peu vive faisant craindre le retour de la suppuration.

DISCUSSION

Dr. MAURICE (Paris). Les auristes sont trop chirurgiens et pas suffisamment physiologistes et phonéticiens. Dans l'oto-sclérose il existe des troubles fonctionnels indépendants des lésions anatomiques, ce que l'autopsie ne révèle pas. Le traitement que nous conseillons et que nous pratiquons est une rééducation auditive au moyen de vibrations rapides et sonores qui entraînent l'ouïe du malade, qui mobilisent, plus physiologiquement qu'un stylet ou qu'une pompe, les osselets ankylosés et les tympans épaissis. Mon instrumentation est entre les mains de nombreux spécialistes qui ont pu confirmer mes résultats dans leurs publications ; citons les D^rs^ Granat, Royet, Belousteguigoitia, Humphris, etc. Nos succès comportent environ 75 pour cent de résultats heureux et 5 pour cent d'insuccès absolus, ceci sur plus de 200 cas traités.

SECTION XVI

OTOLOGY

DISCUSSION No. 4

DIE ANATOMIE UND KLINIK DER NICHT EITRIGEN LABYRINTHERKRANKUNGEN

Referat von Professor Dr. G. ALEXANDER, Wien

Vorbemerkung

Gruppierung und Begrenzung des Stoffes. — Der ehrenvollen Einladung der Kongressleitung, das vorliegende Referat abzuhalten, bin ich gerne gefolgt. Der Titel 'Nicht eitrige Labyrintherkrankungen' ist vom Komitee selbst gewählt worden und ich habe ihn unverändert angenommen. Allerdings gibt diese Nomenklatur nur eine Abgrenzung aller anderen Labyrintherkrankungen gegenüber den eitrigen Labyrintherkrankungen und ist für die Gruppierung des Themas nicht ohne weiteres ausreichend.

Ich musste davon Abstand nehmen, Ihnen einen vollständigen Literaturbericht zu bieten, da der grosse Umfang der Literatur das Referat allzu umfangreich gestaltet hätte. Ich konnte auf die Vollständigkeit der Literaturangaben umso eher verzichten, als in der letzten Zeit mehrfach derartige Referate verfasst worden sind und zuletzt Brühl (Politzer, *Geschichte der Ohrenheilkunde*, II. Band) eine erschöpfende Literaturübersicht gegeben hat. In meinem Referate lege ich vor allem Wert darauf, Ihnen im Projektionsapparat an der Hand von histologischen Präparaten und Diapositiven von solchen die bei den nicht eitrigen Erkrankungen des Ohrlabyrinths vorkommenden Veränderungen zu demonstrieren. Ich glaube, dass ein derartiger Vorgang, der Sie mit sämtlichen, in Betracht kommenden pathologisch-anatomischen Veränderungen bekannt macht, interessanter und für die Eröffnung der Diskussion erspriesslicher ist als die trockene Aufzählung literarischer Daten. Ich erwähne dabei, dass der Demonstration die histologische Zergliederung 77 einschlägiger Fälle zugrunde gelegt ist und dass das histologische Material sämtlich meiner eigenen Laboratoriumssammlung entstammt.

Gruppierung, Aetiologie und *Vorkommen.* — Es ist notwendig, für den

Zweck des Referates, die nicht eitrigen Labyrintherkrankungen in übersichtliche Gruppierung zu bringen. Die nicht eitrigen Labyrintherkrankungen umfassen im weitesten Sinne folgende Formen:

(A) Die kongenitalen Labyrinthanomalien und die intraembryonal erworbenen Labyrintherkrankungen.

(B) Die postembryonal auftretenden Labyrintherkrankungen.

Die Gruppe (A) umfasst:

I. Die kongenitale labyrinthäre Schwerhörigkeit.

II. Die kongenitalen Krankheiten des statischen Labyrinths.

III. Die progressive labyrinthäre Schwerhörigkeit des jugendlichen Alters.

IV. Die kongenitale Taubheit.

V. Die kongenitalen Krankheiten des inneren Ohres bei Kretinismus; die endemische, konstitutionelle Schwerhörigkeit und Taubheit.

Unter den postembryonal auftretenden Labyrintherkrankungen sind die primären und die sekundären zu unterscheiden. Unter einer primären Labyrintherkrankung ist eine solche zu verstehen, bei welcher das Labyrinth infolge einer von aussen her wirkenden Schädlichkeit oder infolge einer Allgemeinerkrankung als erster und als einziger Abschnitt des Gehörorgans erkrankt. Unter einer sekundären Labyrintherkrankung versteht man eine Erkrankung des inneren Ohres, die sich durch Uebergreifen der Erkrankung aus anderen Abschnitten des Gehörorgans entwickelt.

Die postembryonal auftretenden, nicht eitrigen Labyrintherkrankungen ergeben danach folgende Gruppierung:

(*a*) Die primären Erkrankungen des inneren Ohres.

I. Die (akuten) traumatischen Erkrankungen des inneren Ohres.

II. Die chronischen traumatischen Erkrankungen des inneren Ohres (die professionelle labyrinthäre Schwerhörigkeit und Taubheit).

III. Die Erkrankungen des inneren Ohres bei Stoffwechselanomalien.

Gicht.

Diabetes.

IV. Die Erkrankungen des inneren Ohres bei Erkrankungen des Blutes und der blutbildenden Organe.

Anämie.

Chlorose.

Progressive, perniziöse Anämie.

Leukämie.

Skorbut.

Hämorrhagische Diathese.

V. Die Erkrankungen des inneren Ohres bei den konstitutionellen Krankheiten.

1. Die Krankheiten des inneren Ohres bei lymphatischer Konstitution und Rachitis.

2. Die nicht kongenitalen Krankheiten des inneren Ohres bei endemischem Kretinismus.

VI. Die Erkrankungen des inneren Ohres bei Erkrankungen der Kreislaufsorgane.

VII. Die Erkrankungen des inneren Ohres bei Erkrankungen der Niere.

VIII. Die Erkrankungen des inneren Ohres bei Erkrankungen des Gehirns, des Rückenmarks und bei Neurosen.

Hirntumoren, akuter und chronischer Hydrocephalus.
Neurasthenie.
Hysterie.
Multiple Sklerose.
Tabes.
Paralyse.
Acusticustumor.
Tumoren der Dura.
Erkrankungen des inneren Ohres bei Morbus Basedowii.

IX. Die toxischen Erkrankungen des inneren Ohres, Chinin, Salizylsäure, Arsen, Blei, Alkohol, Nikotin, Radium.

X. Die infektiösen (nicht eitrigen) Erkrankungen des inneren Ohres.

1. Bei den akuten Infektionskrankheiten.

Scharlach.
Morbillen.
Diphtherie.
Meningitis cerebrospinalis epidemica.
Pertussis.
Mumps.
Typhus.
Influenza.
Erysipel.
Gelenkrheumatismus.

2. Bei den chronischen Infektionskrankheiten.

Tuberkulose.
Syphilis.

(*b*) Die sekundären nicht eitrigen Erkrankungen des inneren Ohres.

I. Infolge von akuter und
II. Chronischer Mittelohreiterung.
III. Infolge von chronischem Adhäsivprozess.
IV. Infolge von Otosklerose.

Das gesamte Thema musste für den Zweck des vorliegenden Referates eine Beschränkung erfahren. Nicht einbezogen sind in das vorliegende Referat die in Klammern gestellten Gruppen. Die kongenitale Taubheit und die luetischen Erkrankungen des inneren Gehörorgans einschliesslich der Ohrerkrankungen bei Tabes und Paralyse sind in dem vorliegenden Referate nicht berücksichtigt, da sie Gegenstand selbständiger Referate des Kongresses bilden.

A. Die kongenitalen Labyrinthanomalien und die intraembryonal erworbenen Labyrintherkrankungen

Die kongenitale Taubheit wird, um den Umfang des Referates nicht zu sehr wachsen zu lassen, hier nicht besprochen. Wir beschränken uns auf die Besprechung der folgenden kongenitalen Labyrintherkrankungen.

I. *Die kongenitale labyrinthäre Schwerhörigkeit.* — Die ziemlich seltene Erkrankungsform gelangt ein- oder doppelseitig zur Beobachtung. Ein einwandfreier ätiologischer Faktor ist nicht auffindbar. In vielen Fällen ist eine hereditäre Belastung vorhanden, indem Vater oder Mutter schwerhörig oder taub sind oder kongenitale Taubheit in der Familie, bezw. in der Blutsverwandtschaft beobachtet worden ist.[1] In anderen Fällen fehlt jede erbliche Belastung bezüglich des Gehörorganes, dagegen sind andere hereditärdegenerative Kennzeichen vorhanden. So sieht man die kongenitale labyrinthäre Schwerhörigkeit kombiniert mit geringfügigen Abnormitäten am Gesicht,[2] an den Extremitäten oder am Rumpfe, oder es handelt sich um eine allgemeine nervöse Belastung und das Vorkommen von Nerven- und Gehirnerkrankungen in der Aszendenz, oder endlich um hereditäre Lues. Ein grosser Teil der Fälle von endemischer, kretinischer Labyrinthschwerhörigkeit (s. u.) gehört gleichfalls hierher.

Die anatomischen Veränderungen bei der kongenitalen labyrinthären Schwerhörigkeit bestehen, soweit die bisher anatomisch untersuchten Fälle allgemein gültige Schlüsse zulassen, in einer kongenitalen Hypoplasie des Ganglion spirale und des peripheren Anteiles des Schneckennerven. Die Kerne, Wurzeln und die zentrale Verzweigung des Cochlearis erweisen sich dagegen vollständig intakt. Das Cortische Organ und die Stria vascularis ergeben, wie bei der labyrinthären Schwerhörigkeit des Erwachsenen, verschiedene Formen und Grade der degenerativen Atrophie.

Auch kongenitale Pigmentanomalien werden hier wie in Fällen von kongenitaler Taubheit (hereditäre degenerative Taubstummheit — Hammerschlag) beobachtet.

Die Verwandtschaft mancher Fälle von kongenitaler labyrinthärer Schwerhörigkeit mit der klinischen Form der Otosklerose und den kongenitalen Formen der kretinösen, labyrinthären Schwerhörigkeit kann heute nicht mehr bezweifelt werden.

Kongenitale Bildungsanomalien im Bereiche der Schnecke und des Hörnerven stellen sicher in vielen Fällen von Otosklerose die primordialen Veränderungen dar ; zu diesen Veränderungen treten noch in embryonaler Zeit oder später erst die für die Otosklerose charakteristischen Veränderungen der knöchernen Labyrinthkapsel.

[1] Skrowaczewski, P., ' Zur Kenntnis des Hörvermögens der Deszendenz kongenital tauber Eltern,' *Monatsschr. f. Ohrenheilk.*, No. 9, 1913.

[2] Mauthner, O., ' Kongenitale Taubheit und erhaltene statische Erregbarkeit bei Missbildung des äusseren und mittleren Ohres,' *Arch. f. Ohrenheilk.*, Bd. lxxxiii, 1910.

Symptome. Tritt die Erkrankung einseitig auf und fehlen irgendwelche Erscheinungen von seiten des Vestibularis, so kann die labyrinthäre Schwerhörigkeit jahrelang symptomlos bestehen, bis sie endlich zufällig entdeckt wird. Die Entdeckung erfolgt, wenn durch eine interkurrente Erkrankung eine Ohruntersuchung nötig wird, oder wenn sich das Kind bei zufälligem Verschlusse des anderen Ohres (im Bett bei Seitenlage auf der gesunden Seite) plötzlich als schwerhörig erweist.

Tritt die Erkrankung beiderseits auf oder ist sie mit Vestibularsymptomen verbunden, so wird sie natürlich weit früher entdeckt werden.

Die Untersuchung des Hörvermögens ergibt eine zumeist mässige Schwerhörigkeit. Subjektive Geräusche scheinen sich nur selten vor der Pubertät einzustellen. Ist ein *subjektives Geräusch* vorhanden, so wird es gewöhnlich als *hohes*, nicht sehr *intensives Pfeifen* empfunden.

Vestibularsymptome sind sehr selten. Sie bestehen in einer geringen Verminderung des Gleichgewichtes mit konsekutiven geringen Gangstörungen (breitspuriger Gang).

Bogengangssymptome in Form von Schwindelanfällen und Erbrechen habe ich in derartigen Fällen nie beobachtet. Die Reflexerregbarkeit des statischen Labyrinthes war stets normal.

Verlauf und Prognose. Die Prognose der Erkrankung ist in einzelnen Fällen nicht ungünstig, die Schwerhörigkeit behält zeitlebens den gleichen geringen Grad oder nimmt in geringem Masse zu. *Das kritische Alter ist die Pubertätszeit.* Zeigt sich in der Pubertätszeit keine oder eine unwesentliche Abnahme des Hörvermögens, so ist auch für die spätere Zeit die *Prognose günstig.* Auch in allen Fällen, in welchen die Erkrankung einseitig aufgetreten und nicht durch Vestibularsymptome kompliziert ist, kann die Prognose günstig gestellt werden. Dagegen besteht in Fällen, in welchen die Schwerhörigkeit auf der erkrankten Seite in der Pubertät zunimmt, auch eine Erkrankungsgefahr für das andere, bis dahin gesunde Ohr.

Eine ungünstige Prognose bieten alle diejenigen Fälle von doppelseitiger labyrinthärer Schwerhörigkeit, bei welchen sich in der Zeit der Pubertät eine rasche Abnahme des Hörvermögens einstellt und bei welchen Anamnese bezüglich kongenitaler Erkrankungen des Gehörorgans (Schwerhörigkeit oder kongenitale Taubstummheit) eine familiäre Belastung ergibt. Prognostisch ungünstig sind auch diejenigen Fälle, bei welchen die kongenitalen Anomalien nicht nur den kochlearen, sondern auch den vestibularen Apparat betreffen.

Behandlung. Eine Besserung des Zustandes oder eine Wiederherstellung der normalen Funktion ist vollkommen ausgeschlossen. In stationär bleibenden Fällen kann daher von jeder Lokalbehandlung abgesehen werden. In progredienten Fällen ist die systematische Galvanisation des Acusticus angezeigt. Die eine Elektrode wird knapp über dem Tragus oder in der Fossa mastoidea angesetzt, die andere am Nacken, auf der Brust oder in der Hand. Von manchen Autoren wird die Verwendung einer geteilten Anode an beiden Ohren (Kathode am Nacken)

oder Galvanisation quer durch den Kopf empfohlen. Der galvanische Strom wird dreimal wöchentlich durch 5–6 Wochen appliziert, dann folgt eine Pause von 4–6 Monaten. Ausserdem ist die Anwendung von Jod, Phosphor, der Gebrauch von Jodbädern oder eine antiluetische Kur (selbst in anamnestisch vollständig negativen Fällen) gerechtfertigt.

II. *Die kongenitalen Erkrankungen des statischen Labyrinthes.* — Hier handelt es sich ausnahmslos um Fälle mit überaus interessanten Anomalien der reflektorischen Erregbarkeit des Bogengangapparates. So kann sich bei einem im übrigen durchaus normalen Labyrinth der Bogengangapparat auf beiden Seiten für die Erregung auf dem Drehstuhl unerregbar zeigen. Selbst nach intensiver, langdauernder Drehung kommt nicht der geringste Schwindel oder Nystagmus zustande. Derselbe Fall ergibt aber positive Reaktion, d. h. positive und normale Erregbarkeit, wenn der Bogengangapparat nicht durch Körperdrehung, sondern kalorisch oder elektrisch geprüft wird. Andererseits gibt es aber auch Fälle, in welchen die kalorische oder galvanische Erregbarkeit negativ ist, während die übrige Erregkarkeit in normaler Weise erhalten ist.

Die Fälle sind ziemlich selten und werden nur zufällig gelegentlich anderweitiger Untersuchung des Gehörorganes entdeckt. Die kongenitale Anomalie selbst verläuft vollständig symptomlos und verursacht dem Patienten nicht die geringsten Beschwerden. So wurden derartige ‘ galvanische Versager ’ von Kreidl und Pollak gelegentlich der unfangreichen Untersuchungen dieser Autoren über das Verhalten der Labyrinthnormalen bei Drehung oder galvanischer Reizung entdeckt.

Zu den kongenitalen Anomalien des statischen Labyrinthes ist auch die bei hereditärer Lues mitunter beobachtete *Uebererregbarkeit* oder abnormale Erregbarkeit des Bogengangapparates zu zählen (s. d.).

In diesen Fällen erhalten wir *Kompressions- oder Aspirations-Nystagmus*, während das normale, statische Labyrinth auf die Luftdruckerhöhung und -verminderung im Gehörorgan oder im Mittelohr nicht reagiert.

Für die anderen Qualitäten der Erregbarkeit des statischen Labyrinthes ergeben sich in diesen Fällen normale Verhältnisse, oder es kann trotz der gesteigerten mechanischen Erregbarkeit eine der anderen Formen der Erregbarkeit vorübergehend oder dauernd vermindert, ja sogar erloschen sein. Allerdings scheint in diesen Fällen auch die positive, mechanische Erregbarkeit sehr zu variieren.

Eine Behandlung dieser Erkrankungsfälle kommt nach dem oben gesagten nicht in Betracht.

III. *Die progressive, labyrinthäre Schwerhörigkeit des jugendlichen Alters.* Politzer hat als erster über Fälle berichtet, bei welchen am Ende des zweiten Dezenniums eine rasch zunehmende beiderseitige Schwerhörigkeit eintritt, die nach kürzerer oder längerer Zeit zu hochgradiger Herabsetzung des Hörvermögens oder völliger Taubheit führt. Wertvolle Beiträge zur Kenntnis dieser Zustände verdanken wir Manasse.

Die Funktionsprüfung ergibt in solchen Fällen alle Symptome einer Erkrankung des schallperzipierenden Apparates. Politzer erblickt das

anatomische Substrat dieser Erkrankung in einer idiopathischen Atrophie des Acusticus. Ob tatsächlich alle Fälle überhaupt einem einheitlichen anatomischen Typus entsprechen, ist fraglich. Sicher gehört ein Teil der Fälle der Gruppe der erworbenen labyrinthären Schwerhörigkeit an. In einer anderen Gruppe von Fällen entwickelt sich die progressive Schwerhörigkeit des jugendlichen Alters auf der Basis einer kongenitalen Labyrinthschwerhörigkeit.

Die Erkrankung bleibt fast stets auf die Schnecke beschränkt und greift auf das statische Labyrinth nicht über. In ätiologischer Beziehung sind erbliche Belastung mit Ohr- oder Geisteskrankheiten oder anderen wichtigen kongenitalen Bildungsanomalien und hereditäre Lues hervorzuheben. Das Haupt- und oft einzige Symptom der Erkrankung bildet eine in kurzen Epochen ziemlich rasch zunehmende Labyrinthschwerhörigkeit. *Subjektive Geräusche* sind im Kindesalter nur selten vorhanden. Die Untersuchung ergibt ein normales Trommelfell, keinerlei Veränderungen im Mittelohr und führt mit völliger Klarheit zur Diagnose der inneren Ohrerkrankung.

Die Erkrankung tritt am *Acusticus isoliert* auf, kann jedoch ausnahmsweise auch mit der Erkrankung anderer Hirnnerven, vor allem mit Erkrankungen des Opticus und des Olfactorius vergesellschaftet sein.

Manche dieser Fälle gehören in das Erkrankungsgebiet der Akromegalie, der Schädeldeformitäten (Turmschädel) und der so seltenen Leontiasis ossea, bei welcher durch die Knochenwucherung an der Schädelbasis die Hirnnerven in ihren Kanälen komprimiert werden und allmählich atrophieren.

Behandlung. Die Behandlung ist leider aussichtslos, die Prognose des Leidens in allen Fällen ungünstig. An einen therapeutischen Erfolg kann nicht gedacht werden.

Die Behandlung beschränkt sich auf Galvanisation des Acusticus und auf die innere Verabreichung von Jod und Phosphor. Fallweise sind Salvarsaninjektionen angezeigt. Bei Akromegalie kommt die chirurgische Behandlung des Grundleidens in Betracht.

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B. Die postembryonal auftretenden nicht eitrigen Labyrinth-Erkrankungen

(*a*) Die primären Erkrankungen des inneren Ohres

I. *und* II. *Die akuten und chronischen traumatischen Erkrankungen des inneren Ohres*

Sowohl einmalige heftige Schalleindrücke als auch anhaltender Lärm können pathologische Veränderungen im Labyrinth nach sich ziehen. Die Anatomie dieser Veränderungen ist von Stein (*Anat. d. Taubstummh.*, Bd. iii, 1906) und Wittmaack (*Zeitschr. f. Ohrenheilk.*, Bd. liv, 1907) festgestellt worden. Es entwickeln sich Degenerationen im inneren Gehörorgane — ob hiebei der Kopfknochenleitung oder der Luftleitung mehr Bedeutung zukommt, ist derzeit noch nicht klargestellt. Nager und Siebenmann (*Verhandl. d. Ges. deutscher Naturf. u. Aerzte*, Karlsruhe, 1912) und Yoshii (*Zeitschr. f. Ohrenheilk.*, Bd. lviii) schreiben bei der Entstehung der professionellen Schwerhörigkeit der Luftleitung die Hauptbedeutung zu. Die Klinik des Kapitels der professionellen Schwerhörigkeit ist von Röpke (*Berufskrankh. d. Ohres*, 1902) erschöpfend behandelt worden.

Eingehende experimentelle Untersuchungen über die Schädigung des Gehörorgans durch Schalleinwirkung hat Wittmaack (*Zeitschr. f. Ohrenh.*, Bd. liv) angestellt. Wittmaack unterscheidet eine akute Form, die sich mit grosser Schnelligkeit auf sämtliche Nervenfasern und Ganglienzellen ausdehnt, und eine mehr chronische Form mit langsamerem Fortschreiten. Wittmaack findet die primäre Schädigung im Nervenganglienapparat, die degenerativen Veränderungen im Cortischen Organ stellen sich sekundär ein. Die degenerative traumatische Neuritis des Octavus durch Schalleinwirkung ist ihrem Wesen nach der senilen, bezw. arteriosklerotischen, infektiösen, kachektischen, diabetischen Neuritis nervi octavi an die Seite zu stellen. Siebenmann und Yoshii (*Verhandl. d. Deutschen otolog. Ges.*, Bd. xvii, 1908) fanden bei ihren Experimenten nach der Einwirkung von Tönen oder Detonationen auf das Gehörorgan unter anderem primäre Erkrankungen im Cortischen Organ, mehr oder weniger lokalisiert nach der Helmholtz'schen Theorie. Traumatische Schädigungen des inneren Ohres vermag nach Blegvad (*Nord. med. Ark.*, Bd. xxxix) das Kopftelephon herbeizuführen, zumal bei nervösen oder nervös veranlagten Leuten. Die Verbindung von traumatischer Labyrinthverletzung und allgemein traumatischer Neurose vermag an sich leichte Schwindelanfälle mit einem Komplex schwerer und abnormer Begleiterscheinungen

(vasomotorische Störungen, Störungen der Herztätigkeit, Angstgefühl, Ohnmacht u.s.f.) zu umgeben.

Als traumatische Verletzungen wurden an Caissonarbeitern von Alt *Monatsschr. f. Ohrenheilk.*, 1897) und Heller Hyperämie des Labyrinthes und kleine Hämorrhagien gefunden.

Akute Erkrankungen durch Luftdruck können zu Blutergüssen im inneren Ohre führen (Caissonarbeiter, Luftschiffer u.s.f.) (Alt und Heller). Erschütterungen des Kopfes können nach Rhese (*Zeitschr. f. Ohrenheilk.*, Bd. lii, p. 320) labyrinthschädigend wirken. Die Erscheinungen treten zum Teil sofort nach der Verletzung zutage, zum Teil entwickeln sie sich allmählich.

Nach Mauthner (*Archiv f. Ohrenheilk.*, Bd. lxxxvii) handelt es sich bei den Läsionen des inneren Ohres nach mechanischen oder sensugenen Traumen, nicht um ein isoliertes Befallensein des inneren Ohres. Anamnestische Daten und objektive Zeichen bestätigen vielmehr überall den überstandenen oder noch bestehenden Grad der nebenhergehenden traumatischen Gehirnerschütterung. Die für die innere Erkrankung charakteristische Verkürzung der Kopfknochenleitung nach Traumen hat somit, wie auch der Schwindel, häufig eine zerebrale (meningitische), beziehungsweise zerebellare Komponente. Hofer und Mauthner (*Verletzungen des Ohres bei katastrophalen Explosionen*, Wien, Safár, 1913) haben bei der Untersuchung der Folgeerscheinungen einer katastrophalen Pulverexplosion einige Fälle von schwerer, und eine beträchtliche Anzahl leichter, traumatischer Innenohrläsionen feststellen können. Die leichten Läsionen zeigten ausnahmslos einen günstigen Verlauf.

Die professionelle Schwerhörigkeit ist eingehend von Röpke (*Berufskrankheiten des Ohres*, 1902) behandelt worden. Histologische Untersuchungen über die bei Kesselschmieden auftretenden Veränderungen im innerer Ohre sind von Habermann (*Archiv f. Ohrenheilk.*, Bd. xxx und lix), Brühl (*Zeitschr. f. Ohrenheilk.*, Bd. lii) und Zange (*Archiv f. Ohrenheilk.*, Bd. lxxxvi) vorgenommen worden. In allen Fällen fanden sich degenerative Veränderungen in der Schnecke und im Nervenganglienapparat des Octavus.

Stenger (*Archiv f. Ohrenheilk.*, Bd. lxxix) unterscheidet :

(*a*) Traumatische Schädigungen des inneren Ohres mit Verletzung der knöchernen Labyrinthkapsel ;

(*b*) Solche ohne Verletzung der knöchernen Labyrinthkapsel ;

(*c*) Schädigungen des inneren Ohres ohne nachweisbare Knochenverletzung. Die dritte Gruppe begreift die sogenannte Labyrintherschütterung in sich. Nach experimentellen Studien ist Stenger geneigt, anzunehmen, dass bei der Labyrintherschütterung des Menschen Blutungen in dem Nervenapparat des inneren Ohres eine Rolle spielen. Daneben kommt aber auch den Druckschwankungen der Peri- und Endolymphe eine nicht geringe Bedeutung zu. Klinisch erscheint es für die forensische Beurteilung der Fälle von grösster Bedeutung, dass dem Ohrenarzte Gelegenheit geboten wird, Verletzte möglichst kurze Zeit

nach dem Trauma zu untersuchen. Späterhin ist oft die traumatische Aetiologie des labyrinthären Symptomenkomplexes nicht mit Sicherheit oder überhaupt nicht mehr konstatierbar.

Die anatomischen Befunde der Labyrinthfraktur sind verschieden; vorherrschend sind ausgedehnte Blutungen, die mechanisch oder in Form akut einsetzender Degenerationen zur Funktionslosigkeit des inneren Ohres führen. Durch das Labyrinth gehende Knochenbrüche sind nicht häufig. Genaue Befunde einschlägiger Fälle liegen von Politzer (*Arch. f. Ohrenheilk.*, Bd. ii), Manasse (*Zeitschr. f. Ohrenheilk.*, Bd. lix, p. 121) und Lange (*Zeitschr. f. Ohrenheilk.*, Bd. lii, p. 37) vor. Die verschiedenen Grade und Formen der Verletzungen sind ausführlich von Passow (*Die Verletzungen des Gehörorgans*, Wiesbaden, 1905) und Manasse (*Verhandl. d. Deutsch. otol. Ges.*, XIX. Vers., Referat) mitgeteilt worden. Beide Autoren berücksichtigen auch eingehend die Literatur.

Die Behandlung der Schädelbasisfrakturen hat Voss (*Verhandl. der Deutsch. otol. Ges.*, Bd. xviii) bearbeitet. Voss favorisiert die operative Behandlung.

Der nach Kopftraumen auftretende Schwindel kann nach Bárány (*Verhandl. d. Deutsch. otol. Ges.*, Bd. xvi, 1907) mit Sausen und Schwerhörigkeit verbunden sein oder auch ohne diese Symptome auftreten. Er kann sich ferner sofort nach der Verletzung oder später einstellen und ist im ersten Falle direkt durch die Verletzung des Vestibularapparates, im letzteren Falle durch die Folgezustände des Trauma (z. B. durch eine traumatisch erzeugte Sklerose der feinen Hirnarterien) verursacht.

Zu den traumatischen Labyrintherkrankungen sind auch manche Formen der Seekrankheit zu zählen.

III. *Die Erkrankungen des inneren Ohres bei Anomalien des Stoffwechsels.*

Degenerative Neuritis bei Diabetes mellitus beschreibt Wittmaack (*Zeitschr. f. Ohrenheilk.*, Bd. liii). Das Primäre sieht Wittmaack stets in der Erkrankung der Nervenzellen und Nervenfasern; die Atrophie des Cortischen Organs entwickelt sich sekundär.

Degenerative Veränderungen im inneren Ohre sind endlich beobachtet worden bei der Gicht (Ebstein, *Arch. f. klin. Med.*, Bd. lviii, und Gowers, *Handb. d. Nervenk.*, Bonn, 1892). Wittmaack hat degenerative Veränderungen im inneren Ohre bei Diabetes gefunden, Siebenmann (*Zeitschr. f. Ohrenheilk.*, Bd. xxxvi) und Panse (*Archiv f. Ohrenheilk.*, Bd. lxx, 1907) in Fällen von Krebskachexie, Manasse (*Zeitschr. f. Ohrenheilk.*, Bd. lii, 1906) und Brühl (*Zeitschr. f. Ohrenheilk.*, Bd. l) bei Nephritis.

IV. *Die Labyrintherkrankungen bei Erkrankungen des Blutes und der blutbildenden Organe*

1. Die lymphomatösen Labyrintherkrankungen: Die Erkrankungen des inneren Ohres bei Leukämie, Chlorose und den verwandten Krankheiten.

Aetiologie. Das hauptsächlichste ätiologische Moment ist in der hämorrhagischen Diathese gelegen.

Anatomie. Hyperämie des Gehörorgans, damit auch des Labyrinths, ist in Fällen von Lymphomatose ungemein häufig. Am meisten charakteristisch sind die lymphomatösen Labyrinthblutungen, die entweder umschrieben oder diffus auftreten; sie kommen entweder isoliert vor oder kombiniert mit Hämorrhagien des Mittelohrs oder endokraniellen Blutungen. Bei ausgedehnten Labyrinthblutungen füllen sich sämtliche Hohlräume des Labyrinths mit Blut, die dünnen Wandteile des häutigen Labyrinths (Membrana vestibularis, freie Sacculuswand) können einreissen. Durch die Blutung wird das Neuroepithel komprimiert und es entwickeln sich ausgedehnte mechanische Läsionen. Die Blutung nimmt von den Aestchen der Arteria auditiva interna oder den Kapillaren der Stria vascularis ihren Ausgang. Mässige Labyrinthblutungen können zur Kompression des membranösen Labyrinths, langsam und wiederholt erfolgend, zur teilweisen Obliteration des Labyrinths führen. Aber auch Blutungen im inneren Gehörorgan und entlang des Acustico-Facialis werden beobachtet. Durch Blutergüsse an den Wasserleitungen im inneren Gehörgang wird die normale Lymphzirkulation im inneren Ohre aufgehoben. Hiedurch können Gestaltveränderungen der häutigen Teile, besonders Ektasien des Sacculus oder des häutigen Schneckenkanals hervorgerufen werden. Nach längerem Bestande führen Labyrinthblutungen zu degenerativen Veränderungen an den Nervenendstellen, vor allem im Cortischen Organ, zur Ausscheidung von Fibrin, später Bindegewebsnetzen, endlich zur Knochenneubildung in den perilymphatischen und endolymphatischen Räumen und zur Ausscheidung von Blutpigment im Labyrinth. In vielen Fällen handelt es sich nicht um reine Blutung, sondern um eine hämorrhagische, mit Blutung verbundene Labyrinthentzündung. Die entzündlichen Veränderungen bestehen in kleinzelligen, anscheinend rein lymphomatösen, jedoch von Mikroorganismen durchsetzten und später eitrig zerfallenen Infiltraten, die herdförmig innerhalb des Gewebes neben Blutungen nachgewiesen werden können.

Lymphoide Infiltrate finden sich im perilymphatischen Bindegewebspolster des inneren Ohres, mitunter in der Stria vascularis, im Ligamentum spirale, am Hirnstamme und im Wurzelgebiete des Octavus.

Den Labyrinthhämorrhagien können akute Adhäsivprozesse vorausgehen. Die Peri- und Endolymphe, die normalerweise bei Doppelfärbung ungefärbt bleibt, nimmt dann im histologischen Bilde zumeist eine eosinrote, manchmal blaurote Farbe an und lässt eine streifen- oder strahlenförmige Struktur erkennen.

In länger dauernden Fällen können sich sekundäre Veränderungen in Form von pathologischer Bindegewebs- und Knochenwucherung und degenerativer Atrophie des Sinnesepithels, sowie des peripheren und zentralen Acusticus einstellen.

Lymphoide Infiltrate und Tumoren finden sich in Fällen von Leukämie,

Chlorom und Lymphosarkom. In Fällen von chronischer Leukämie können ausnahmsweise ohne Vorangehen von Blutungen oder lymphoider Infiltration mässige degenerative Veränderungen am Octavus und seinen peripheren Nervenendstellen auftreten.

Anämie des Labyrinths bildet das Ergebnis von bindegewebiger Verödung der Blutgefässe nach Labyrinthblutung, bezw. nach Verlegung der regionären kleinen Arterien durch lymphoide Tumoren.

Häufigkeit und Vorkommen. Labyrintherkrankungen finden sich sehr häufig in den Fällen von akuter und akut verlaufender Leukämie, annähernd gleich häufig in den Fällen von myelogener, lymphatischer oder gemischter Leukämie und Chlorom. Seltener sind die Erkrankungen des inneren Ohres in Fällen von chronischer Leukämie und Lymphosarkom. Labyrinthblutungen treten besonders bei jenen Kranken auf, bei denen auch an anderen Körperregionen Hämorrhagien entstanden sind; besonders bei den akuten Lymphomatosen werden Ohrblutungen beobachtet, die sich hauptsächlich auf Grund einer akuten Hyperämie der Ohrregion der Schädelbasis oder des ganzen Kopfes entwickeln. Die Labyrinthblutungen können isoliert bestehen oder mit Mittelohr- bezw. mit endokraniellen Blutungen verbunden sein. Häufig gehen die Labyrinthblutungen mit Blutungen im Acustico-Facialis und seinen Nervenscheiden einher.

Symptome. Umschriebene Labyrinthblutungen können symptomlos verlaufen. Ausgedehnte Labyrinthblutungen führen in Form einzelner Attacken oder unvermittelt zu völliger Taubheit und zu Verlust der Reflexerregbarkeit des statischen Labyrinths.

Befund. Der otoskopische Befund bietet zumeist nichts charakteristisches oder weist auf eine gleichzeitige hämorrhagische Erkrankung des schalleitenden Apparates hin. Ausnahmsweise ist, ohne dass Symptome von seiten des schalleitenden Apparates vorhanden wären, eine periphere Blutgefässinjektion am Trommelfell festzustellen. Die Funktionsprüfung ergibt alle Zeichen einer Erkrankung des inneren Gehörorgans.

Diagnose. Die Diagnose bereitet gewöhnlich keine Schwierigkeiten. Es empfiehlt sich in allen Fällen von hämorrhagischer Otitis oder bei Verdacht auf Labyrinthblutungen einen mikroskopischen Blutbefund aufzunehmen, der bezüglich der Aetiologie der Labyrintherkrankung Aufschluss geben wird. Labyrinthblutungen unbekannter Aetiologie gibt es nicht. Der klassische Fall Ménières 1862 gehört sicher in die Gruppe der leukämischen Labyrinthblutungen.

Behandlung. Die Behandlung beschränkt sich auf symptomatische Massnahmen.

Verlauf und Prognose. In Anbetracht der Grundkrankheit ist der Verlauf in Fällen von perakuter Leukämie oder Chlorom ein absolut ungünstiger. Rein degenerative Veränderungen in Fällen von chronischer Leukämie können stationär bleiben. Eine Labyrinthblutung als Todesursache ist nicht denkbar.

V. *Die Krankheiten des inneren Ohres bei den Konstitutionskrankheiten.— Die Krankheiten des inneren Ohres bei lymphatischer Konstitution und Rachitis.*

Bei lymphatischer Konstitution und Rachitis kommen zunächst Veränderungen des Blutgefässgehaltes des inneren Ohres in Betracht (Anämie des Labyrinths), die sich in lästigen subjektiven Geräuschen, Gehörstörungen und dadurch verursachten Störungen des Allgemeinbefindens äussern können. Bedeutendere Abnormitäten der Labyrinthkapsel infolge von hochgradiger Rachitis können wohl kaum zu Veränderung en des häutigen Labyrinths oder des Hörnerven führen.

VI. *Die Krankheiten des inneren Gehörorgans bei endemischem Kretinismus. — Die endemische, konstitutionelle Schwerhörigkeit und Taubheit.*

Meinen Ausführungen liegen die Erfahrungen zugrunde, die ich in der Beobachtung des von v. Wagner in Evidenz gehaltenen und behandelten Materiales von endemischem Kretinismus gewonnen habe. Das Material von v. Wagner umfasst den Bezirk Judenburg in Steiermark, in welchem der endemische Kretinismus ganz besonders verbreitet ist. Meine Untersuchungen habe ich im Jahre 1904 begonnen und seither ununterbrochen fortgesetzt.

Vorkommen. Schwerhörigkeit ist bei Kretinismus eine ungemein häufige Erscheinung. Bei der Untersuchung eines grossen Materiales hat sich herausgestellt, dass kaum ein Viertel sämtlicher Kretinen eine normale Hörschärfe besitzt. In der grossen Mehrzahl der Fälle findet sich eine mittelgradige Herabsetzung des Hörvermögens, 20–30 Prozent der schwerhörigen Kretinen weisen eine hochgradige Herabsetzung der Hörschärfe auf, bei ungefähr 5 Prozent findet sich vollkommene Taubheit.

In der Mehrzahl der Fälle sind die *Ohrenerkrankungen* der Kretinen *kongenitalen Ursprunges* oder bestehen wenigstens seit frühester Kindheit. In anderen Fällen entwickeln sich die Hörstörungen wie auch die übrigen kretinischen Symptome nach akuten *Infektionskrankheiten* und nach anderen schweren Allgemeinerkrankungen oder sie treten nach diesen Erkrankungen mindestens deutlicher als vorher (oder unvermittelt) zutage. Die kretinische Taubheit ist dagegen, wie die sonstigen schweren kretinischen Zeichen, stets kongenitalen Ursprungs.

Bircher schlug vor, die mit dem endemischen Kretinismus verbundene, ihn häufig charakterisierende Form der Taubstummheit als endemische Taubheit gegenüber allen anderen Formen der Taubheit zu isolieren. Die *endemische Taubstummheit Birchers* entspricht der endemischen konstitutionellen Taubheit Hammerschlags. Bircher betont die Verschiedenheiten in der körperlichen und geistigen Entwicklung der taubstummen Kretinen: ‘Man trifft solche, die abgesehen von Gehör und Sprachmangel nach allen Richtungen wohl ausgebildet sind, während wiederum andere physisch und psychisch alle Grade der Dekadenz zeigen.’

Bloch gelangt nach seinem Materiale zu der Aufstellung des Begriffes der *dysthyren Schwerhörigkeit*. Er betont die Häufigkeit der mittelgradigen Herabsetzung der Hörschärfe und die Häufigkeit von Sprachstörungen. Bloch hebt auch den günstigen Einfluss der Schilddrüsenfütterung auf die Schwerhörigkeit der Kretinen hervor.

Siebenmann wendet sich gegen die Formulierung des Begriffes der dysthyren Schwerhörigkeit. Er stützt sich darauf, dass er in einem Falle von totaler Aplasie der Schilddrüse das Gehörorgan, besonders das Labyrinth, anatomisch intakt gefunden hat. Dies ist jedoch meiner Erfahrung nach nicht beweisend. Auch ich habe an dem grossen, von mir untersuchten Kretinenmaterial in dem einen oder anderen Falle bei nicht nachweisbarer Schilddrüse eine ziemlich gute Intelligenz und ein gutes Hörvermögen feststellen können; zumindest war in einzelnen dieser Fälle das Labyrinth intakt. Bloch hat allerdings Labyrinthveränderungen bei Kretinen weit häufiger gefunden, als ich solche in dem von mir untersuchten Materiale beobachten konnte, und hebt auch hervor, dass die dysthyre Schwerhörigkeit keineswegs häufig oder gar stets eine hochgradige ist. Er verweist auf Fälle mit Hörstörungen allerleichtesten Grades, bei welchen sich die Hypothyreoiden der Hörstörung im gewöhnlichen Leben nicht bewusst werden, und bei denen dieselbe erst durch eine genaue Untersuchung des Ohres aufgedeckt werden kann. Bloch fasst möglicherweise unter der dysthyren Schwerhörigkeit verschiedene Krankheitsformen zusammen, die pathologisch-anatomisch und ätiologisch nicht zusammengehören. Trotzdem vermag man jedoch unter dem Eindrucke, den man durch die Beobachtung eines grösseren Kretinenmateriales gewinnt, und angesichts der Tatsache des überraschend hohen Prozentsatzes der nicht normal hörenden, kropfigen Kretinen und der Häufigkeit hochgradiger Schwerhörigkeit oder Taubheit bei den Hypo- und Athyreoiden an dem Begriff der Bloch'schen dysthyren Schwerhörigkeit festzuhalten.

Kocher nimmt einen zentralen Sitz der Taubheit an und spricht von einer *auditiven sensorischen Aphasie*.

v. Wagner unterscheidet gewisse *Typen von Kretinen*. Der zwerghafte Vollkretin ist charakterisiert durch die vollständige Ausbildung aller kretinischen Symptome: zwerghaften Wuchs, hochgradiges Myxödem, vollständiges Ausbleiben der Geschlechtsentwicklung. In diesen Fällen besteht Hypothyreoidismus; man findet nur einen kleinen atypischen Kropfknoten oder die Schilddrüse ist überhaupt nicht nachweisbar. Zu diesem Typus gehören viele Fälle von hochgradigem Blödsinn, hochgradiger Schwerhörigkeit oder vollkommener Taubheit; seltener finden sich hier Kretinen ohne nennenswerte Schädigung des *Gehörvermögens* und mit guter Intelligenz.

In anderen Typen des Kretinismus tritt als hauptsächliches Merkmal die *endemische Taubheit* zutage; es bestehen schwere Hörstörungen oder gänzliche Taubheit und bedeutende Beeinträchtigung der psychischen Funktionen.

Unter den zwerghaften Halbkretinen sind leichtgradige Mittelohr- oder Labyrinthaffektionen nicht selten, schwere Hörstörungen oder Taubheit werden dagegen bei diesem Typus des Kretinismus nicht angetroffen.

Bei dem Typus des kretinischen Zwerges (charakterisiert durch Zwergwuchs, Myxödem und kindliche Genitalien) finden sich keine nennenswerte Störungen der Intelligenz oder des Gehörorganes.

Ein sehr häufiger Kretinentypus wird durch die starke Ausbildung des *Kropfes* beherrscht. In diesen Fällen, in welchen alle anderen kretinischen Zeichen hinter den Kropf zurücktreten, ist ein normal Hörender kaum zu finden. Fast ausnahmslos zeigen alle diese Fälle Hörstörungen, doch ist völlige Taubheit selten. In dieser Gruppe finden sich sowohl Halb- als Vollkretinen und sowohl Fälle mit mindergradiger als mit hochgradiger Hörstörung; der taubstumme Vollkretin und der taubstumme Halbkretin gehören gleichfalls diesem Typus an. Endlich findet sich noch ein Kretinentypus, der durch *Taubstummheit und Kropf* charakterisiert ist. In diesem Falle bestehen keinerlei Wachstumsstörungen, keine kretinische Gesichtsbildung, kein Myxödem, im Bereiche der Genitalsphäre keine Störungen. Die Intelligenz ist in diesen Fällen von kretinischer Taubheit gewöhnlich nicht erheblich gestört, doch ist eine gewisse Gemütsabstumpfung und Schwerfälligkeit fast in allen Fällen ausgeprägt.

Anatomie. In einer grossen Anzahl von Fällen bilden katarrhalische Veränderungen der Mittelohrräume die Ursache der Schwerhörigkeit. Eine bedeutende Vergrösserung der Rachentonsille, sowie chronische Verdickungen und ödematöse Schwellungen der Nasenrachenschleimhaut, stellen an Kretinen einen ungemein häufigen Befund dar. Die katarrhalischen Mittelohrveränderungen entwickeln sich auf der Grundlage der chronischen Nasenrachenveränderungen, doch werden typische Formen des exsudativen Mittelohrkatarrhs nur ausnahmsweise angetroffen. Die Häufigkeit der bei Kretinen gefundenen Mittelohrerkrankungen hängt auch damit zusammen, dass die myxomatösen Schwellungen der Schleimhaut des Nasenrachentraktes sich auf die Tuben- und Trommelhöhlenschleimhaut fortpflanzen (v. Wagner).

Moss stellte (*Zeitschr. f. Ohrenheilk.*, Bd. xi) in einem Falle von Idiotie (fötaler Chondrodystrophie) Hyperostose der hinteren und inneren Wand der Trommelhöhle beiderseits fest.

Hammerschlag (*Archiv f. Ohrenheilk.*, Bd. lv) fand an einem zwerghaften Mädchen linkerseits die Mittelohrräume verkleinert und den Steigbügel mit dem Amboss starr verbunden. Die Steigbügelschenkel waren plump, die Platte auf ein Drittel der normalen Grösse verkleinert.

Siebenmann konstatierte in einem von ihm anatomisch untersuchten Falle von Aplasie der Schilddrüse keinerlei Veränderungen, die eine wesentliche Funktionsstörung des Gehörorgans hätten bedingen können (s. o.). In einem anderen Falle fand Siebenmann bei einem idiotischen Kretin das Cortische Organ niedriger als sonst und den Tunnelraum auffallend weit.

Habermann (*Zeitschr. f. Ohrenheilk.*, Bd. xlvii, XIII. Vers. d. Deutsch. otol. Ges. 1904) fand Entwicklungsstörungen am Cortischen Organ und Verlagerung der Ganglienzellen des Ganglion spirale.

Veränderungen am Ohrskelett und am häutigen Labyrinth hat Manasse nachgewiesen. Er fasst die Veränderungen am Knochen als die primären und kongenitalen, die an den Weichteilen als die sekundären auf.

Die Veränderungen des inneren Ohres bestehen in manchen Fällen in degenerativer *Atrophie des Hörnerven und der labyrinthären Nervenendstellen.* In einzelnen Fällen sind sowohl die Nervenendstellen des statischen Labyrinthes als auch das Cortische Organ von der Atrophie betroffen. In anderen Fällen ist die Atrophie auf das Cortische Organ allein beschränkt. Diese letztere Erkrankungsform nähert sich dem Typus der sacculo-cochlearen Degeneration (Degeneration der Pars inferior) bei kongenitaler Taubheit.

Einen typischen Befund in Fällen von hochgradiger Schwerhörigkeit stellt bei Kretinismus die *Ausfüllung der Nische des Schneckenfensters* durch schleimiges Bindegewebe oder Fettgewebe dar. Dieser Gewebspropfen erstreckt sich einerseits bis in die Trommelhöhlenschleimhaut, andererseits reicht er bis an die Membran des Schneckenfensters. Mitunter wird auch die Nische des Vorhofsfensters und das Steigbügellumen durch schleimiges Bindegewebe ausgefüllt getroffen.

Von vornherein ist zu erwarten, dass sich in den Fällen von kongenitaler Taubheit bei Kretinismus, die ja den Haupttypus der endemischen, konstitutionellen Taubheit und Schwerhörigkeit darstellt, sehr verschiedene anatomische Ohrbefunde ergeben. In einem von mir beobachteten Falle von hochgradiger labyrinthärer Schwerhörigkeit waren in der Labyrinthkapsel *pathologische Knochenherde* vom Typus der bei der Otosklerose gefundenen *Ostitis vasculosa* entwickelt. In demselben Falle fand sich auch eine *degenerative Atrophie des Nervus octavus.* In manchen Fällen von Taubheit scheint eine mehr oder weniger vollständige Obliteration des häutigen Labyrinthes mit Aplasie der Nervenendstellen und einem in Resten entwickelten oder vollständig verkümmerten Octavus die anatomische Ursache der Taubheit zu bilden.

Partielle Obliteration des Schneckenkanales fand ich an einem kretinischen, hochgradig schwerhörigen Hunde. Das Cortische Organ wies in diesem Falle streckenweise gänzliches Fehlen der Haarzellen und einen teilweisen Defekt der Pfeilerzellen auf (Alexander).

In fast sämtlichen Fällen von hochgradiger Schwerhörigkeit und Taubheit bei endemischem Kretinismus scheint die *Degeneration der Stria vascularis* einen charakteristischen Befund zu bilden. In einem Falle fanden sich Epitheldupliкaturen und Septenbildung im häutigen Schnekkenkanal vom Typus der bei Tanzmäusen, bei kongenital tauben, unvollkommen albinotischen Katzen und Hunden gefundenen Septa.

Die bindegewebigen Anteile des Labyrinthes werden in einzelnen Fällen hochgradig atrophisch gefunden, sogar das Ligamentum spirale der Schnecke kann bis auf wenig Faserreste atrophiert sein. Dagegen

findet sich in denselben Fällen oft eine erhebliche *Bindegewebswucherung* im Canalis spiralis, so dass das atrophische Ganglion spirale von einer dicken Bindegewebskapsel umschlossen erscheint.

In einem Falle habe ich auch eine bedeutende Verdickung der membranösen Wände des Vorhofes, vor allem der äusseren Utriculuswand beobachtet.

Im Gebiet der Acusticuskerne und der zentralen Verzweigung des Acusticus, sowie in der Ausbildung der Rinde des Schläfenlappens, liessen sich in den Fällen von kretinischer Taubheit, die ich anatomisch zu untersuchen Gelegenheit hatte, keine pathologische Veränderung feststellen. Der periphere, hochgradig atrophische Octavus besitzt im Hirnstamm vollkommen normal entwickelte Kerne und zentralwärts verlaufende Faserbündel. Doch ist die Möglichkeit, dass in einzelnen Fällen Veränderungen des zentralen Acusticus die Ursache von Hörstörungen bilden können, nicht von der Hand zu weisen.

Sehr wichtig für die Deutung der Hörstörungen ist das psychische Verhalten der Kretinen. Durch das Phlegma der Kretinen, durch ihre motorische und psychische Stumpfheit, kann selbst bei einer nicht bedeutenden Herabsetzung der Hörschärfe ein hochgradiger Hördefekt vorgetäuscht werden. In den höchstgradigen Fällen von Kretinismus kann selbst bei normalem Gehörorgan jede Reaktion auf die Schalleindrücke ausbleiben.

Ueber subjektive Geräusche klagen die Kretinen nur ausnahmsweise ; die wenigen Fälle meiner Beobachtung betrafen ältere Kretinen mit wenig ausgeprägtem kretinen Habitus, gut entwickelter Psyche und leichtgradigen, katarrhalischen Mittelohraffektionen. Labyrinthschwindel bestand nur in 2 Fällen. In einem Falle handelte es sich um Ertaubung nach Meningitis cerebrospinalis an einem kretinösen Kinde. In einem anderen Falle lag eine mittelgradige Labyrinthschwerhörigkeit vor.

Funktioneller Befund und Funktionsprüfung. In Fällen von leichtgradigem Kretinismus unterscheidet sich die Vornahme der Funktionsprüfung in keiner Weise von jener an normalen Kindern. Auch hier können wir nicht erwarten, vor dem 8.–10. Lebensjahr einen verlässlichen Stimmgabelbefund zu erlangen, besonders wenn es sich um die Durchführung einer genauen und vollständigen Stimmgabelprüfung handelt. Sind dagegen die psychischen Störungen des Kretinismus bedeutend entwickelt, so wird eine genaue Funktionsprüfung unmöglich. Man muss sich dann damit begnügen, die wichtigsten Daten festzustellen (Perzeptionsdauer durch die Kopfknochenleitung, Hörbarkeit des Uhrtickens durch die Kopfknochen, Perzeptionsdauer der c^4-Gabel), um eine etwa vorhandene Labyrintherkrankung erkennen zu können. Reagiert endlich der Kretin auf akustische Eindrücke nur rudimentär, so erübrigt oft lediglich die Entscheidung, ob ein Hörvermögen überhaupt vorhanden ist oder ob Taubheit besteht.

Ein nicht unwichtiges Hilfsmittel bei der Konstatierung der Hörfähigkeit mancher Kretinen ist mir der Gang derselben. Der hochgradig

schwerhörige oder taube Kretin hat einen scharrenden, schleifenden, tastenden Gang. Breitspurigen Gang habe ich nur in denjenigen Fällen gefunden, in welchen das statische Labyrinth ausser Funktion war. *Spontanen Nystagmus* sah ich nur in 5 Fällen. In zwei derselben bestand eine degenerative Labyrintherkrankung, in den übrigen Fällen handelte es sich um hochgradig nervöse, sehr unruhige Kretinen, die ausserdem an epileptiformen Krämpfen litten.

Die Reflexerregbarkeit des statischen Labyrinthes ist in den meisten Fällen vollständig normal. In einzelnen Fällen meiner Beobachtung dauerte der reflektorisch durch 10 Drehungen oder der kalorisch ausgelöste Nystagmus beiderseits auffallend lange an (durch 30–40 Sekunden, gegenüber der normalen Zeit von 15–20 Sekunden). Da jedoch in diesen Fällen weder Spontannystagmus, noch Symptome von seiten des statischen Labyrinthes bestanden, so kann in dieser erhöhten Reflexerregbarkeit keine Krankheitserscheinung, sondern nur eine im Rahmen des Normalen befindliche Varietät erblickt werden. In einzelnen Fällen von kretinischer Taubheit war die Reflexerregbarkeit des statischen Labyrinthes erhalten, in anderen aufgehoben.

Behandlung. Die durch die Veränderungen des inneren Ohres beding ten Hörstörungen der Kretinen können in manchen Fällen durch Thyreoidinfütterung günstig beeinflusst werden. Unter den von mir beobachteten Fällen befinden sich mehrere Kretinen mit sicher konstatierter Schwerhörigkeit, bei welchen im Laufe der von v. Wagner eingeleiteten Thyreiodinbehandlung eine Besserung der Hörschärfe eingetreten ist. Die Perzeptionsdauer durch die Kopfknochen hat in diesen Fällen zugenommen, c^4 wird bedeutend länger perzipiert und die vorher durch die Kopfknochen nicht vernommene Uhr wird nun durch die Kopfknochen gehört. Zwei dieser Fälle beobachte ich bereits seit 7 Jahren. An beiden liess sich feststellen, dass die Besserung der Hörschärfe verhältnismässig rasch einsetzte und, nachdem sie einen gewissen Grad erreicht hatte, konstant blieb. Das Hörvermögen hat sich an diesen beiden Kretinen im Verlaufe der letzten 5 Jahre nicht mehr verändert. Trotz fortgesetzter Thyreoidinfütterung ist es nicht gelungen, die Veränderungen des inneren Ohres gänzlich zu beheben, es ist aber erfreulicherweise auch kein Rückfall eingetreten und die einmal erreichte Besserung ist nun seit 5 Jahren konstant.

In Fällen von kretinischer Taubheit ist die Thyreoidinfütterung aussichtslos.

In Fällen von Hörstummheit (psychischer, kretinöser Aphasie) *werden durch die Thyreoidinbehandlung meistens überraschende Erfolge erzielt.*

In der kretinischen Degeneration wird auch die endemische Taubheit mit Erfolg bekämpft. v. Wagner hat in dieser Richtung wertvolle Versuche angebahnt, indem er im Bezirk Judenburg in Steiermark in Familien, in denen bereits nachweislich kretinische Kinder vorhanden sind, den graviden Müttern und später den Neugeborenen Thyreoidintabletten verabreichen lässt.

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VII. *Die nicht eitrigen Erkrankungen des inneren Ohres bei Erkrankungen der Kreislauforgane (Arteriosklerose)*

Das Vorkommen von Ohrerkrankungen in Fällen von allgemeiner Arteriosklerose ist seit langem bekannt. Man ist vor allem gewohnt, die Altersschwerhörigkeit ätiologisch auf Veränderungen des Gehörorgans zurückzuführen, die durch die Arteriosklerose verursacht werden. Die Häufigkeit der arteriosklerotischen Labyrintherkrankungen ist in früherer Zeit, besonders wenn man nicht bloss das Greisenalter sondern auch jüngere Altersstufen berücksichtigt, gewaltig unterschätzt worden. Es ist ein Verdienst meines Assistenten C. Stein, durch jahrelanges, genaues Studium einschlägiger Fälle gezeigt zu haben, dass die Arteriosklerose sehr häufig zu Störungen von seiten des inneren Gehörorgans führt und dass für eine grosse Anzahl von inneren Ohrerkrankungen oder Einzelsymptomen von solchen, für welche bisher ätiologische Merkmale nicht aufgefunden werden konnten, Arteriosklerose als Ursache festgestellt werden kann. C. Stein hat eine erhebliche Anzahl von Fällen jugendlichen Alters gefunden, bei welchen Symptome von seiten des inneren Ohres bestanden, irgendwelche anderweitige Ohrerkrankung nicht vorlag und die folgende genaue interne Untersuchung sichere Zeichen der Arteriosklerose gegeben hat. Nach Stein ist es vor allem die Anämie, die sich bei den Erkrankungen des Zirkulationsapparates in erster Linie als klinisch bedeutsam für das Gehörorgan erweist. Die Anämie führt mitunter sowohl zu vorübergehenden, weit häufiger jedoch zu bleibenden anatomischen Veränderungen im inneren Gehörorgan.

Seligmann (*M. f. O.*, Bd. xl, p. 109) sieht in der progressiven, nervösen Schwerhörigkeit durch Acusticusatrophie eine Aufbrauchskrankheit des nicht stark genug angelegten Hörnerven.

Zerebrale Taubheit durch Apoplexie ist von Boenninghaus (*Zeitschr. f. Ohrenheilk.*, Bd. xlix, 1905) beschrieben worden. Es handelte sich um einen 45 jährigen Patienten mit alter rechtsseitiger und frischer linksseitiger Apoplexie im Bereiche des Schläfelappens.

Anatomie. Ich habe als erster den genauen histologischen Befund eines Falles von durch Arteriosklerose verursachter, labyrinthärer Schwerhörigkeit mitgeteilt. Es handelte sich um eine reine Erkrankung der häutigen Schnecke und des Hörnerven ohne wesentliche Beteiligung des übrigen Labyrinths, der mittleren und der äusseren Ohrsphäre, und unter völliger Intaktheit der Gegend der Labyrinthfenster. Das histologische Bild führte zur Aufstellung des Begriffes der progressiven Atrophie des Cortischen Organs. Diese Atrophie setzte mit Veränderungen und Schwund der Sinneszellen im engsten Sinne ein, erfasste, um sich greifend, weitere Zellgruppen der Papilla basilaris und endete mit dem degenerativen Totaldefekt der Papille. Die Stria vascularis, das Ligamentum spirale, das Ganglion spirale und der Nervus cochlearis nahmen an der Atrophie teil. In den Wänden der Arterienblutgefässe des inneren Ohres liessen sich deutlich arteriosklerotische Veränderungen nachweisen.

Meine Befunde sind von Manasse und Brühl bestätigt worden. Manasse, der die progressive labyrinthäre Schwerhörigkeit als klinische Einheit darstellt, beschreibt seine Befunde sehr eingehend.

Siebenmann, Brühl (*Zeitschr. f. Ohrenheilk.*, Bd. l), Manasse (*Zeitschr. f. Ohrenheilk.*, Bd. lii, 1906), Wittmaack (*La Clinique*, 1907) und Hennebert haben Gehörorgane von Greisen untersucht; die dabei festgestellten Veränderungen sind nach ihrer Aetiologie als arteriosklerotische zu betrachten.

Steinbrügge (*Orth. Pathol. Anat.*, 1891) fand bei allgemeiner Arteriosklerose, bei Krankheiten des Herzens, Ecchymosen im Labyrinth.

In die Gruppe der arteriosklerotischen Veränderungen des inneren Ohres gehören auch die von Alagna erhobenen Befunde von Degeneration des Ganglion vestibulare und der Radix vestibularis acustici bei einem alten Manne, der an Herzschwäche und Nephritis zugrunde gegangen war.

In die Gruppe der arteriosklerotischen Veränderungen des inneren Ohres gehört auch der von Alagna (*Zeitschr. f. Ohrenheilk.*, Bd. l) erhobene Befund von Degeneration des Ganglion vestibulare und des Radix vestibularis und acustici bei einem alten Manne, der an Herzschwäche und Nephritis zugrunde gegangen war.

Parenchymatöse Neuritis des Nervus acusticus mit sekundärer Degeneration des Ganglion spirale und des Cortischen Organs in den Basalteilen, während trotz der seit fünf Jahren bestehenden totalen Taubheit die Papilla basilaris in den oberen Windungen verhältnismässig gut erhalten war, bei einem 61 jährigen Mann, wird von Panse (*Archiv f. Ohrenheilk.*, Bd. lxx, 1 und 2, p. 20, 1906) beschrieben.

Wittmaack (*Zeitschr. f. Ohrenheilk.*, Bd. liii) beobachtete degenerative Neuritis des Octavus bei einem altersschwachen Hund.

Symptome. Nach C. Stein gehören subjektive Gehörsempfindungen zu den ersten Erscheinungen der arteriosklerotischen Ohrerkrankung; gleichzeitig mit ihnen oder kurze Zeit nach ihrem Beginn, nimmt auch das Hörvermögen ab. Die Abnahme der Hörschärfe erfolgt progredient, meist jedoch nur langsam und unmerklich. Attackenweise Verschlechterungen des Gehörs sind selten; sie schliessen sich gewöhnlich an schwere Erkrankungen, mitunter an psychische oder physische Traumen an (C. Stein). Der Schwindel der Arteriosklerotiker ist zumeist ein cerebraler, kann jedoch als Folge einer neuritischen Veränderung des Vestibularnervs oder, hervorgerufen durch eine Erkrankung des Labyrinthes selbst (Blutungen, degenerative Vorgänge), labyrinthären Ursprunges sein, und weist dann alle Charaktere desselben mit wechselnder Intensität auf.

Nach Stein treten mitunter bei arteriosklerotischen Ohrerkrankungen verschiedenartige, schmerzhafte oder unangenehme Sensationen im Bereiche des Gehörorganes auf. Stein führt dieselben auf lokale Zirkulationsstörungen (Verminderung oder Unterbrechung der Blutzufuhr zu den sensiblen Nervengebieten des Ohres) zurück und schlägt für sie den Namen ' Otalgia angiosclerotica ' vor.

Befund und Diagnose. Die Untersuchung ergibt alle Zeichen einer

Erkrankung des inneren Ohres. Der interne Befund selbst lässt gewöhnlich über die der Ohrerkrankung zugrundeliegende Erkrankung nicht im Zweifel (Erhöhung des Blutdruckes, rigide, geschlängelte Arterien, Accentuation des II. Aortentones etc). Die arteriosklerotische Ohrerkrankung kann jedoch auch die Teilerscheinung einer ausschliesslich cerebralen Gefässerkrankung sein. Sie tritt dann gewöhnlich in Gemeinschaft mit den Symptomen derselben (Kopfschmerzen, Abnahme des Gedächtnisses, geistige Ermüdbarkeit, etc.) auf.

Die Symptome von seiten des statischen Labyrinthes bestehen in einem vom Normalen abweichenden Verhalten der Reflexerregbarkeit des Vestibularapparates, doch erweist sich das statische Labyrinth in der grössten Zahl der Fälle nicht geschädigt.

Die Therapie der arteriosklerotischen Ohrerkrankung hat die zugrundeliegende Gefässerkrankung ins Auge zu fassen. Wir verweisen diesbezüglich auf die Lehrbücher der internen Medizin. In vorgeschrittenen Fällen ist keine Besserung der Krankheitserscheinungen von seiten des Ohres zu erwarten, doch wird es durch Anwendung entsprechender, hygienish diätetischer Massnahmen in vielen Fällen gelingen, das Fortschreiten des Prozesses einzudämmen oder wenigstens raschen Verschlechterungen entgegenzuwirken. In nicht zu weit vorgeschrittenen Fällen erweist sich nach Stein das Diuretin in Dosen von 0,5 gr. (4–5 mal täglich), längere Zeit fortgesetzt, sehr wirksam. Es beeinflusst vor allem den Schwindel der Arteriosklerotiker sehr günstig, vermag aber auch die subjektiven Geräusche zu mildern und etwa bestehende schmerzhafte Sensationen wesentlich zu vermindern, oft sogar gänzlich zum Verschwinden zu bringen. Auch Theobromin und Amylnitrit (V. Urbantschitsch) wird mit gutem Erfolge verwendet. Günstige Wirkung (meist allerdings nur vorübergehend) sahen Stein und Fellner auch von Vasotininjektionen. Von der lokalen Therapie ist wenig zu erwarten. Lufteinblasung und Massage sind dringend zu widerraten. Erleichternd wirkt mitunter bei intensivem Ohrensausen die galvanische Behandlung. (Anode am kranken Ohr.) In manchen Fällen ist die Katalyse nach Urbantschitsch von guter Wirkung.

VIII. *Die nicht eitrigen Erkrankungen des inneren Ohres bei den Krankheiten des zentralen und peripheren Nervensystemes*

Bei akutem und chronischem Hydrocephalus können infolge der endokraniellen Drucksteigerung im Gehörorgan zunächst mechanisch bewirkte Veränderungen am häutigen Labyrinth auftreten, die später zu degenerativen Prozessen im Nervenganglionapparat führen. Hierher gehören Veränderungen der Aquaeductae (Erweiterung des acquaeductae cochleae Habermann (Naturf.-Vers., Heidelberg, 1889). Der Befund der Depression der Reissner'schen Membran ist mit Recht als Artefakt zu deuten. Pathologische Bedeutung hat weit eher die Einwölbung des Schneckenkanals.

Cerebrale Taubheit durch Apoplexie ist von Bönninghaus (*Zeitschr. f. Ohrenkeilk.*, Bd. xlix, 1905) beschrieben worden. Es handelt sich um einen 45jährigen Patienten mit alter rechtsseitiger und frischer linksseitiger Apoplexie im Bereiche des Schläfelappens.

In Fällen von Acusticustumoren finden sich zumeist weitgehende Zerstörungen des Octavus und des häutigen Labyrinths. Im ganzen entspricht der Befund einer hochgradigen Atrophie des Nerven und des Sinnesepithels (v. Frankl-Hochwart, Alexander). Interessant ist, dass inmitten der Tumormassen in vielen Fällen färberisch vollkommen intakte Bündel des Octavus gefunden werden. Der klinische Befund zeigt gewöhnlich eine vollkommene Funktionslosigkeit des inneren Ohres bei völlig intaktem Mittelohr und der konkomitierenden Zeichen von Hirndruck, Labyrinth- und Kleinhirnschwindel. Auf die Details des hierbei gefundenen Spontannystagmus einzugehen, würde zu weit führen. Operativ werden derzeit die Acusticustumoren von der hinteren Schädelgrube nach Freilegung des Kleinhirns angegangen. In letzter Zeit sind Vorschläge für den translabyrinthären Operationsweg gemacht worden (Ruttin, Kümmel). Wie bei den Acusticustumoren sind auch bei den regionären Tumoren der Dura (Sarkom, Angiosarkom) die primären Veränderungen im Hörnerv zu finden. Die Angiosarkome der Dura führen zumeist bilateral zu umfangreichen Wucherungen im inneren Gehörorgan mit Zerklüftung und hochgradiger Atrophie des Octavus und zu Knochenkonsumption mit pathologischer Erweiterung des inneren Gehörgangs. Der Nervus cochlearis degeneriert in der Mehrzahl der Fälle vollständig. Widerstandsfähiger zeigt sich der Nervus vestibularis, die bedeutendste Resistenz bietet der Nervus facialis.

Bei Hysterie werden mitunter schwere Affektionen des inneren Ohres vorgetäuscht. Klarheit schafft eine genaue, zusammenhängende Funktionsprüfung. Das charakteristische Merkmal der Funktionsstörung im Cochleargebiete ist die fraktionierte Stimmgabelperzeption (Hammerschlag). Bei der Untersuchung des statischen Labyrinths werden zumeist heftige Gleichgewichtsstörungen beobachtet, die sich jedoch in der kritischen Beurteilung sehr leicht als nicht labyrinthäre herausstellen (Fehlen des Spontannystagmus, keine Beeinflussung der Fallrichtung durch Veränderung der Kopfstellung).

Durch Neurasthenie und Neurosen können bestehende geringgradige Veränderungen oder obsolente Krankheitsprozesse des Labyrinths mit schweren Symptomen ausgestattet werden. Besonders die neurasthenische und neurotische Komponente bei den Erkrankungen des statischen Labyrinths ist bemerkenswert. Anhaltend heftige Schwindelanfälle, bedeutende Gleichgewichtsstörung in Fällen, in welchen die Funktionsprüfung des statischen Labyrinths nur geringgradige und die Anamnese eine lange Dauer dieser Veränderungen ergibt, können geradezu als charakteristisch gelten. Anderseits führen mitunter chronische Labyrintherkrankungen zu allgemeiner Neurasthenie, die ich auf Grund von klinischer Beobachtung als labyrinthogene Neurasthenie bezeichnet

habe (*Wien. med. Woch.*, 1910 ?). Es handelt sich um Fälle von chronischer Mittelohreiterung mit geringen anatomischen oder klinischen Symptomen von seiten des Labyrinths. Trotzdem können in solchen Fällen jahrelang heftige Schwindelanfälle und chronische Gleichgewichtsstörungen bestehen. Zu diesen Krankheitserscheinungen führen dann die Kenntnisse, die sich derartige Kranke in der Otologie anzueignen wissen, und auf der Grundlage geringer irritativer Zustände im Labyrinth vermögen derartige Neurastheniker schwere labyrinthäre Störungen vorzutäuschen. Der genaue Vergleich des Symptomenkomplexes abgewogen nach den objektiven Erscheinungen (geringer Spontannystagmus, erhaltene Reflexerregbarkeit) mit den objektiven Zeichen in Fällen von schweren anatomischen Veränderungen des statischen Labyrinths führen zur richtigen Diagnose. Diese letztere ist in diesen Fällen um so wichtiger, weil es sich bei solchen Kranken nicht selten um die Frage eines operativen Eingriffes im Labyrinth handelt.

Damit kommen wir zur Frage der operativen Behandlung quälender Labyrinthsymptome bei nicht eitrigen Erkrankungen des Ohrlabyrinths. Sowohl in Fällen von fehlenden subjektiven Geräuschen als auch in Fällen von anhaltendem Labyrinthschwindel ist die Labyrinthzerstörung, bezw. die Durchschneidung des Nervus octavus, vorgeschlagen worden. Ich kann einen operativen Eingriff bei den oben genannten Symptomen nicht indiziert finden.

IX. *Die toxischen Erkrankungen des inneren Gehörorganes*

Kirchner (*Berl. klin. Woch.*, 1881, und *Monatsschrift f. Ohrenheilk.*, Bd. v, 1883) und Wittmaack (*Pflüg. Archiv*, Bd. xcv, 1903) haben experimentell festgestellt, dass durch das Chinin der Nervenganglionapparat des inneren Ohres verändert wird. Sekundär stellen sich sodann pathologische Veränderungen im Cortischen Organ selbst ein. Aehnliche, nur weniger intensive Wirkung übt die Salizylsäure und das Arsen (Haike, *Archiv f. Ohrenheilk.*, Bd. lxiii; Blau, *Archiv f. Ohrenheilk.*, Bd. lxi und lxv) auf das Labyrinth aus. Zytowitsch (*Russ. Wratsch*, 1908 und 1910) und v. Frankl-Hochwart stellten einen das Labyrinth schädigenden Einfluss des Alkohols und Nikotins fest. In chronischen Fällen handelt es sich nicht um eine direkt auf das Labyrinth ausgeübte toxische Wirkung, sondern um eine indirekte Schädigung des Labyrinths infolge der durch Alkohol oder Nikotin verursachten Arteriosklerose.

Alagna (*Zeitschr. f. Ohrenheilk.*, Bd. lix, p. 361) beobachtete nach experimenteller Injektion von Nikotin schwere Degenerationen des Ganglion spirale.

Wittmaack hat degenerative Neuritis des Cochlearis (*Zeitschr. f. Ohrenheilk.*, Bd. li) experimentell bei Tieren durch Vergiftung mit salicylsaurem Natron festgestellt. Die Veränderungen betrafen übereinstimmend vorzugsweise das Ganglion spirale der Schnecke und setzten sich bei grösserer Stärke und längerer Dauer der Giftwirkung auch auf

die Nerven und das Cortische Organ fort. Dagegen wurde das Ganglion vestibulare wie überhaupt der vestibulare Teil des Hörnerven mit seinem Endorgan weit weniger ergriffen.

Atrophie des Sinnesepithels im vestibularen und cochlearen Anteil des Labyrinths als Folge der Einwirkung von Radiumstrahlen hat Marx (*Zeitschr. f. Ohrenheilk.*, Bd. lix, p. 192) gefunden. Die Veränderungen zeigten sich durchschnittlich ein halbes Jahr nach der experimentellen Radiumeinwirkung.

X. *Die infektiösen, nicht eitrigen Erkrankungen des inneren Ohres*

1. *Bei den akuten Infektionskrankheiten.*—Aetiologie und Vorkommen. Bei sämtlichen akuten Infektionskrankheiten besteht die Möglichkeit der Entwicklung akuter Neuritiden des Octavus, die zumeist zu bleibenden mehr oder minder hochgradigen Veränderungen des Nerven und des Labyrinths führen. Nur ausnahmsweise bilden sich die entzündlichen Veränderungen unter Wiederherstellung der normalen Funktion vollkommen zurück.

Gleichzeitig mit der Erkrankung des Nerven, oder kurze Zeit danach, erleidet auch das Labyrinth pathologische Veränderungen, sei es, dass es an der Entzündung teilnimmt oder dass rein sekundär degenerative Veränderungen im Labyrinth auftreten.

Besonders charakteristisch ist das Auftreten und der Verlauf dieser akuten Neuritiden oder Neurolabyrinthitiden bei der Parotitis epidemica (Mumps).

An zweiter Stelle der Häufigkeit der infektiösen Neurolabyrinthitis steht der Typhus; seltener werden hiehergehörige Fälle bei Influenza, Erysipel oder akutem Gelenksrheumatismus beobachtet. Degenerative Neuritiden des Octavus kommen endlich bei der Diphtherie, Scharlach, Masern, Pertussis, Pneumonie, Meningitis cerebrospinalis in Betracht. Ob bei der Dysenterie der Säuglinge nicht eitrige Erkrankungen des Labyrinths auftreten, ist bisher nicht entschieden.

Anatomie. Ecchymosen im Labyrinth wurden bei Mumps, Typhus, sowie bei anderen akuten Infektionskrankheiten festgestellt. Moos (*Zeitschr. f. Ohrenheilk.*, Bd. xiii) fand in Fällen von Pertussis Labyrinthhämorrhagien. Wittmaack (*Zeitschr. f. Ohrenheilk.*, Bd. liii) fand konkomitierende Neuritis nervi acustici bei Scharlacheiterung, degenerative Neuritis mit Atrophie des Hörnerven nach Typhus. Degenerative Veränderungen im Ganglion spirale nach experimenteller Injektion von Diphtherietoxin hat Alagna (*Zeitschr. f. Ohrenheilk.*, Bd. lix) festgestellt. Lewin (' Ueber Neuritis (Neurolysis) acustica toxica und über die Veränderungen der zugehörigen Ganglien bei der Diphtherie,' *Zeitschr. f. Ohrenheilk.*, Bd. lxvii, 1913) fand degenerative Veränderungen im Octavus und in den peripheren Octavus-Ganglien in einer beträchtlichen Anzahl von Fällen mit Diphtherie.

Die anatomischen Labyrinthveränderungen in dem Frühstadium der

Erkrankung sind nicht genau bekannt, besonders für die nicht eitrigen Erkrankungen des inneren Gehörorgans im Verlaufe des Typhus sind exsudative Prozesse im inneren Ohre mit frühzeitigen Veränderungen der Stria vascularis anzunehmen. In einzelnen Fällen können auch Labyrinthblutungen oder hämorrhagische Entzündungen die Ursache der Erkrankung bilden: die Labyrinthveränderungen im Spätstadium der Erkrankung bestehen in Degeneration des Cortischen Organs, der Stria vascularis und des Ganglion spirale, sowie des Schneckennerven. In einzelnen Fällen sind der Vestibularapparat und Vestibularnerv von der Erkrankung gleichfalls ergriffen, in anderen Fällen bleibt der Vestibularapparat mit seinem Nerv normal.

Die Erkrankungen des Gehörorgans bei epidemischer Parotitis.

Anatomie, Vorkommen. Bei epidemischer Parotitis ist das Auftreten einer Neurolabyrinthitis zu befürchten. Die Entzündung kann auf den vestibularen oder den cochlearen Teil des Octavus, resp. des Labyrinths, beschränkt sein. In einem Teil der Fälle dehnt sich jedoch die Erkrankung auf den gesamten Octavus aus und kann sogar auf den Facialis übergreifen, so dass sie das ganze Nervenpaket des inneren Gehörorganes umfasst.

In einem Teil der Fälle handelt es sich sicher um akut-entzündliche, rückbildungsfähige Veränderungen vom Typus der serösen Entzündung; in anderen Fällen liegt eine infektiöse Entzündung vor, die unvermittelt oder infolge von sekundären, degenerativen Prozessen im Labyrinth oder im Octavus zur Beeinträchtigung oder Vernichtung der physiologischen Funktion des inneren Ohres führt. In der Mehrzahl der Fälle tritt die Ohrerkrankung einseitig auf.

Die das Gehörorgan betreffenden Störungen bei Mumps bringt Mauthner (*Die Erkrankung des Nervus octavus bei Parotitis epidemica*) in drei Gruppen:

I. Fälle mit dauernder Aufhebung der Funktion (Taubheit oder Taubheit mit statischer Unerregbarkeit).

II. Hochgradige Störungen: (*a*) mit vorübergehender Aufhebung der Funktion; (*b*) mit vorübergehender schwerer Schädigung der Funktion.

III. Geringgradige Störungen (meist nur durch die kontinuierliche Reihe und die exakte Vestibularisprüfung sicher nachweisbar).

Die klinischen Zeichen aller drei Gruppen nähern sich am meisten dem Bilde der akuten toxischen und infektiösen Neuritis des Nervus octavus. Die geringgradigen Störungen (Gruppe III), welche Mauthner zum ersten Male beschrieben hat, sind geeignet, den Rahmen des klinischen Bildes einigermassen zu erweitern und analoge Störungen bei anderen akuten Schädigungen vermuten zu lassen.

Wittmaack hat als erster den experimentellen und pathologischen Nachweis der toxischen und infektiösen Acusticusneuritis erbracht.

Befund. Die Krankheitserscheinungen entsprechen dem Symptomenkomplex der serösen Labyrinthitis oder der circumscripten Labyrintheiterung, nur ausnahmsweise der diffusen Labyrintheiterung.

Verlauf, Prognose. Der Verlauf der Ohrerkrankungen beim Mumps ist ungemein verschlieden. In einer beträchtlichen Anzahl der Fälle kann vollkommene Heilung mit Wiederherstellung der physiologischen Funktion des Labyrinths eintreten, auch wenn auf dem Höhepunkt der Entzündung das Hörvermögen hochgradig herabgesetzt oder erloschen ist (Urbantschitsch, Fournier u. a.). Es handelt sich hierbei um diejenigen Fälle, bei welchen die Mumpslabyrinthitis unter dem Bilde der Labyrinthitis serosa auftritt und verläuft. Das statische Labyrinth erhält in manchen Fällen seine Reflexerregbarkeit vollkommen wieder, ja selbst in Fällen, in welchen die Entzündung auf das gesamte Labyrinth ausgedehnt war und infolge von circumscripter, auf die Schnecke beschränkter Eiterung bleibende Ertaubung eingetreten ist, kann Heilung mit vollkommener funktioneller Wiederherstellung des statischen Labyrinths beobachtet werden.

So teilte auch Mauthner aus meiner Abteilung einen Fall mit, in welchem die Anamnese erkennen liess, dass die (in diesem Falle beiderseitige) Mumpslabyrinthitis ursprünglich das akustische und das statische Labyrinth betroffen hatte (es hatten Taubheit, Schwindelanfälle, Gleichgewichtsstörungen und Erbrechen bestanden). Die Patientin gelangte ein Jahr nach Ablauf des Mumps zur Untersuchung ; hierbei zeigte sich rechtsseitige Taubheit, dagegen vollkommen normale Reflexerregbarkeit des rechten statischen Labyrinthes.

Manche Fälle von Mumpslabyrinthitis heilen mit bleibender Schwerhörigkeit aus. Es ist auch anzunehmen, dass eine verminderte Erregbarkeit des statischen Labyrinths dauernd fortbestehen kann. Die Facialislähmung heilt gewöhnlich vollständig aus.

Behandlung. Die Behandlung der Mumpslabyrinthitis ist eine rein symptomatische ; sie besteht in Bettruhe und Applikation eines Eisbeutels auf den Kopf. Bei heftigen Schwindelanfällen empfiehlt sich Verdunklung des Zimmers und Galvanisation des Acusticus (mit Erzeugung eines dem pathologischen Nystagmus in der Richtung entgegengesetzten galvanischen Nystagmus). In Fällen von anhaltend heftigem Schwindel kommt die innerliche oder subkutane Anwendung narkotischer Mittel in Betracht. Die Bettruhe ist selbst nach Aufhören der Schwindelanfälle bis zum Rudimentärwerden des Nystagmus einzuhalten. Nach Ablauf des Mumps und der Reizerscheinungen von seiten des statischen Labyrinths können Heissluftbehandlung, Schwitzkuren und Galvanisation des Octavus (2–3 mal wöchentlich, Anode am Ohr, Stromstärke 4–6 Milliampères, Dauer jeder Sitzung 5–10 Minuten) durch einige Wochen empfohlen werden. Von der Anwendung von Pilocarpin- oder Fibrolysininjektionen habe ich keinen Erfolg gesehen.

Die nicht eitrigen Labyrintherkrankungen bei Typhus.

Die im Verlaufe des Typhus vorkommenden Labyrintherkrankungen entsprechen in einer erheblichen Anzahl der Fälle einer eitrigen Labyrinthentzündung, doch kommen auch Fälle von Neuritis nervi octavi oder der Labyrinthitis serosa vor. Nach den Untersuchungen von Manasse und

Sporleder ist die Typhustaubheit die Folge eines innerhalb des Labyrinths oder am Hörnerven verlaufenden entzündlichen Prozesses. In einem Falle von Typhustaubheit hat Manasse chronisch entzündliche Veränderungen am häutigen Labyrinth (Bindegewebsneubildung, degenerative Atrophie des Neuroepithels und des Nervenganglienapparates) und in der knöchernen Labyrinthkapsel (pathologische Knochenherde) nachgewiesen.

Die *Symptome* bestehen in labyrinthärer Schwerhörigkeit, subjektiven Geräuschen, in Anfällen von Labyrinthschwindel und Gleichgewichtsstörungen. Der nervöse Charakter der subjektiven Geräusche lässt sich in diesen Fällen mit Hilfe des galvanischen Stromes genau feststellen. Appliziert man die Anode am Ohre (Tragus oder Warzenfortsatz), die Kathode an einem indifferenten Punkte, so tritt nach Stromschluss auf Seite der Anode eine bedeutende Abschwächung des subjektiven Geräusches ein oder dasselbe wird für die Zeit der Stromdurchleitung gänzlich aufgehoben. Befindet sich die Kathode am Ohr (nach Umkehrung des Stromes), so ergibt sich nach Stromschluss eine Verstärkung des subjektiven Geräusches. Die Schwindelanfälle und die Gleichgewichtsstörungen zeigen in der Mehrzahl der Fälle nur eine geringe Intensität (allgemeines, mehr kontinuierliches Schwindelgefühl ohne deutlichen Charakter des Drehschwindels, Gefühl der Unsicherheit).

Prognose. Die im Verlaufe des Typhus auftretenden, nicht eitrigen Labyrintherkrankungen können mit vollkommener Wiederherstellung der Labyrinthfunktion zur Heilung kommen, doch werden auch Fälle von bleibender Schwerhörigkeit oder gänzlicher Taubheit beobachtet.

Behandlung. In Fällen von Labyrintherkrankung können Ohrensausen und Schwindelgefühl durch *Galvanisation* vermindert oder wenigstens vorübergehend beseitigt werden. Von gutem Einfluss ist die richtige Seitenlagerung des Patienten (entgegengesetzt der Richtung des Spontannystagmus). Besteht Brechneigung, so ist grösste Vorsicht bei der Verabreichung der Nahrung angezeigt, man gebe nur kühle und flüssige Kost in geringen Einzelmengen.

Von einer Lokalbehandlung der typhösen Labyrinth- und Octavuserkrankungen muss abgesehen werden.

Die nicht eitrigen Labyrintherkrankungen im Verlaufe der Influenza.

Vorkommen. Mitunter entwickeln sich im Verlaufe der Influenza infektiöse Neuritiden des Octavus (oft verbunden mit Neuritiden des Trigeminus, Abducens oder Facialis). Fälle von Ertaubung und hochgradigen nervösen Hörstörungen nach Influenza (Neurolabyrinthitis) sind von Biehl, Hegener, Lannois und Wittmaack mitgeteilt worden.

Die Erscheinungen bestehen im typischen Symptomenkomplex der Erkrankung des inneren Ohres (Einschränkung der oberen Tongrenze, Schwerhörigkeit, Spontannystagmus, Drehschwindel, Gleichgewichtsstörungen ; in hochgradigen Fällen vollständige Taubheit).

Bezüglich der Behandlung wäre nur das im Kapitel der nicht eitrigen Labyrintherkrankungen bei Typhus Gesagte zu wiederholen.

In seltenen Fällen kann sich im Verlaufe eines *Erysipels* eine nicht

eitrige Labyrintherkrankung vom Typus der akuten Labyrinthitis serosa entwickeln. Die Heilung hier zumeist mit Wiederherstellung normaler Hörschärfe oder mit geringer Herabsetzung derselben.

Im Verlaufe des *akuten Gelenkrheumatismus* können auf der Höhe der Erkrankung leichte oder mittelgradige Hörstörungen auftreten. Die Untersuchung ergibt in diesen Fällen den Bestand einer Neurolabyrinthitis. Schwerere Formen der Neurolabyrinthitis mit Ausgang in bleibende Schwerhörigkeit oder Taubheit entwickeln sich im Verlaufe des akuten Gelenkrheumatismus nur ausnahmsweise. Infolge der Verwendung von Salizylpräparaten können bei akuten Gelenkrheumatismus auch toxische Labyrinth- oder Octavusaffektionen auftreten. Nach den Untersuchungen von Wittmaack und Blau findet die Salizylsäure wie das Chinin und Antipyrin seinen Angriffspunkt im peripheren Acusticusganglion. Die im Verlaufe des akuten Gelenkrheumatismus auftretende Mittelohrentzündung unterscheidet sich nicht von den genuinen Formen dieser Erkrankung.

2. *Bei den chronischen Infektionskrankheiten.* — Experimentell hat Wittmaack (*Zeitschr. f. Ohrenheilk.*, Bd. li) degenerative Neuritis des Octavus durch Infektion mit Tuberkulose an Tieren erzeugt.

Im übrigen handelt es sich bei den nicht eitrigen Erkrankungen des inneren Gehörorgans bei *Tuberkulose* zumeist um sekundäre degenerative Atrophie des Nervenganglienapparates und der labyrinthären Nervenendstellen infolge von chronisch entzündlichen tuberkulösen Mittelohrveränderungen. Die grösste Mehrzahl der bei Tuberkulose beobachteten Erkrankungen des inneren Ohres betreffen eitrige Erkrankungen des Ohrlabyrinths.

Die Erkrankungen des inneren Gehörorgans bei *Syphilis* sind von diesem Referate ausgenommen, da sie in einem besonderen, die Salvarsanbehandlung der syphilitischen Ohrenkrankheiten behandelnden Vortrag besprochen werden.

(*b*) Die sekundären nicht eitrigen Labyrintherkrankungen (I–III)

Sekundäre nicht eitrige Labyrinthaffektionen bei akuter Mittelohreiterung sind selten. Zezold (*Lehrbuch d. Ohrenheilk.*, 2. Aufl., München, 1909) hat darauf aufmerksam gemacht, dass Einengung der oberen Tongrenze in Fällen von akuter Otitis auf eine Miterkrankung des inneren Gehörorgans schliessen und eine bleibende Herabsetzung der Hörschärfe nach Heilung der Otitis befürchten lässt. Die hiehergehörigen Fälle folgen dem Typus der serösen Labyrinthitis, doch scheint es, dass sich geringe Veränderungen in den Schneckenskalen, die später zu Bindegewebsbrückenbildungen oder zur wirklichen Fibrombildung in den Skalen führen, symptomlos einstellen können. In diesen Fällen kommt es sodann zur degenerativen Atrophie des Cortischen Organs der Schneckenbasis.

In Fällen von *chronischer* Mittelohreiterung sind sekundäre degenerative Veränderungen im inneren Ohre ungemein häufig. Sie sind in der

Mehrzahl der Fälle auf den cochlearen Teil des Labyrinths beschränkt und entwickeln sich schleichend unter allmählicher Abnahme der schon durch die Mittelohrerkrankung geschädigten Hörschärfe. Den Ausgangspunkt für die Erkrankung scheint in vielen Fällen die Fensterregion zu bilden, ohne dass an einen kontinuierlichen oder metastatischen Uebergang des eitrigen Mittelohrprozesses als sochen in das innere Ohr gedacht werden könnte. Der Mechanismus der Veränderungen selbst ist nicht genau bekannt. Ein ziemlich häufiger Typus ist der, dass ausgehend von pathologischen Bindegewebsmassen in der Nische des Schneckenfensters sich Bindegewebsnetze in der Scala tympani entwickeln. Dieselben führen zu einer Bewegungseinschränkung der basilaren Membran ; damit ist die Schnecke in dem befallenen Teil der basilaren Membran nicht mehr funktionstüchtig und das Cortische Organ atrophiert nach dem Typus der Inaktivitätsatrophie. Gleichzeitig atrophieren die entsprechenden Teile des Nervus cochlearis und des Ganglion spirale. In seltenen Fällen scheint es sich hier um primäre Neuritiden zu handeln.

Diese rein degenerativen, nicht eitrigen, sekundären Labyrinthveränderungen haben selbstverständlich mit den eitrigen Sekundärerkrankungen des Labyrinths nichts zu tun. Im allgemeinen ist die sekundäre Labyrinthdegeneration bei chronischer Mittelohreiterung progredient und kann bei Persistenz der Mittelohreiterung mit völliger Taubheit enden. Die zelligen Metaplasien des Cortischen Organs, die dabei beobachtet werden, entsprechen ganz denjenigen Befunden, die ich (*Arch. f. Ohrenheilk.*, Bd. lvi, 1903) bei der durch Arteriosklerose verursachten, labyrinthären Schwerhörigkeit festgestellt habe.[1] Vom prophylaktischen Standpunkt ist hervorzuheben, dass, je weniger umfangreich die Degeneration ist, desto eher die Aussicht gegeben ist, dass durch Heilung der Mittelohreiterung der degenerative Prozess im inneren Ohr zum Stillstand kommt. Eine besondere Art der labyrinthären degenerativen Erkrankung bildet die postoperative Labyrinthdegeneration, über die aus meiner Abteilung C. M. L. Brown (*Archiv f. Ohrenheilk.*, Bd. lxxx) berichtet hat.

Die Veränderungen im Ohrlabyrinth bei Otosklerose. — Die charakteristischen anatomischen Befunde bei Otosklerose, die Ostitis vasculosa, weisen darauf hin, dass es sich um eine Erkrankung des schallleitenden Apparates handelt. Die pathologische Verwandtschaft der Otosklerose mit den Affektionen des inneren Ohres ist erst durch neuere Befunde erwiesen worden. Dahin gehört erstlich der Befund otosklerotischer Knochenherde in einem Falle von Politzer (*Anatomie der Taubstummheit*, 1. Lief., Bergmann, 1904) und in einem von Alexander beschriebenen Falle von kongenitaler Taubheit (*Anatomie der Taubstummheit*, 2. Lieferung, 1905), sodann der Nachweis des vergesellschafteten Vorkommens otosklerotischer Knochenveränderungen in der Labyrinthkapsel verbunden mit degenerativer Atrophie des Nervus cochlearis und des Corti'schen Organs bei endemischem Kretinismus, endlich zeigt auch die verfeinerte

[1] Nach mir hat Manasse die gleichen Befunde erhoben (P. Manasse, *Ueber chronische, progressive, labyrinthäre Taubheit*, Wiesbaden, Bergmann, 1906).

Funktionsprüfungsmethode in klinischen Fällen von Otosklerose die überraschende Häufigkeit von Veränderungen des inneren Gehörorgans. Derartige Symptome sind in Fällen von vorgeschrittener Otosklerose nicht überraschend. Es zeigt sich jedoch, dass auch bei Frühformen der Otosklerose ein labyrinthärer Symptomenkomplex auftreten kann, ja in manchen Formen der Otosklerose sind die labyrinthären Veränderungen primär vorhanden. Damit geht die klinische Form der Otosklerose über in die Gruppe der hereditär degenerativen Ohrerkrankungen. Ich denke, es ist vorteilhafter, in dem Komplex dieser letzteren Erkrankungen vom *Otosklerosetypus* zu sprechen ; er ist mitunter gekennzeichnet durch den charakteristischen Trommelfellbefund (normales Trommelfell mit rotem Promontorialschimmer), durch die charakteristischen klinischen Merkmale (lästiges Ohrensausen, progrediente Abnahme der Hörschärfe, Parakusis Willieii). Die Funktionsprüfung ergibt jedoch in solchen Fällen neben Symptomen von Seite des schallleitenden auch solche von Seite des schallperzipierenden Apparates.

Für die bei Otosklerose beobachteten Labyrintherkrankungen ist es allerdings fraglich, ob in allen Fällen die Veränderungen am häutigen Labyrinth und am Hirnnerv stets als sekundäre Veränderungen aufzufassen sind. Es ist zuzugeben, dass bei der grossen Mehrzahl der Fälle von Otosklerose, die Veränderungen am häutigen Labyrinth und im Hörnerv sich im späteren Verlaufe der Otosklerose als rein sekundäre Atrophie zeigen.

Auf Grund von genaueren Befunden der letzten Zeit ist jedoch anzunehmen, dass in einzelnen Fällen die Erkrankung des häutigen Labyrinths und des Nervus octavus gleichzeitig mit der Erkrankung der Labyrinthkapsel auftritt oder ihr sogar vorausgeht, sei es, dass zu dieser Zeit die knöcherne Labyrinthkapsel noch vollständig normal ist oder ihre Erkrankung noch nicht manifest geworden ist.

DER GEGENWÄRTIGE STAND UNSERER KENNTNISSE VON DEN NICHT EITRIGEN LABYRINTHERKRANKUNGEN

Resümee

I. *Anatomie*

Die in Fällen von nicht eitrigen Erkrankungen des inneren Ohres festgestellten anatomischen Veränderungen lassen sich in drei Gruppen bringen :

1. Blutungen einschliesslich neurolabyrinthärer Hyperämie oder Anämie.

2. Exsudative, nicht eitrige Prozesse.

3. Degenerative Prozesse : (*a*) primäre, (*b*) sekundäre.

(*a*) *Ausdehnung der anatomischen Veränderung.* — Nervus octavus und Labyrinth bieten den pathologischen Schädigungen gegenüber im allge-

meinen ein Ganzes dar. Erkrankungen, die primär den Nerven und den Ganglienapparat befallen, führen sehr bald zu konsekutiven Veränderungen des Neuroepithels (Alexander, Manasse, Wittmaack). Primär degenerative Veränderungen in den letzteren können ausnahmsweise längere Zeit den Nervenganglienapparat mehr weniger unbeeinflusst lassen (Alexander, *Arch. f. Ohrenh.*, Bd. lxxiv). In der grossen Mehrzahl der Fälle ist der Untergang von Sinneszellen sehr bald von regionärer Zerstörung des Nervenganglienapparates gefolgt. Den Typus der im Nervenganglienapparat einsetzenden, nicht eitrigen Labyrintherkrankung stellen die infektiösen Neuritiden infolge von toxischer Schädigung, sowie die im Verlaufe von akuten und chronischen Infektionskrankheiten beobachteten, nicht eitrigen Erkrankungen des inneren Ohres dar. Typisch peripheren Beginn in den Neuroepithelien zeigen manche akute, traumatische Verletzungen. Die arteriosklerotischen Erkrankungen des inneren Ohres stellen sich zum Teil als primäre Erkrankungen des Octavus dar, zum Teil erkrankt Labyrinth und Nerv gleichzeitig. Bei jeder länger dauernden Labyrintherkrankung sind pathologische Veränderungen im Nervus octavus zu gewärtigen.

Der Cochlearis und der Vestibularis zeigen gegenüber derselben Schädlichkeit nicht die gleiche Resistenz. Der Nervus vestibularis ist bei weitem widerstandsfähiger als der Nervus cochlearis. Auf den cochlearen oder vestibularen Teil des Labyrinths beschränkte Erkrankungen lassen den primären Sitz der Schädigung im Nerven erwarten ; besonders bei den akuten, nicht eitrigen Erkrankungen des Labyrinths selbst ist die Erkrankung zumeist auf das gesamte Labyrinth ausgedehnt (Wittmaack). Als Ursache der grösseren Widerstandsfähigkeit des Nervus vestibularis und des Vestibularapparates wird sein bedeutenderes phylogenetisches Alter und die gegenüber dem Cochlearapparat dickeren Nervenfasern, grösseren Ganglien- und Sinneszellen herangezogen. Von den Typen der auf den Cochlearis beschränkten neurolabyrinthären Erkrankungen hebe ich die arteriosklerotischen, infektiösen und die sekundären Labyrintherkrankungen im Verlaufe von chronischer Mittelohreiterung hervor.

Auffallend ist, dass die Wurzeln des Octavus jenseits von seinem Eintritt in den Hirnstamm und die Kerne des Octavus infolge von peripheren Labyrinth- oder Octavuserkrankungen nur geringgradige und erst nach längerer Zeit auftretende pathologische Veränderungen erleiden. Besonders degenerative Prozesse im peripheren Gehörorgan können ohne jede Veränderung im Kern des Octavus ablaufen, und selbst für die Fälle von Zerstörung des gesamten Labyrinthes durch Labyrinthblutung sind nicht immer Degenerationserscheinungen in der Octavusregion des Hirnstammes festzustellen. In Fällen, in welchen bei peripheren Labyrinthblutungen Veränderungen in dem Octavuskern gefunden worden sind, könnten diese zum Teile auch auf gleichzeitig erfolgten Blutungen im Hirnstamme selbst ätiologisch zurückgeführt werden.

Sicher ist, dass das periphere Gehörorgan und der VIII. Hirnnerv mit den Kernen des VIII. Hirnnerven nur in einem lockeren, nutritiven

Zusammenhang stehen, es sei denn, dass wir nach den uns bisher zur Verfügung stehenden Färbungs- und Imprägnationsmethoden nicht imstande sind, feinste Zellenveränderungen in den Strängen und Zellen der Wurzeln festzustellen.

Wittmaack (*Verhandl. d. d. otol. Ges.*, 1911) fand nach endokranieller Quetschung des Acusticus an der Katze eine absteigende Degeneration des Cochlearis und Atrophie des Cortischen Organs. Auch in diesen Folgeerscheinungen der endokraniellen Quetschung wird der Vestibularis resistenter gefunden als der Cochlearis.

Beiläufig sei bemerkt, dass auch in Fällen von kongenitaler Taubheit die Kerne des VIII. Hirnnerven trotz vollkommener Funktionsunfähigkeit des Labyrinthes und hochgradiger Atrophie des VIII. Hirnnerven nach unserer Färbungsmethode intakt und normal gefunden werden.

Einzelne Gruppen der Labyrintherkrankungen sind endlich auch mit histologischen Veränderungen in der perilymphatischen und knöchernen Labyrinthkapsel verbunden. Es gilt dies vornehmlich von den sekundären Labyrintherkrankungen, in welchen sich die Erkrankung am häutigen Labyrinth oder am Hörnerv bereits auf der Grundlage periostitischer Veränderungen der knöchernen Labyrinthkapsel mit Veränderungen des Endost der peripheren Räume entwickelt, und es gilt dies auch für die Otosclerose, die mit ostitischen Veränderungen der knöchernen Labyrinthkapsel verbunden ist.

Frühzeitige Veränderungen der Stria vascularis finden sich besonders in den Fällen von arteriosklerotischer Labyrintherkrankung. In einzelnen Fällen ist die Annahme gerechtfertigt, dass die primären Veränderungen in der Stria vascularis sich entwickeln.

Als eine besondere Form der nicht eitrigen Labyrintherkrankung ist die Labyrinthitis ossificans Alexander anzusehen, die in einzelnen Fällen den anatomischen Endausgang von infektiöser Neurolabyrinthitis darstellt.

Eine Anzahl von Befunden der älteren Literatur sind endlich durch Kontrolluntersuchungen (Alexander, Brühl, Nager, Wittmaack und Laurowitsch) an ideal konservierten, sowie an mazerierten Gehörorganen von Menschen und durch experimentelle Untersuchungen an Tieren als Artefakte erkannt worden. Dahin gehören eine ganze Anzahl von Veränderungen im Octavus. Der Befund disseminierter, anscheinend nekrotisch zerfallener Stellen, die sogenannte Lückendegeneration durch Schrumpfung des Nerven und des Ganglion können pathologische Veränderungen vorgetäuscht werden. Abhebung des Epithels am Labyrinth von seiner Unterlage, Verbiegung an der basilaren Membran oder an den Sinneszellen, Abhebung des Skalenendost, Lageveränderungen der Reissner'schen Membran (Depression der Reissner'schen Membran), abnorme Lage der Cortischen Membran u.s.f. sind als Artefakte erkannt worden, die zum Teil als Kadavererscheinung anzusehen sind, zum Teil auf die histologischen Methoden, die histologische Behandlung des Präparates (Fixation, Entkalkung) zurückgeführt werden. Auch das Ganglion spirale und die Stria vascularis unterliegt frühzeitig kadaverösen Veränderungen. Nager hielt es für

möglich, dass auch die Corpora amylacea im Nervus octavus ein Kunstprodukt darstellen, dagegen ergab die Untersuchung von Alexander und Obersteiner, dass dies nicht der Fall ist. Die Corpora amylacea deuten zwar nicht in allen Fällen auf krankhafte Veränderungen des Nervus octavus, dürfen jedoch nie als Kunstprodukt betrachtet werden.

Auch der histologische Befund der Labyrinthblutung muss mit einiger Vorsicht gewertet werden, da zirkumskripte Labyrinthblutungen auch als agonale Erscheinung auftreten können.

Nach Nager und Yoshii (*Zeitschr. f. Ohrenheilk.*, Bd. lx) lassen sich kadaveröse Veränderungen im inneren Ohr schon eine Stunde nach dem Tode feststellen.

Nager (*Zeitschr. f. Ohrenheilk.*, Bd. li, p. 250) berichtet über postmortale, künstlich erzeugte Degenerationsherde im Hörnerven.

Bei den *Funktionsprüfungsmethoden* ist ein bedeutender Fortschritt zu verzeichnen. Die Untersuchungsmethoden des Cochlear-Apparates sind durch Hegener, Bezold, Schäfer, Wanner u. a. mächtig gefördert worden. Die grössten Fortschritte sind auf dem Gebiete der Funktionsprüfungsmethoden des statischen Labyrinths zu konstatieren. Die Exaktheit dieser Prüfungsmethoden ist mustergiltig für sämtliche labyrinthären Untersuchungsarten. Vor allem kommt in Betracht, dass bei der klinischen Prüfung des statischen Labyrinths die funktionellen Veränderungen aus dem Ausfalle bestimmter Reflexe erschlossen werden können. Diese Reflexe bestehend im Nystagmus, Schwindel, reflektorischen Gleichgewichtsstörungen und labyrinthärer Gleichgewichtsverminderung sind wohl studiert und gestatten eine genaue klinische Beobachtung. Die genannten Reaktionen sind vom Willen, der Aufmerksamkeit und dem Konzentrationsvermögen des Patienten völlig unabhängig ; bei der Funktionsprüfung des statischen Labyrinths laufen wir nicht wie bei der cochlearen Prüfung Gefahr, das Prüfungsresultat durch Unaufmerksamkeit des Patienten, Verwirrtheit des Untersuchten oder endlich durch den Versuch der Simulation entwertet zu sehen. Für den Nystagmus ist weder Aggravation, noch Simulation seitens des Untersuchten möglich. Selbst bei Trübung oder Verlust des Bewusstseins, und in der Narkose, somit in einem Allgemeinzustand des Untersuchten, in welchem von einer Prüfung der akustischen Fähigkeiten keine Rede mehr sein kann, können wir den funktionellen Zustand des Vestibularis noch ermitteln. Zur richtigen klinischen Wertung der labyrinthären Gleichgewichtserscheinungen bedarf es allerdings grosser Erfahrung seitens des Untersuchers.

Es gilt, die Vestibularisprüfung derart konzis zu gestalten, dass sie ohne Unannehmlichkeiten für den Untersuchten und ohne bedeutenden Schwindel durchgeführt werden kann.

Wir müssen darauf hinarbeiten, schon geringe Formen und Grade des bei der Funktionsprüfung ausgelösten labyrinthären Nystagmus diagnostisch verwerten zu können. So ist es bei der kalorischen Prüfung sehr zu empfehlen, den Kaltwasser-Nystagmus sofort nach seinem Auftreten

durch Nachspülen warmen Wassers zu paralysieren (Bárány u. a.). Mit Untersuchungen, die uns instandsetzen sollen, bei der Prüfung auf dem Drehstuhl auf zehn volle Umdrehungen zu verzichten und mit einer halben bis einer Drehung auszukommen, bin ich derzeit beschäftigt. Das im übrigen vorzügliche Nystagmometer (Buys) hat bisher keine klinische Verwendbarkeit finden können, da das Belichtungsresultat (Nystagmograph) durch nicht vermeidbare willkürliche Kontraktionen der Lidmuskeln sehr beeinträchtigt wird.

Leider ist es bisher nicht gelungen, die physiologisch auslösbaren akustischen Reflexe der klinischen Funktionsprüfung dienstbar zu machen. Nur der Tensorreflex gibt uns einige Aussicht, dass es in Zukunft mit Hilfe verfeinerter Beleuchtungsapparate gelingen könnte, aus der Wahrnehmung der der Tensorkontraktion folgenden Stellungsänderung des Trommelfelles klinisch verwertbare Schlüsse zu ziehen.

Aus den bedeutenden Fortschritten in der Erkenntnis der Aetiologie und Pathologie der nicht eitrigen Erkrankungen des Ohrlabyrinths folgt selbstverständlich eine Verfeinerung der *Diagnostik*.

Exakte Wege in der Differentialdiagnose der nicht eitrigen Acusticus- und Labyrinthkrankheiten hat Wittmaack (*Zeitschr. f. Ohrenheilk.*, Bd. l, p. 127, und *Med. Klinik*, Bd. i, 1905) angebahnt.

In diagnostischer Beziehung ist besonders auf das Kapitel der Otosklerose zu verweisen. War es ursprünglich nötig, die Otosklerose als besondere Form einer Schallleitungserkrankung von den übrigen Erkrankungen des schalleitenden Apparates zu trennen, so zeigen sich jetzt immer mehr Beziehungen der Otosklerose zu den kongenitalen und erworbenen Labyrinthanomalien.

Ein wichtiger Fortschritt in der Diagnostik der Erkrankungen des inneren Gehörorgans zeigt sich in dem notwendigerweise erfolgten Aufgeben der Ménière'schen Nomenklatur. Es ist am Platze, kurz über den klassischen Fall Ménière's (*Gazette médicale de Paris*, 1861) zu berichten.

In diesem wiederholt ausführlich zitierten Falle handelt es sich um ein junges Mädchen, bei welchem infolge von Erkältung (Reise in einer Winternacht während der Menstruation) plötzlich häufige Schwindelanfälle, Erbrechen, totale Taubheit entstanden waren. Am fünften Tage starb die Patientin und die Sektion ergab: Das Zentralnervensystem intakt und lediglich eine rote plastische Masse (etwa hämorrhagisches Exsudat) im Vorhofe und in den Bogengängen. Eine Todesursache konnte nicht festgestellt werden. Berücksichtigen wir die neueren Erfahrungen über Erkrankungen des Gehörorgans bei Leukämie, bezw. Lymphomatosen, so lässt sich mit grosser Wahrscheinlichkeit annehmen, dass es sich in diesem ersten Ménière'schen Falle keineswegs um eine Erkrankung *sui generis* gehandelt hat, sondern um eine Erkrankung des Vestibularapparates bei Leukämie unter apoplektischem Einsetzen der Symptome. Der Bestand der Leukämie wurde eben *intra vitam* nicht erkannt und entging auch der Feststellung bei der Sektion.

Ménière hat sich durch die Mitteilung dieses Falles um die Klinik

der Erkrankung des inneren Gehörorgans die grössten Verdienste erworben. Die klinischen Erscheinungen des Falles bestanden in plötzlicher Ertaubung verbunden mit Drehschwindel, Gleichgewichtsstörungen, Nausea, Konvulsionen und Erbrechen. Durch die Autopsie des Falles wurde nun für den Menschen zum erstenmal der Nachweis erbracht, dass der Komplex dieser Erscheinungen auf eine Erkrankung des Labyrinths zurückgeführt werden müsse. Bis zu dieser Zeit war man trotz der bereits vorliegenden Resultate des Tierexperimentes geneigt, einen Schwindel der oben beschriebenen Art auf eine Erkrankung des Cerebellum, nicht aber auf eine Erkrankung des Labyrinths zurückzuführen. Die Lücke in der Ménière'schen Mitteilung betrifft aber die Aetiologie. Ménière selbst erwähnt, dass die Autopsie des Falles in jeder anderen Richtung negativ war und lediglich die beiderseitige Labyrinthblutung nachgewiesen wurde. Die Frage der eigentlichen Todesursache wird in der Arbeit Ménières nicht diskutiert. Die Meningen waren normal; die Körperorgane normale. Ein mikroskopischer Blutbefund wurde nicht erhoben. Das Knochenmark ist nicht untersucht worden. Einerseits ist ausser Zweifel, dass die Patientin nicht an der Labyrinthblutung gestorben ist, da eine Labyrinthblutung als Ursache für den Exitus letalis nicht gelten kann, andererseits wurde eine anderweitige anatomische Veränderung im Körper nicht aufgefunden. Aus der Arbeit Ménières resultiert die Aufstellung des Begriffes der Ménière'schen Krankheit. Die Defekte dieses klinischen Begriffes waren von vornherein nicht zu ersehen, zeigten sich jedoch sehr bald in einer uferlosen Verwirrung der Untersucher, der erst v. Frankl-Hochwart in seinem Buche über den Ménière'schen Symptomenkomplex zu steuern versuchte. Das von Ménière selbst mitgeteilte Beispiel der Ménière'schen Krankheit ist isoliert geblieben. Man musste schliesslich auf den ganzen Begriff verzichten, fasste nur die Ménière'sche Trias die Symptome als Ménière'schen Symptomenkomplex auf, und fand nun, dass sich dieser Komplex bei verschiedenen Allgemeinerkrankungen und Erkrankungen des Gehörorgans findet. Auch die Nomenklatur 'Ménière'scher Symptomenkomplex' war nach mehr als einer Richtung eine gezwungene; vor allem sind die Symptome der Ménière'schen Trias: Taubheit, Schwindel, Erbrechen, einander nicht koordiniert, da das Erbrechen nur das Zeichen eines ziemlich heftigen Schwindels und eine Folgeerscheinung des Schwindels ist. Weiterhin ist es sicher, dass Taubheit und heftigster Schwindel nur bei einer hochgradigen Erkrankung des inneren Gehörorgans vorkommen, während geringgradige Erkrankungsformen dieses Ohrbezirkes sich nur in Schwerhörigkeit und mässigem Schwindel äussern. Endlich ist es klar, dass bei zirkumskripten Erkrankungen des Labyrinths einmal nur die Schnecke, das andere Mal nur das statische Organ erkrankt sein kann. Man wird im ersten Falle als Symptom nur die Hörstörung, im zweiten Falle nur den Schwindel zu erheben haben. Der Nachhall der Ménière'schen Mitteilung war jedoch ein so mächtiger, dass man sich nicht vollständig von der Ménière'schen Nomenklatur befreien konnte. Man hielt an dem

Ménière'schen Symptomenkomplex fest und sprach dann von einem inkompletten Ménière, unterschied den Ménière'schen Symptomenkomplex mit und ohne anderweitige Erkrankung des inneren Ohres, so dass ein Ménière'scher Symptomenkomplex ohne Schwindel, ein Ménière'scher Schwindel ohne Hörstörung entstand; endlich sprach man sogar von einem Morbus Ménière sine Ménière. In meiner Habilitationsschrift habe ich als erster den Vorschlag gemacht, die Ménière'sche Nomenklatur durch die exakte Diagnostik von Labyrinthkrankheiten zu gruppieren und durch die Krankheiten des akustischen und statischen Labyrinths zu ersetzen. In der Folgezeit haben es die bedeutenden Fortschritte in der Untersuchung und Diagnostik des statischen Labyrinths mit sich gebracht, dass ohne irgend einen diesbezüglich gefassten Beschluss die Ménière'sche Nomenklatur vollständig fallen gelassen wurde. Ich schlage vor, nur für den Symptomenkomplex, der durch paroxysmales Auftreten dem klassischen Ménière'schen Falle entspricht, angesichts des hohen Verdienstes Ménières um die Klinik der Innerohrerkrankung, die Nomenklatur 'Ménière'sche Krankheit' zu belassen.

In der Deutung des Ménière'schen Falles selbst stehe ich heute auf demselben Standpunkt, den ich 1903 in meiner Habilitationsschrift und neuerlich 1906 in meiner Arbeit über die lymphomatösen Ohrerkrankungen vertreten habe. Der klassische Fall Ménières von *ad exitum* gelangter, unter Ertaubung und Schwindel eingetretener Labyrinthblutung, mit im übrigen vollständig negativem Sektionsbefunde, war nichts anderes als eine Labyrinthblutung bei perakuter myelogener Leukämie. Wir wissen heute, dass einzelne Formen der Leukämie ohne Veränderungen in den Lymphdrüsen und der Milz verlaufen und sich lediglich auf makroskopische Veränderungen des Knochenmarkes und Veränderungen in der Zusammensetzung des Blutes beschränken können. Das Knochenmark wurde bei der Autopsie des Ménière'schen Falles nicht untersucht, der mikroskopische Blutbefund nicht aufgenommen.

Politzer (*Lehrbuch der Ohrenheilk.*, 5. Aufl)., F. Alexander und Manasse (*Zeitschr. f. Ohrenheilk.*, Bd. lv) wollen mit Ménière'scher Krankheit diejenigen Formen der inneren Ohrerkrankung bezeichnen, bei denen Schwindel, Erbrechen und Schwerhörigkeit apoplektiform erscheinen. Dagegen legen sie keinen Wert darauf, ob das betreffende Gehörorgan vorher gesund oder schon erkrankt gewesen war. Sie glauben sogar, dass meist letzteres der Fall ist. Es handelt sich dann um den Typus der chronischen, progressiven, labyrinthären Schwerhörigkeit mit Blutungen in den Hörnerv. F. Alexander und Manasse untersuchten klinisch und anatomisch einen Kranken, der an chronischer, progressiver, labyrinthärer Schwerhörigkeit gelitten hatte, plötzlich einen Ménière'schen Anfall bekam und einige Tage später an Gehirnapoplexie starb. Bei der Sektion wurden neben den Zeichen der chronischen, labyrinthären Schwerhörigkeit Labyrinthblutungen gefunden, und zwar im Ramus cochlearis und vestibularis und in sämtlichen Nervenverzweigungen bis kurz vor der Nervenendigung, dagegen niemals in den Labyrinthhohlräumen.

Prophylaxe und Therapie. — Auf dem Gebiete der Prophylaxe der

nicht eitrigen Labyrintherkrankungen sind eine Reihe von Erfolge zu verzeichnen. In prophylaktischer Beziehung sind besonders in den Fällen von chronisch traumatischer, labyrinthärer Schwerhörigkeit (professionelle Schwerhörigkeit, Berufskrankheiten des Labyrinths) Erfolge erreicht. In vielen Staaten sind durch Ohrenärzte Massnahmen zur Bekämpfung der professionellen Schwerhörigkeit ins Leben gerufen worden. Die Erkenntnis, dass manche in der internen Medizin verwendeten Mittel zu einer Schädigung des Nervenganglienapparates des Octavus Anlass geben können, führte dazu, diese Mittel aus der Therapie zum Teil oder gänzlich auszuschliessen. Endlich sei an die Erfolge der Thyreoidinbehandlung der endemischen konstitutionellen Schwerhörigkeit erinnert, sowie an die Erfolge der Prophylaxe bei den nicht eitrigen Neurolabyrinthitiden im Verlaufe von akuten Infektionskrankheiten.

Die Fortschritte in therapeutischer Beziehung sind in den Spezialartikeln gewürdigt worden. Besonders durch das Erkennen der Aetiologie haben wir für manche Affektionen des inneren Gehörorganes (arteriosklerotische Erkrankungen, endemische Schwerhörigkeit u.s.f.) eine in therapeutischer Bedeutung fruchtbare Basis gewonnen. Den Vorschlägen der chirurgischen Behandlung mancher, bei nicht eitriger Labyrintherkrankung auftretender Symptome kann ich mich nicht anschliessen. Die operative Zerstörung der Schnecke oder Durchschneidung des Acusticus in Fällen von subjektiven Ohrgeräuschen sind in Anbetracht der relativen Gefährlichkeit des Eingriffes der langwierigen Nachbehandlung und der Unsicherheit im Resultate (auch nach totaler Taubheit können subjektive Geräusche bestehen bleiben) nicht zu empfehlen. Ebenso wenig vermag ich zur Heilung von Labyrinthschwindel der Extraktion des Steigbügels (Crokett, *Transactions of the American Otol. Soc.*, Bd. xli, p. 116, 1908) oder gar der operativen Eröffnung des Vorhofs oder der Bogengänge das Wort zu reden. Ich hebe dabei hervor, dass es sich in den Fällen von protrahiertem heftigen Schwindel nie um einen reinen labyrinthären Schwindel handeln kann, da selbst nach bedeutenden Verletzungen des Labyrinths der Schwindelanfall an Intensität rasch abnimmt. Gerade in den Fällen, in welchen der Patient durch die Heftigkeit seiner Schwindelanfälle desolat wird, handelt es sich um Kombinationsformen von labyrinthärem und neurasthenischem oder neurotischem Schwindel. In solchen wird eine Erleichterung und schliessliches Schwinden der Schwindelanfälle auch durch die Behandlung der Neurasthenie bezw. der Neurose erzielt; die Schwindelanfälle verlieren ihren heftigen Charakter und schwinden endlich gänzlich. Ich möchte es nur der Neurasthenie dieser Patienten anrechnen, wenn sie sich offenbar in Unkenntnis der Schwere des Eingriffes zu einer Bogengang- oder Vestibularoperation behufs Heilung der Schwindelanfälle entschliessen. Eine Reihe hierher gehöriger Fälle habe ich mitgeteilt, darunter befand sich einer, der schon von anderer Seite zur Labyrinthoperation bestimmt war. Der Patient ist ohne Operation durch eine leichte Wasserkur und roborierende Diät, sowie durch Beobachtung geistiger und körperlicher Ruhe, von seinen Schwindelanfällen vollständig geheilt worden.

SECTION XVI

OTOLOGY

DISCUSSION No. 4

DIE ANATOMIE UND KLINIK DER NICHT EITRIGEN LABYRINTHERKRANKUNGEN

Referat von Professor Dr. K. VON EICKEN, Giessen

Ueber die nicht eitrigen Erkrankungen des Labyrinths

Das Verdienst, das Interesse der ärztlichen Welt auf die Erkrankungen des innern Ohres gelenkt zu haben, gebührt zweifellos dem französischen Arzt Ménière. Er beschrieb zum erstenmal ein Krankheitsbild, das sich in apoplektiform auftretendem Schwindel, Erbrechen, Ohrensausen mit nachfolgender mehr oder weniger hochgradiger Hörstörung äusserte. Beobachtungen, die denen Ménières mehr oder weniger glichen, sich unter Umständen auch recht wesentlich von ihnen entfernten, suchte man in der Folgezeit insgesamt unter dem Namen Ménière'sche Krankheit oder Ménière'scher Symptomenkomplex zu vereinigen. So wurde diese Bezeichnung ein Sammelname, unter dem sich alle möglichen der Art und Entstehung nach ausserordentlich verschiedenartigen Erkrankungsformen zusammenfanden — das sehr verdienstvolle Werk v. Frankl Hochwarts liefert uns für diese Behauptung den allerbesten Beweis.

Dem innern Ohr kommt eine doppelte Funktion zu. Es dient einerseits der Aperzeption von Tönen und Tongemischen, andererseits der Orientierung des Körpers im Raum bei Ruhe und Bewegung. Durch zahlreiche klinische und pathologisch-anatomische Beobachtungen, sowie durch ausgedehnte experimentelle Studien, darf man jetzt wohl mit Sicherheit annehmen, dass der Schnecke und dem in ihr enthaltenen Cortischen Organ die Hörfunktion zufällt, während die übrigen Abschnitte statischen Zwecken dienen.

Auf die Untersuchungstechnik der Organgruppen einzugehen ist heute nicht meine Aufgabe. Wir werden uns nur mit ihren Reiz- und Ausfallserscheinungen zu beschäftigen haben, soweit sie nicht durch Einbruch von Eiter in die Labyrinthräume selbst und durch die Schädigung der Syphilis hervorgerufen sind. Auch auf eine Schilderung der Formveränderungen des Labyrinths, welche durch Missbildungen und Ent-

wicklungshemmungen auftreten, möchte ich verzichten, weil sie streng genommen nicht zu den Erkrankungen des Labyrinths gehören. Ebenso soll nicht die Rede sein von den Befunden des innern Ohres, die man bei Taubheit erhoben hat.

Die professionelle Schwerhörigkeit

Habermann war der erste, der als Ursache der Schwerhörigkeit der Kesselschmiede durch mikroskopische Untersuchung eine degenerative Neuritis des Nerv. cochlearis vor allem in der Basalwindung verbunden mit einer hochgradigen Degeneration des Cortischen Organs nachwies. Ueber ähnliche anatomisch untersuchte Fälle berichteten Brühl und Zange. Anderweitige histologische Untersuchungen von Menschen, die durch Lärmwirkung eine nervöse Schwerhörigkeit erworben haben, beispielsweise von Lokomotivführern, Arbeitern aus grossen lärmenden Betrieben, Militärpersonen oder Schützen, deren Ohren durch die Wirkung von Flinten- oder Kanonenschüssen Not gelitten haben, liegen bisher nicht vor, doch werden wir wohl nicht fehlgehen, wenn wir auch bei ihnen Veränderungen im Cortischen Organe annehmen.

Dass es bei Tieren gelingt, an verschiedenen Stellen des Cortischen Organs Degenerationsherde zu erzeugen, die bei Verwendung hoher Töne der Schneckenbasis, bei Verwendung tieferer Töne der Schneckenspitze genähert sind und deren Umfang und Schwere sich offenbar nach der Dauer der schädigenden Wirkung und der Intensität der Töne richtet, sei hier nur beiläufig erwähnt.

Heftig umstritten worden ist die Frage, ob bei den bei Menschen beobachteten Schädigungen die Zuführung des Schalls durch Knochenleitung eine verderblichere Rolle spiele, als diejenige durch Luftleitung. Friedrich, Wittmaack, und Jaehne erkennen der Knochenleitung die Hauptrolle beim Zustandekommen dieser Schädigungen zu, während die Bezold'sche Schule diese Annahme verwirft. Aus der menschlichen Pathologie liegen Beobachtungen von Reinking an zwei Schlossern vor, die beide seit ihrer Jugend eine einseitige Mittelohreiterung hatten. Nur auf der Seite mit gesundem Mittelohr liess sich eine typische professionelle nervöse Schwerhörigkeit nachweisen, während auf der Seite mit krankem Mittelohr das Aperzeptionsvermögen für hohe Töne nicht geschädigt war. Die Versuche, welche ich an Meerschweinchen angestellt habe, denen einseitig der Ambos entfernt wurde, sowie vor allen Dingen die neuerdings von Hössli auf dem Kongress der deutschen otologischen Gesellschaft in Stuttgart publizierten experimentellen Studien, sprechen mit grösster Wahrscheinlichkeit dafür, dass der Knochenleitung eine irgendwie nennenswerte Rolle nicht zukommt. Für die Prophylaxe gegen das Zustandekommen akustischer Schädigungen ergibt sich daraus, dass es keinen Zweck hat, die Arbeiter und Angestellten, z. B. die Kesselschmiede und Lokomotivführer, dadurch vor akustischen Schädigungen zu schützen, dass man sie auf eine Gummiplatte bringt. Ebensowenig Zweck hat auch wohl die militärische Vorschrift, beim Schiessen Watte

im Gehörgang tragen zu lassen, da nur ein fester obturierender Verschluss beider Gehörgänge gegen die Schädigung des Knalls schützen dürfte.

Durch zahlreiche experimentelle Studien (Yoshii und Siebenmann, von Eicken, Marx, Wittmaack, Röhr) ist der Beweis erbracht, dass die aerotympanale Leitung sicher allein imstande ist, das Cortische Organ zu zerstören. Sehr beachtenswert ist auch die Selbstbeobachtung Hegeners, der nach längerem Experimentieren mit sehr hohen Pfeifentönen von ca. 10,000 v. d. eine 36 Stunden dauernde Taubheit für diese Töne sowie ein hohes Sausen bei sich fand, Erscheinungen, die allmählig wieder verschwanden.

Eine gründliche Studie über die Einwirkung berufsmässigen Telephonierens auf den Organismus mit besonderer Berücksichtigung des Gehörorgans verdanken wir Blegwad, der fand, dass bei gesunden Mädchen eine Schädigung nicht eintrat. Nur in verhältnismässig wenigen Fällen und vielleicht nur bei nervös disponierten Individuen bewirkte das berufsmässige Telephonieren Kopfschmerzen und subjektive Störungen, wie Sausen, Druck, Fülle und Eingenommenheit des Ohres, übermässige Reizbarkeit auf akustische Einflüsse. Für möglich hält Blegwad eine organische Schädigung durch starkes Läuten und beim Einschlagen eines Blitzes in die Telephonleitung. Capart jr. hat mehrere Berichte über derartige Schädigungen aus der Literatur gesammelt.

Mit ganz wenigen Worten sei auf eine Affektion hingewiesen, die vielleicht manche von Ihnen auf der Reise hierher befallen hat, ich meine die Seekrankheit. Worauf sie in letzter Linie beruht, und weshalb einzelne Menschen von diesem Uebel völlig verschont bleiben, während andere recht heftig darunter leiden, entzieht sich heute noch völlig unserer Kenntnis. Bárány beobachtete auf einer Seefahrt, bei der der Dampfer Auf- und Abwärtsbewegungen ausführte, dass er die vertikale Schiffsbewegung bei Rückenlage fast gar nicht unangenehm empfand, dass er aber sofort übel wurde, sobald er den Kopf aufrichtete. Eine ähnliche Beobachtung machte Bárány auf einer 'scenic railway'. Bei steiler Abwärtsfahrt hatte er ein unangenehmes Gefühl im Magen, das bei Vorwärtsneigen des Kopfes und Rumpfes um 90 Grad völlig schwand. Bei der Seekrankheit handelt es sich offenbar um Reizwirkungen auf die Otolithenmembran. So viel Mittel auch gegen dies Uebel angegeben worden sind, so wenig haben sie sich doch allgemein bewährt. Ob die Lumbalpunktion, welche Bárány für sehr schwere Fälle vorgeschlagen hat, bisher überhaupt zur Anwendung kam, entzieht sich meiner Beurteilung.

Dauernde Schädigungen der Otolithenmembranen des Utriculus und Sacculus, wie sie Wittmaack durch ausserordentlich schnelle Rotation — 2,000 Umdrehungen in der Minute — bei Meerschweinchen erzeugte, finden in der menschlichen Pathologie kein Analogon. Wittmaack beobachtete bei diesen Tieren, dass die Ablösung der Otolithenmembran dauernd bestehen blieb und dass sich auch eine gewisse aber nie vollständige Degeneration der zugehörigen Sinneszellen, Nervenzellen und Nervenfasern hinzugesellte. Die anfänglich schweren Störungen des

Gleichgewichts der Tiere gingen nach einiger Zeit zurück, dagegen blieben die Nystagmusbewegungen bei Drehungen längere Zeit aus, um schliesslich wieder teilweise zu erscheinen. Hörstörungen traten bei diesen Wittmaack'schen Versuchen nie auf und auch die Bogengänge zeigten keinerlei Veränderungen.

Ehe ich auf die Schädigungen durch mechanische, aktinische, chemische Reize, sowie die Folgen der Infektionskrankheiten und Vergiftungen und die schädigenden Wirkungen von Tumoren eingehe, sei es mir gestattet, kurz die Beziehungen der Otosklerose zum inneren Ohr zu besprechen, ein Leiden, welches nach dem Vorschlag von Siebenmann auch als progressive Spongiosierung der Labyrinthkapsel, nach Manasse als Ostitis chronica metaplastica bezeichnet wird. Lebhaft diskutiert wurde die Frage, ob bei dieser Krankheit das nervöse Endorgan der Schnecke in Mitleidenschaft gezogen wird oder nicht. Hammerschlag hält die hereditär degenerative Taubheit, die progressive labyrinthäre Schwerhörigkeit und die Otosklerose für verschiedene Erscheinungsformen einer einzigen, genetisch einheitlichen Erkrankung, Manasse, dem Panse sich anschliesst, geht so weit, dass er die Diagnose auf Otosklerose nur aus dem pathologisch-anatomischen Bilde, nicht aber aus der Funktionsprüfung gestellt wissen will. Die Mehrzahl der Otologen hält heute wohl noch an der alten Bezold'schen Trias fest und fordert, dass das Krankheitsbild der Stapesankylose beibehalten wird. So vor allem Siebenmann, Denker, Scheibe, Brühl, Politzer und viele andere.

Auch über die Genese der Knochenerkrankung gehen die Ansichten weit auseinander. Habermann neigt zu der Ansicht, dass die Erkrankung von der Periostlage der Schleimhaut ausgeht. Dagegen sprechen die Fälle, wo das Leiden weit vom Periost entfernt im Knochen sitzt, insbesondere die Fälle mit circumscripten Inseln in der Nähe des inneren Gehörgangs, wie sie von Lindt, Jörgen Möller und Manasse beschrieben wurden. Politzer und mit ihm viele andere halten den Beweis für erbracht, dass es sich bei der Otosklerose um eine primäre Erkrankung der Labyrinthkapsel handelt.

Ueber die histologischen Veränderungen, die sich bei der Otosklerose abspielen, ist eine einheitliche Auffassung noch nicht erzielt worden. Bezüglich dieser Frage verweise ich auf die Ausführungen Manasses und diejenigen Siebenmanns. Nach der Auffassung Siebenmanns beruht eine konkomitierende Erkrankung des Nervus cochlearis auf dem Einfluss der bis ans Endost selbst vorgedrungenen Wandveränderungen der Labyrinthräume und ist da, wo solche fehlen, auf andere Ursachen zurückzuführen.

Brühl kommt zu dem Schluss, dass Labyrinthatrophie weder zum klinischen noch anatomischen Bild der Otosklerose gehört.

Ohne allen Zweifel gibt es Fälle, in denen der Spongiosierungsprozess zunächst die Stapesgegend frei lässt und bei denen es evtl. erst später oder gar nicht zur Beteiligung der Stapesgegend kommt. Für derartige Fälle müssen wir der Anschauung Manasses beipflichten, dass die Diagnose

Otosklerose nicht gestellt werden kann, falls nicht etwa das von Schwartze zuerst erwähnte Symptom des Durchscheinens der geröteten Promontorialwand vorhanden ist.

In therapeutischer Beziehung sind zur Bekämpfung der Otosklerose sehr verschiedenartige Vorschläge gemacht worden. Die von Siebenmann empfohlene Phosphortherapie dürfte wohl die meisten Anhänger haben und auch ich kann mich dem Eindruck nicht verschliessen, dass die durch Monate und Jahre fortgesetzte regelmässige Verabreichung kleiner Phosphormengen noch am ehesten geeignet ist, wenn auch nicht das Leiden zu heilen, so doch die Progredienz des Prozesses zu verlangsamen. Erwähnt sei, dass Siebenmann und Beck (New York) gelegentlich bei Otosklerose durch Adrenalininjektionen Besserung der Ohrgeräusche beobachteten. Dundas Grant empfiehlt Chinin in kleinen Dosen, Pritchard grosse Dosen von Acid. hydrobromic. oder Salizyltabletten und warnt vor grossen Chinindosen. Davis empfiehlt Chinin mit Acid. hydrobromic. und Tct. nucis vomicae, Beck, der, wie schon erwähnt, auch den Nutzen der Adrenalininjektionen rühmt, experimentierte ausserdem mit Extrakten von Schilddrüse, Milz, Thymus, Pankreas und Hoden, ohne dadurch eine Beeinflussung der Symptome zu erreichen. Denker hat den Vorschlag gemacht, wegen des häufigen Auftretens der Otosklerose in der Gravidität, bei der sich erfahrungsgemäss gelegentlich Hypophysen- und Knochenveränderungen zeigen, ausser Phosphor Hypophysin und Pituitrintabletten zu geben, evtl. auch Keimdrüsenextrakte: Ovarialtabletten (Merk), Spermintabletten (Poehl). Schliesslich sei erwähnt, dass Schwarz verblüffende Erfolge durch Röntgenbestrahlung gesehen haben will. Die schon früher von Passow bei der Stapesfixation geübten Anbohrungsversuche sowie die neuerdings von Bárány vorgeschlagene Eröffnung des hinteren vertikalen Bogenganges ist wohl allgemein aufgegeben worden.

Die Schädigungen des Labyrinths durch grob mechanische Einwirkung

Eine ausgezeichnete Zusammenstellung der sämtlichen traumatischen Schädlichkeiten, die das Ohr in Mitleidenschaft ziehen, findet sich in der Monographie Passows, *Die Verletzungen des Gehörorgans*. Ich möchte deshalb nur auf die Arbeiten kurz einzugehen, die seit jener Publikation erschienen sind.

In einer gründlichen Studie über die Beteiligung des innern Ohres nach Kopferschütterung mit vorzugsweiser Berücksichtigung derjenigen Fälle, bei denen die Hörfähigkeit für die Sprache gar nicht oder in einem praktisch nicht in Betracht kommenden Grade gelitten hat, verdanken wir Rhese. Dieser Autor untersuchte 100 junge Leute, bei denen Altersveränderungen des Gehörorgans sowie berufliche Schädigungen durch Beschäftigung im Lärm, ferner Erkrankungen, spez. Organ- und Nervenerkrankungen, ausgeschlossen waren, weil bei ihnen die Folge des Schädeltraumas um so prägnanter hervortreten musste. Bei den sofort nach der Kopfverletzung vorgenommenen Untersuchungen ergab

sich eine immer wiederkehrende Symptomenreihe (ich zitiere hier Rhese fast wörtlich) : Nystagmus beim Blick nach der nicht verletzten Seite, bezw. in stärkerem Grade beim Blick nach dieser Seite, Gleichgewichtsstörungen, Verkürzung der Knochenleitung, Verkürzung der Perzeptionsdauer in Luftleitung für den ganzen Tonbereich, hochgradige zu der Hörweite für Sprache in starkem Gegensatz stehende Herabsetzung der Hörweite für die Taschenuhr, eine durch ihre Häufigkeit auffallende Ermüdbarkeit des Hörnerven Stimmgabeltönen gegenüber, teils in einem dem normalen Ohr gegenüber gesteigerten Grade des positiven Ausfalls des Rinné'schen Versuchs, starke Gefässfüllung der oberen Gehörgangswand auf der verletzten Seite, zuweilen Ekchymosen und Blutkrusten hier und am Trommelfell. Diese an und für sich nicht eindeutigen Symptome, unter denen Nystagmus fehlen und zu denen Schwindelgefühl, Gleichgewichtsstörungen und subjektive Geräusche hinzutreten können, geben durch ihr Zusammentreffen ein circumscriptes Bild. Als Gradmesser für alle auf das Ohr bezogenen subjektiven Beschwerden — Schwindelgefühl, subjektive Geräusche — kann im allgemeinen der Grad der bestehenden Hörstörung gelten. Jedenfalls scheinen wirklich belästigende subjektive Geräusche bei normaler bezw. nur wenig herabgesetzter Hörweite für Flüstersprache selten zu sein ; Schwindel und Gleichgewichtsstörungen sind hartnäckiger, sie können auch bei guter Hörfähigkeit viele Jahre bestehen bleiben, allerdings in einem die Erwerbsfähigkeit nicht beeinträchtigenden Grade. Die traumatische Herkunft einer Reihe von Folgezuständen der Kopferschütterung wird häufig noch Jahre nach dem Unfall durch die Ohruntersuchung wahrscheinlich gemacht. Unter ihnen spielt der mit Tachykardie einhergehende vasomotorische Symptomenkomplex eine Hauptrolle, er kann Schwindelgefühl und Gleichgewichtsstörungen verstärken, bezw. bestimmte Formen dieser Störungen hervorrufen ; er scheint auch in einer Reihe von Fällen unmittelbarer Folgezustand einer Labyrinthläsion zu sein. Auch hier entspricht meistens der Grad der Ohrensymptome dem Grad der sonstigen Störungen, doch gilt das nur *ceteris paribus*, da auf die Einwirkung der vasomotorischen Störungen auch noch andere Faktoren Einfluss zu haben scheinen. Je weniger von organischer Läsion des innern Ohres nachweisbar ist, um so mehr ist auf die vorzugsweise psychogene Entstehung zu rekurrieren.

Genaue pathologisch-anatomische Untersuchungen über die Folge von Schädelbasisfrakturen auf das innere Ohr verdanken wir Habermann, Panse, Manasse, seinem Schüler Théodore, Barnick, Lange, Nager, Sokai, Stenger u. a. Während man früher als Ursache für die Labyrinthschädigungen molekulare Veränderungen der nervösen Teile (Schwartze), Lähmungen der vasomotorischen Nerven (Friedmann), Alterationen der Neurone als Folge übermässiger Erschütterung der Endolymphe, Lockerung des Zusammenhangs der Zellen des Cochlearis und Vestibularis (Spira), Schädigungen der zentralen Akustikusbahnen (Oppenheim) annahm, gewinnt die früher schon von Politzer, dann von Gradenigo und Müller

vertretene Ansicht, dass Blutungen im Labyrinthinnern die hauptsächlichste Ursache für die Schädigungen sind, mehr und mehr an Wahrscheinlichkeit.

Beobachtenswert in dieser Hinsicht ist namentlich die Arbeit von Sokai, der 9 Felsenbeine von 5 an Schädelbasisfraktur gestorbenen Menschen untersuchte, bei denen die Labyrinthkapsel selbst nicht frakturiert war. Er fand lediglich Veränderungen in den Nervenstämmen und im häutigen Labyrinth, und zwar regelmässig Blutungen im Hörnervenstamm, 8 mal Zerreissung von Nervenfasern im Ramus cochlearis, 4mal im Ramus vestibularis, ausserdem Blutungen an den verschiedensten Teilen des innern Ohres. Sokai ist daher geneigt, diejenigen Fälle von traumatischer Taubheit, die bei der Autopsie keine gröberen Veränderungen, sondern nur degenerative Atrophie der nervösen Elemente zeigen, als durch Blutungen und Gewebszerreissungen in den Labyrinthweichteilen entstanden zu erklären. Als Prototyp eines solchen Falles führe ich die Beobachtung von Théodore aus der Manasse'schen Klinik an, in welchem es sich ebenfalls um eine Commotio cerebri bezw. labyrinthi ohne Knochenveränderung handelt.

Als hauptsächlich befallen erwies sich das Ganglion spirale, in dem sich ein Schwund der nervösen Elemente und reichliche Bindegewebsneubildung, im stärksten Masse in der Basalwindung, viel weniger in den übrigen Windungen, zeigte. Das Cortische Organ zeigte merkwürdigerweise auch in der Basalwindung nur eine geringe Atrophie; das Ligamentum spirale wies hydropische Degeneration auf, in der Scala vestibuli fand sich Neubildung von Bindegewebe sowie Pigmentzellen. Der Befund glich also in hohem Masse dem, den wir bei der chronischen degenerativen Neuritis anzutreffen pflegen.

Dass bei diesen Verletzungen nicht alle Teile des Labyrinths gleichmässig schwer betroffen werden müssen, geht aus mehreren Beobachtungen mit Sicherheit hervor. Es ist deshalb wohl nicht notwendig mit Ruttin anzunehmen, dass wenn bei einem Menschen, nach einem Schädeltrauma gutes Hörvermögen bei kalorischer und rotatorischer Unerregbarkeit des Vestibularapparates besteht, eine zentrale Ursache der Funktionsstörung bestehen muss.

Sehr berechtigt ist die Warnung Lermoyez und Hautants, bei vorausgegangenem Schädeltrauma mit folgender Taubheit das Erhaltensein der Reaktion des Vestibularapparates nicht als Beweis für das Nichterfolgtsein einer Labyrinthverletzung anzusprechen.

Die Veränderungen, welche sich bei Individuen finden, die eine schwere Schädelbasisfraktur längere Zeit überleben, haben Manasse, Nager u. a. geschildert. Manasse teilt diese in folgende 3 Gruppen ein: 1. das Trauma und seine Heilung: es fand sich eine feine symmetrische Fissur im Labyrinth, die nicht durch das ganze Felsenbein ging, sondern nur durch einen Teil des Vestibularapparates, die Schnecke aber fast vollständig frei liess. Die Fraktur war fast überall bindegewebig, nur an ganz vereinzelten Stellen knöchern geheilt. 2. Die Knochenneubildung

im Labyrinth: es bestand hier von der Stelle der Verletzung ausgehend eine durch Periostitis ossificans hervorgerufene Knochenneubildung, die eine starke Verdrängung, Abschnürung mit Cystenbildung, sowie vollständigen Schwund der Weichteile zur Folge hatte. 3. Sekundäre degenerative Atrophie der nervösen Teile.

Was die Atrophie bei Schädeltraumen aller Art, die das innere Ohr in Mitleidenschaft ziehen, angeht, so hat der Vorschlag von Neumann und Voss, bei allen eitrigen Prozessen des äussern Gehörganges und des Mittelohres aktiv vorzugehen und evtl. das Labyrinth breit zu eröffnen, wohl aus dem Grunde bisher wenig Anklang gefunden, weil wir einerseits wissen, dass bei Schädelbasisfrakturen der Knochen des Labyrinths häufig nicht betroffen wird, also keine Kommunikation zwischen Mittelohr und dem Labyrinth eintritt und weil andererseits beim frisch Verletzten die Frage, ob eine solche Kommunikation zu Stande gekommen ist, nur in seltenen Fällen zu beantworten sein wird. Ruhe, Eisumschläge und Narkotika werden sicher noch lange das Feld beherrschen. Für die späteren Stadien dürften die namentlich von Manasse empfohlenen Pilokarpininjektionen von Nutzen sein.

Labyrinthitis serosa

Die klinische Erfahrung hat gelehrt, dass es gelegentlich bei der akuten und chronischen Mittelohreiterung spontan, sowie nach Radikaloperationen zu Schädigungen des Labyrinths ohne Kontinuitätstrennung desselben kommen kann, die wir unter dem Namen Labyrinthitis serosa zusammenfassen (Siebenmann, Nager, Alexander, Scheibe, Herzog, Ruttin, Leidler, Urbantschitsch, Grünberg, Bondy, Biehl, Woyatschek, Herzfeld u. a.).

Sehr schwer zu entscheiden ist die Frage, ob es vollauf berechtigt ist, die Labyrinthitis serosa streng von der eitrigen Labyrinthitis zu trennen. Gegen die Bezeichnung Labyrinthitis serosa hat sich Scheibe ausgesprochen, weil mit den Veränderungen des Chemismus der Peri- und Endolymphe meist auch wohl zelliges Exsudat im Labyrinth auftritt. Auch Bárány hält eine sichere Unterscheidung zwischen seröser und eitriger Labyrinthitis schon deshalb für unmöglich, weil wir nie wissen, ob die sog. seröse Labyrinthitis nur auf toxischer Grundlage oder auf einer abgeschwächten Infektion beruht. Aus diesem Grunde hat man auch vorgeschlagen, die Affektion als Labyrinthitis toxica (O. Mayer), bezw. als entzündliches kollaterales Oedem des Labyrinths (Voss) zu bezeichnen. Durch die Beobachtungen von Uchermann, Rupprecht, und Ruttin ist es zwar sicher erwiesen, dass chemische in das Mittelohr gebrachte Agentien imstande sind, auch das Labyrinth zu reizen. Bei einem Patienten Uchermanns kam es offenbar durch Jodoformgazetamponade zu einer vorübergehenden Irritation des Labyrinths. Rupprecht hat einen Fall beobachtet, der nach Chromsäureätzung in der Gegend des runden Fensters unter Zeichen schwerer Labyrinthreizung ertaubte. Im

Laufe von einigen Wochen gingen diese Erscheinungen unter Wiederherstellung des Gehörs zurück. Ruttin erwähnt, dass seinerzeit in Wien nach Radikaloperationen die Wundhöhle mit 10 Prozent Formalinlösung ausgespült wurde, wodurch in vielen Fällen ganz akute Labyrinthitiden einsetzten, so zwar, dass am ersten oder zweiten Tage nach der Operation alle Erscheinungen der Labyrinthitis auftraten, jedoch allmählig wieder zurückgingen.

Nach den Untersuchungen Meyers ist es auch durchaus wahrscheinlich, dass es vor der eitrigen Erkrankung des innern Ohres ein Stadium der Labyrinthitis geben muss, während dessen Bakterien noch nicht in das Labyrinth eingedrungen sind. Meyer konnte bei einem an einer Scharlach-Otitis gestorbenen Individuum feststellen, dass die Paukenschleimhaut eine diphtherisch-nekrotische Entzündung aufwies, während im Labyrinth ausser einer ausgesprochenen Hyperämie sich fibrinöses Exsudat in der Cysterna perilymphatica und an der Innenseite des runden Fensters fand. Die Faserung des Ringbandes war verwischt. Leukocyten fanden sich auf und in dem Ligamentum spirale der Basalwindung, die Sinneszellen zeigten eine beginnende Degeneration, die Endolymphe in den Vorhofssäckchen und in den Ampullen war geronnen. Während nun in der Pauke sich massenhaft Kettenkokken befanden, war das Labyrinth, das Ringband und die Fenstermembran frei von diesen.

Wittmaack, Holmgreen und Uchermann stellen mit Recht für die Labyrinthitis serosa das Postulat auf, dass der Liquor cerebrospinalis frei von Bakterien sei, und Wittmaack spez. macht darauf aufmerksam, dass bei einer sicher eitrigen Labyrinthitis mit positivem Streptokokkenbefund des Liquor cerebrospinalis das Gehör bei einem seiner Patienten noch sicher z. T. erhalten war.

Trotz der verschiedenen ausgeführten Bedenken dürfte es sich aus praktischen Gründen dennoch empfehlen, an der Bezeichnung Labyrinthitis serosa festzuhalten. Wir werden in Analogie zu den histologisch sicher festgestellten circumscripten Labyrintheiterungen (Siebenmann, Yoshii, Görke) auch eine circumscripte Labyrinthitis serosa von einer diffusen zweckmässiger Weise unterscheiden, da uns die klinische Beobachtung lehrt, dass bei diesem Reizzustande des Labyrinths nicht alle seine Teile gleichmässig betroffen werden müssen.

Auf Grund der experimentellen Studien Herzogs war man längere Zeit geneigt, anzunehmen, dass eine Reizung des Labyrinths stets das ganze Labyrinth befallen müsse, bald aber mehrten sich die Beobachtungen, aus denen mit völliger Sicherheit hervorging, dass es auch circumscripte Erkrankungen des Labyrinths gibt. Durch weitere experimentelle Studien hat sich Herzog übrigens selbst dazu bekannt, dass seine Lehre von der uneingeschränkten Ausbreitung entzündlicher Vorgänge im Labyrinth fallen gelassen werden müsse.

Ausser den Labyrinthfenstern kommen für die Ueberleitung von Reizerscheinungen sicher auch Gefässverbindungen zwischen Pauke und Labyrinth in Frage.

Alexander zeigte ein Präparat Politzers, das eine Anastomose zwischen Labyrinth und Paukengefässen durch das Promontorium hindurch durchaus wahrscheinlich machte, was allerdings von Habermann und Katz bestritten wurde. Auch Alt sagt in der *Enzyklopädie für Ohrenheilkunde*, dass es im Verlaufe von akuten, eitrigen Mittelohrentzündungen durch die anastomotische Gefässverbindung zwischen mittlerem und innerem Ohr zu starker Hyperämie, zu seröser Exsudation, seltener zu eitriger Entzündung des Labyrinths kommt. Ferner hat Manasse eine Gefässkommunikation zwischen Bogengang und Mittelohrschleimhaut gefunden, worüber die von ihm wiedergegebene Abbildung keine Zweifel aufkommen lässt. Ganz besonders wichtig ist die Arbeit Zanges, der auf Grund histologischer Studien den Beweis führte, dass beim Menschen am horizontalen Bogengang und in der Gegend des runden Fensters regelmässig Gefässverbindungen zwischen Mittelohr und Labyrinth bestehen.

Was die Therapie der Labyrinthitis serosa betrifft, so hat sie bei akuten Mittelohrentzündungen in sofortiger Freilegung des Infektionsherdes im Mittelohr zu bestehen, um einer schwereren und dauernden Schädigung der zarten membranösen und nervösen Gebilde des innern Ohres vorzubeugen. Anders liegen die Verhältnisse bei den serösen Labyrinthitiden, die sich im Anschluss an Radikaloperationen ausbilden. Diese Prozesse unterscheiden sich von den gröberen traumatischen Läsionen des Labyrinths dadurch, dass sie nicht unmittelbar im Anschluss an die Operation, sondern erst im Verlauf einer kürzeren oder längeren Zeit 12–24 Stunden *post operationem* in Erscheinung treten. Auch erreichen sie ihren Höhepunkt bisweilen erst einige Tage nach der Operation und klingen meist rascher ab, als die Erscheinungen nach Labyrinthverletzungen. Fieber tritt dabei nicht auf. Unser therapeutisches Verhalten ihnen gegenüber muss ein rein expektatives sein (Voss).

Bei den Schädigungen im Anschluss an Radikaloperationen scheint allerdings die Neigung zur völligen Wiederherstellung der Funktion nicht in dem Masse vorhanden zu sein, wie bei der Labyrinthitis serosa, die durch eine akute eitrige Mittelohrentzündung ausgelöst wird (Alexander).

Ueber das sog. Fistelsymptom bezw. Kompressionsnystagmus

Bei chronischen Mittelohreiterungen, spez. beim Cholesteatom, kommt es in einer nicht ganz geringen Zahl von Fällen — Bárány fand unter 160 Radikaloperierten 11 mal das sog. Fistelsymptom — zur Usur der Labyrinthwand, und zwar am häufigsten am horizontalen Bogengang. Von dieser Regel gibt es nur vereinzelte Ausnahmen, bei denen sich die Fistel an anderen Stellen der Labyrinthkapsel findet; beispielsweise in der Ampulle des vertikalen Bogenganges (Bárány), am Promontorium (E. Urbantschitsch).

Auch sei erwähnt, dass in ganz vereinzelten Fällen bei akuten Eiterungen das Fistelsymptom beobachtet wurde.

Das Symptom ist dadurch charakterisiert, dass bei Kompression vom

Gehörgang aus ein Nystagmus rotatorius horizontalis nach der kranken Seite hin auftritt und bei Aspiration ein schwächerer horizontaler rotatorischer Nystagmus nach der entgegengesetzten Seite. Dabei haben wir uns vorzustellen, dass durch den Druck bezw. die Saugwirkung eine Verschiebung des Labyrinthwassers zwischen Labyrinthwanddefekt und dem runden Fenster bezw. einem zweiten Defekt erfolgt. Der Angriffspunkt der Druckwirkung ist dort zu suchen, wo die Kraft senkrecht einwirkt. Ob dies einmal der Labyrinthwanddefekt, einmal das runde Fenster ist, hängt von individuellen anatomischen Verschiedenheiten und von den jeweils verschiedenen pathologischen Zuständen in der Umgebung des Labyrinthwanddefektes ab. Es ist deshalb begreiflich, dass selbst bei gleicher Lage des Labyrinthwanddefektes entgegengesetzte klinische Symptome beobachtet werden können, weshalb eine Lokalisation des Wanddefektes aus der Art und Richtung des Nystagmus nicht ohne weiteres möglich ist.

In der Tat finden sich auch in der Literatur mehrere Fälle, bei welchen die Kompression Nystagmus in der Richtung zur kranken Seite aufwies.

Ob die Beobachtung von Brünings, nach der bei einzelnen Personen das Fistelsymptom durch Druckdifferenz nicht mehr bestand, während schon auffallend schwache galvanische Ströme eine deutliche galvanische Reaktion mit starker horizontaler Komponente zeigten, in dem Sinne verwertet werden darf, dass man auf Grund dieser galvanischen Reaktion Kapseldefekte diagnostizieren soll, erscheint zweifelhaft, weil, wie Marx u. andere nachgewiesen haben, die galvanische Reaktion auch nach völliger Zerstörung des Labyrinths noch auftritt.

Voraussetzung für das Zustandekommen des Kompressionsnystagmus bei einem Labyrinthwanddefekt ist das Erhaltensein der Funktion des Vestibularapparates. Histologische Untersuchungen derartiger Labyrinthwanddefekte liegen bisher nur drei vor, zwei sind von Wagener beobachtet und einer von Schmiegelow. Der eine Fall von Wagener und der von Schmiegelow betraf einen Defekt im horizontalen Bogengang, während der zweite Fall Wageners eine Usur an der höchsten Stelle des oberen Bogenganges aufwies. In allen drei Fällen war das Endost an der Stelle der Usur in normaler Weise erhalten und das Labyrinth völlig frei von entzündlichen Erscheinungen. Eine freie Kommunikation zwischen Labyrinthinnern und Mittelohr bestand also nicht. Deshalb ist der Vorschlag Scheibes, nicht von einer Labyrinthfistel, sondern von einer Usur der knöchernen Labyrinthwand zu sprechen und nicht von einem Fistelsymptom, sondern von Kompressionsnystagmus, durchaus berechtigt.

Der Vollständigkeit halber sei erwähnt, dass Alexander und Lasalle, denen sich später auch Bárány, Ruttin, Uffenorde, E. Urbantschitsch u. a. anschlossen, zu der Ansicht neigen, dass Luftdruckschwankungen auch bei der Aufhebung der Kontinuität der Gehörknöchelchenkette spez. im Amboss-Steigbügelgelenk, ebenso wie bei Lockerung des Ringbandes Kompressionsnystagmus hervorgerufen werden kann.

Alexander glaubt diese beiden Krankheitsformen dadurch unterscheiden

zu können, dass sich bei einem Labyrinthwanddefekt stets, bei der Lockerung der Gehörknöchelchenkette niemals in der Anamnese Angaben darüber finden, dass zu irgendeiner Zeit einmal spontaner Schwindel aufgetreten ist. Er stiess hier auf den Widerspruch Báránys, der Fälle gesehen hat, bei welchen sich ein Labyrinthwanddefekt fand, ohne dass je vorher Schwindel aufgetreten war.

Scheibe hält eine Lockerung der Symphyse zwischen Stapesplatte und Labyrinthwand bei Cholesteatomeiterungen für sehr unwahrscheinlich und nimmt für die von Alexander beobachteten Fälle einen Defekt an der knöcheren Fussplatte an, wie er durch die histologische Untersuchung Habermanns beschrieben worden ist.

Einen lebhaften Meinungsaustausch hat die Frage angeregt, ob das Fistelsymptom als solches einen Grund zur Radikaloperation abgibt oder nicht.

Als Vorkämpfer der konservativen Richtung hat vor allem Scheibe die Aufmerksamkeit darauf gelenkt, dass das Freilegen der Fistel sehr häufig erst direkt zu einer Ertaubung, also zu einer schweren Labyrinthschädigung führt. Dass er mit dieser Behauptung nicht Unrecht hat, beweist die Auffassung Alexanders, die dahin geht, dass nur Fälle von akuter Eiterung mit Labyrinthfistel nach der Heilung ihr Hörvermögen behalten, während ausgeheilte Fälle mit chronischer Eiterung und Labyrinthfistel auf Grund einer Labyrinthitis serosa schliesslich infolge fortschreitender Degeneration der Endorgane ihr Hörvermögen einbüssen. Scheibe warnt in den Fällen, die aus anderen Gründen der Radikaloperation unterzogen werden müssen, ausdrücklich davor, nach der Fistel zu suchen. Ruttin, der früher auch schon Alexander gegenüber hervorhob, dass ihm Fälle bekannt seien, bei denen bei bestehendem Labyrinthwanddefekt die Radikaloperation weder zu einem Verlust des Hörvermögens noch der Reaktion des Vestibularapparates führte, trat auch Scheibe entgegen mit der Bemerkung, dass man bei einer Radikaloperation die Fistel niemals zu suchen brauche, weil sie stets frei zu Tage liege.

Die Verschiedenheit der Anschauungen Scheibes und Ruttins ist vielleicht dadurch bedingt, dass Scheibe, ebenso wie Siebenmann, prinzipiell die Cholesteatom-Matrix nicht von dem Knochen abheben, sie vielmehr nach Möglichkeit zu erhalten suchen, während viele andere Operateure — unter ihnen wohl auch Ruttin — prinzipiell die Cholesteatom-Matrix mit dem Löffel auskratzen. Interessant in dieser Hinsicht ist die Diskussionsbemerkung Habermanns, dass man durchaus nicht immer gleich die Fistel sehen müsse, wenn der Bogengang frei liege, dass dies vielmehr erst dann geschehe, wenn man die Epidermis wegkratze und dadurch die Fistel und mit ihr zugleich das Labyrinth eröffne.

Aus den Darlegungen Scheibes dürften jedenfalls viele Otologen die Lehre ableiten, bei nachgewiesenem Drucksymptom die innere Paukenhöhlenwand und die Gegend des horizontalen Bogenganges recht sorgsam zu behandeln und grade an dieser Stelle die Epidermis stehen zu lassen.

Sicher hat der Warnungsruf Scheibes dazu beigetragen, es zu vermeiden, die bei der Operation zwar ganz interessante, für den Patienten aber äusserst bedenkliche Druckreaktion an der Fistel selbst mit einer Sonde auszulösen, sowie bei erhaltener Labyrinthfunktion nur auf Grund der bestehenden Reizerscheinungen eine Labyrinthoperation anzuschliessen, oder die Fistel zu erweitern.

Degeneration einzelner Labyrinthabschnitte

Kommt es im Anschluss an Mittelohreiterungen zu einem degenerativen Prozess im Labyrinth, so pflegt dieser in der Regel sowohl den cochlearen, wie den vestibularen Abschnitt gemeinsam zu befallen. Viel seltener sind die Fälle, bei denen die cochleare Funktion erlischt und die vestibulare erhalten bleibt (Neumann, Schmiegelow, Passow, Ruttin).

Bisher konnte nur ein derartiger Fall, und zwar von Ruttin histologisch untersucht werden. Das Präparat stammte von einer Frau, die bei einer chronischen Eiterung ohne Schwindel taub geworden war, kalorisch aber erregbar blieb. Es fand sich eine vollständige Ausfüllung der Cisterna perilymphatica bei intaktem Vestibularapparat.

Das umgekehrte Verhalten, Erlöschen der Vestibularfunktion bei erhaltenem Gehör, scheint bei Eiterung wenigstens bisher noch nie beobachtet worden zu sein. Auch bei anderweitigen Erkrankungen findet sich ein isoliertes Befallensein des Vestibularis recht selten. Als Beispiel sei angeführt der Fall von Frankl Hochwarts, welcher eine Patientin mit intaktem Mittelohr sah, die plötzlich einen typischen vestibularen Schwindel ohne jede Hörstörung bekam. Leidler berichtet von einer Patientin aus der Alexander'schen Abteilung, die wahrscheinlich infolge von Wurstvergiftung mit Schwindel erkrankte, ohne dass das Gehör litt und bei der der Vestibularis unerregbar war. Der Fall Neumanns war mit Trigeminusanästhesie und Herpes auriculae kombiniert. Báránys Fall lag Lues zu Grunde.

Als Kuriosum sei die Beobachtung Ruttins genannt, der eine gekreuzte Ausschaltung auf der rechten Seite des Cochlearis, auf der linken des Vestibularis sah.

Beck beobachtete eine doppelseitige Vestibularaffektion bei gleichzeitig bestehender doppelseitiger Facialisparese auf Grund von Influenza. Die kalorische Reaktion fehlte auf der Höhe der Erkrankung, während die Drehreaktion erhalten aber herabgesetzt war. Mit der Besserung des Leidens kehrte die kalorische Reaktion in normaler Weise wieder.

Bemerkenswert ist auch die histologische Beobachtung von Marx an einem Hasen und einer Maus, die ausgesprochene Manegebewegungen zeigten und bei denen man mit Sicherheit auf hochgradige Veränderungen im Vestibularis nach den klinischen Erscheinungen geschlossen hatte. Die histologische Untersuchung bei dem Hasen ergab ausser einer Mittelohreiterung eine solche in der Schnecke, während sichere pathologische

Veränderungen des Vestibularapparates fehlten. Bei der Maus waren pathologische Veränderungen im Labyrinth überhaupt nicht mit Sicherheit nachzuweisen. Es bleibt hier also nur übrig, entweder eine toxische Wirkung auf das Labyrinth oder eine seröse Labyrinthitis als Ursache der Reizung anzusehen.

Die Tatsache, dass die Schneckenfunktion bei Mittelohreiterungen häufiger geschädigt wird, als die vestibulare Funktion, hat ihre Analogie ja auch in anderen Prozessen, die das innere Ohr schädigen. Ich erinnere an die histologischen Studien, die Stütz bei Anämie und Arteriosklerose erhob, an die Beobachtung Zanges, der bei einem Kleinhirnbrückenwinkeltumor ausgesprochene Degeneration im Cochleargebiet bei fast völliger Unversehrtheit des Nerv. vestibularis fand, an die experimentellen Studien Wittmaacks und Knicks, die Untersuchungen von Felsenbeinen Taubstummer durch Sakaturo Kano etc.

Die nicht durch Mittelohrentzündungen hervorgerufenen Labyrinthschädigungen

Wie ich schon in der Einleitung angedeutet, scheint es bei der ausserordentlich verschiedenartigen Aetiologie von Reiz- und Ausfallserscheinungen des Labyrinths nicht angezeigt, den Namen Ménière'scher Symptomenkomplex beizubehalten. Von Ménière'scher Krankheit sollte nur dann gesprochen werden, wenn ein Mensch unter heftigem Schwindel, Erbrechen, Ohrensausen und Schwerhörigkeit apoplektiform erkrankt. Als die Ursache eines solchen Anfalles haben wir Blutungen in das Labyrinth anzunehnen, wie sie vor allem bei Leukämie, ferner bei Nephritis, perniziöser Anämie, Arteriosklerose und Morbus makulosis Werlhofii beobachtet wurden (Politzer, Sugar, Citelli, Güttich u. a.).

Streng von ihnen trennen sollte man aber die Erkrankungen des innern Ohres, wie sie bei einem ganzen Heer von Infektionskrankheiten: Mumps, Typhus, Meningitis cerebrospinalis, Scharlach, Lues, Blattern, Malaria, rheumatische Affektionen, Sepsis, Osteomyelitis, zur Beobachtung gelangten. Ebenso haben die toxisch wirkenden Substanzen, die das Ohr zu schädigen vermögen, wie das Chinin, Nikotin, Natr. salicyl., Phenacetin, Antifebrin, Coffein, Chloroform, Atropin, Quecksilber, Alkohol, Cyankali, das Gruben-, Leucht- und Sumpfgas, das als Haarfärbemittel gelegentlich verwandte Argentum nitricum, das von den amerikanischen Aerzten als Wurmmittel zuweilen benützte Chenopodium und die Pilzvergiftungen nichts mit der eigentlichen Ménière'schen Krankheit zu tun.

Bei den durch Infektionskrankheiten hervorgerufenen Labyrinthschädigungen ist der sich im Labyrinth abspielende pathologisch-anatomische Prozess durchaus noch nicht für alle einzelnen Krankheitsformen festgestellt. Sicher handelt es sich vielfach um einen rein degenerativen Prozess am Nerven, der sich ohne eigentliche Entzündung abspielt.

Manasse nimmt übereinstimmend mit Brühl für chronische Fälle und ebenso Siebenmann für akute (Tuberkulose, Osteomyelitis, Carcinom,

Kachexie) als primäre Erkrankung eine Neuritis und Perineuritis an, die sich dann peripher weiter verbreitet. Da Manasse stets herdförmige Veränderungen im Hörnervenstamme, in mehreren Fällen aber das Cortische Organ ganz oder fast ganz intakt fand, sieht er die Nervenatrophie als das primäre, die Atrophie des Endorgans als sekundäre Folge der Nervenerkrankung an. Alexander sah bei einem Fall von Maserntaubheit Atrophie der Sinneszellen und des Stützapparates, während der Nerv intakt war. Er glaubt daher, dass es auch eine primäre isolierte Erkrankung des Endorganes des Nerv. cochlearis gebe und der Nerv durchaus nicht immer der Ausgangspunkt des Krankheitsprozesses sein müsse. So unterscheidet Alexander Fälle 1. mit totaler Atrophie des nervösen Apparates, 2. mit Atrophie des Nerven und intaktem Cortischen Organ, 3. mit Atrophie des Endorganes ohne Veränderung des Nerven.

Ausser diesen rein degenerativen Prozessen des Nerven sind wiederholt solche zur Beobachtung gekommen, bei denen sich Bindegewebs- und Knochenneubildungen im Labyrinth fanden, kombiniert mit einer mehr oder weniger ausgesprochenen Atrophie der nervösen Elemente. Diese Art der Labyrintherkrankung, die man als Labyrinthitis fibrosa et ossificans bezeichnet, ist von Nager auch in einem Fall von Carcinomeinbruch in das innere Ohr beobachtet worden. Das Tumorgewebe selbst war in das Vestibulum eingedrungen, während sich in der Schnecke Tumorgewebe nicht nachweisen liess, dagegen nur die eben erwähnten bindegewebigen und knöchernen Umwandlungen.

Fur andere Infektionskrankheiten dürfen wir aber wohl den Beweis als erbracht ansehen, dass es sich im akuten Stadium um eine Otitis interna handelt, die dann bei der Heilung zu einer Otitis interna chronica fibrosa mit sekundärer Labyrinthatrophie führt. Derartige histologische Befunde haben z. B. Marx bei Typhustaubheit, Alexander nach Masern erhoben.

Sicher gestellt ist ferner, dass die das Labyrinth treffenden Schädigungen bei einer Reihe von Infektionskrankheiten in weitem Masse rückbildungsfähig sind. Bekannt ist dies spez. bei Typhus, bei der Meningitis cerebrospinalis, bei Malaria und vor allem bei Lues. Für die reinen Giftwirkungen gilt diese Rückbildungsfähigkeit in noch viel höherem Masse.

Die Therapie dieser Affektionen einzeln hier zu erörtern würde zu weit führen, sie sind zudem im wesentlichen identisch mit den gegen das Grundübel anzuwendenden Massnahmen.

Auf die zahlreichen experimentellen Arbeiten, die der Klärung dieser Verhältnisse dienen sollen, so vor allem diejenigen von Wittmaack, Blau, Herzog, Wittmaack und Laurowitz, Beck u. a. näher einzugehen, ist hier nicht der Ort. Nur so viel sei gesagt, dass es schon bei Tieren recht schwierig ist, postmortale Veränderungen auszuschliessen, und dass bei Menschen doppelte Vorsicht bei der Beurteilung histologischer Befunde angezeigt erscheint, weil wir ja niemals in der Lage sind, das menschliche

Gehörorgan in der Weise zu fixieren, wie das bei Tieren durch intravitale Injektion möglich ist.

Auf die Erscheinungen der Hyperästhesia acustika, des labyrinthären Falschhörens sowie der subjektiven Geräusche möchte ich hier nicht näher eingehen, da zu dem ausgezeichneten von Hegener und Schäfer erstatteten Referat auf der Deutschen Otologischen Gesellschaft in Basel 1909 keine wesentliche neue Gesichtspunkte hinzugetreten sind.

Nicht unerwähnt lassen möchte ich zum Schluss die von Bárány eingeführte Zeigereaktion. Bárány fand, dass beim Hervorrufen eines horizontalen Nystagmus z. B. nach rechts bei aufrechter Kopfhaltung der Arm einer Versuchsperson, die die Weisung hat, den Arm gradeaus zu strecken und ruhig zu halten, dieser kontinuierlich langsam nach links abwich, und zwar mit einer Geschwindigkeit, die vollkommen analog der langsamen Nystagmusbewegung ist, während die rasche Bewegung des Nystagmus fehlt. Bárány hat diesen Versuch weiter ausgebaut und für verschiedene Gelenke verschiedene Stellungen gefunden, in denen sie am besten geprüft werden können. Ist nun durch irgendeinen pathologischen Prozess eine Veränderung im Kleinhirn vorhanden, so wird beim Hervorrufen des Nystagmus der Patient nicht, wie es der Norm entspricht, in typischer Weise vorbeizeigen, sondern im Gegenteil richtig oder gar nach der verkehrten Seite hin zeigen.

Wir haben durch diese Reaktion eine sehr wertvolle Methode gewonnen, die uns in Stand setzt, in Verbindung mit den anderen Untersuchungsmethoden differentialdiagnostisch Kleinhirnprozesse von solchen im Labyrinth mit grosser Sicherheit zu unterscheiden.

SECTION XVI

OTOLOGY

DISCUSSION No. 4

DIE ANATOMIE UND KLINIK DER NICHT EITRIGEN LABYRINTHERKRANKUNGEN

DISCUSSION

Professor KÜMMEL (Heidelberg). Es muss betont werden, dass Ménière allerdings das grosse Verdienst hat, zuerst den von ihm geschilderten Symptomenkomplex in Zusammenhang gebracht zu haben mit den gerade zu jener Zeit von Flourens studierten Läsionen des Bogengangsapparates; aber es ist wohl ganz sicher, dass die anatomischen Befunde, durch welche er diese Theorie zu stützen glaubte, durchaus Artefakte waren. K. möchte auch wünschen, dass für das klinische Krankheitsbild der nicht bakteriellen, nicht progressiven Labyrinthitis nicht ohne Weiteres der Name 'seröse Labyrinthitis' gebraucht werde: es steht ja fest, dass eitrige Labyrinthitis bestand in Fällen, wo die Labyrintherregbarkeit erhalten war, resp. wiederkehrte.

Mr. SYDNEY SCOTT (London). Professor Alexander's statement that in his opinion Ménière's classical case was one of leukæmia, prompted him to recall a case which he fully described in the *Archives of Otology*, 1908, and which led him to believe that Ménière's case was one of hæmorrhagic streptococcal labyrinthitis ending in meningitis serosa, coma, and death. The speaker adopted an unusual technique, by which the acute hydrocephaly was discovered.

Dr. BÁRÁNY (Wien). Ich möchte über einen Fall von Fistelsymptom bei intaktem Trommelfell berichten, zu dem ich vor wenigen Tagen in Brüssel ein Analogon gefunden habe, so dass zu erwarten ist, dass das bei diesen Fällen beobachtete Phänomen sich öfter vorfinden wird. Es handelte sich um einen Mann, der wegen eines erlittenen Unfalls auf die Wiener Ohrenklinik kam. Eine schwere Eisenstange war auf seinen linken Warzenfortsatz gefallen. Er klagte über Schwindel, Verschlechterung des Gehörs, Ohrensausen und Kopfschmerzen. Die Untersuchung

ergab eine Laesio auris internae mit cerebellaren Störungen. Der Patient stand lange Zeit in meiner Beobachtung. Eines Tages kam er nun mit der Klage, dass er, wenn er einen hohen Kragen anhabe und den Kopf nach links drehe, Schwindel bekomme. Es ergab sich, dass bei Druck auf eine Stelle zwischen Warzenfortsatzspitze und Unterkiefer in der Tat ein enorm starker Nystagmus horizontalis und rotatorius hervorgerufen wurde. Ich dachte damals zunächst an eine Fraktur des Warzenfortsatzes und Labyrinths und zog den Schluss, dass ich bei Kompression and Aspiration im Gehörgang ebenfalls, und zwar den entgegengesetzten Nystagmus erhalten müsse. Ich prüfte das Fistelsymptom und fand meinen Schluss bestätigt. Ich nahm daher die Basisfraktur an und demonstierte den Fall mit dieser Diagnose vor. Einige Zeit später stellte ich den Patienten in einem meiner Kurse vor. Einer meiner Hörer fragte mich, wie sich die andere Seite verhalte. Ich musste sagen, ich hatte dieses Symptom auf der anderen Seite nicht geprüft. Als ich aber nun die Prüfung vornahm, fand ich dasselbe Symptom auf der anderen Seite, nur etwas schwächer. Ich gab nun meine Diagnose der Basisfraktur auf und unterzog den Patienten einer neuerlichen genauen Untersuchung. Ich sandte ihn auf die Nervenklinik und erfuhr, dass er dort wegen Lues schon einige Zeit in Behandlung stand. Bei der weiteren genauen Untersuchung fand ich, dass auch Kompression der Carotis am Halse Nystagmus hervorrief, nur viel schwächer als unter dem Warzenfortsatz und entgegengesetzt in der Richtung. Ich hatte ferner den Eindruck, dass ich bei Druck zwischen Warzenfortsatz und Unterkiefer ein daselbst befindliches arteriosklerotisches Gefäss komprimierte, wenn ich den Nystagmus hervorrief. Der Fall blieb trotz dieser Befunde unklar und ich fand keinen analogen. Erst vor einigen Tagen sah ich einen zweiten, vollkommen analogen Fall in Brüssel. Auf dem Otologischen Kongress daselbst stellte Dr. Hennebert zwei Fälle von hereditärer Lues vor, mit dem von ihm zuerst bei diesen Fällen beschriebenen Nystagmus bei Kompression und Aspiration der Luft im Gehörgang. Der eine dieser Fälle hatte bei Kompression und Aspiration ganz enormen Nystagmus. Der Fall erinnerte mich an den von mir in Wien beobachteten. Ich erzählte meinen Fall und forderte Hennebert auf, seinen Fall sofort zu untersuchen, ob sich bei Druck zwischen Warzenfortsatz und Unterkiefer ebenfalls Nystagmus auslösen lasse. Wir untersuchten und fanden den erwarteten Nystagmus in voller Deutlichkeit. Druck auf die Carotid hatte keinen Effekt. Der Fall konnte allen anwesenden Herren demonstriert werden. Es scheint also, dass dieses Phänomen der Lues spezifisch ist, wenn wir auch seine Entstehungsart bisher nicht verstehen. Künftige Beobachtungen müssen hier erst Klarheit bringen.

In reply to Dr. BÁRÁNY, Mr. Scott said the clinical symptoms of the patient he referred to were observed in 1907, before Bárány described his ' pointing tests '.

Dr. Dundas Grant (London) was much interested in Marx's observation as to the effect of radium in producing deafness, and asked whether any further confirmation of these observations had been published.

He expressed surprise that so little had been said with regard to the beneficial effects of small doses of quinine in aural vertigo. In a consecutive series of 36 cases of aural vertigo, in which he had administered small doses of quinine, 28 had been very markedly benefited, the vertigo returning when the quinine was stopped and improving again on its resumption. This was quite in agreement with Dreyfuss's experiments on animals, published by him at the Bordeaux International Congress of Otology in 1904. Dr. Grant applied the caloric test in a number of his cases before and after the administration of the quinine and found that the irritability of the healthy side was diminished by the quinine while the diseased side was unaffected; it appeared, therefore, that in unilateral destruction of the labyrinth the vertigo was relieved by the quinine owing to its sedative action on the opposite side.

Dr. Grant expressed great indebtedness to Professor Bárány, and asked whether much use was made of cold air in the caloric test instead of water; he had a vivid recollection of a case in which, in the earlier days of the labyrinthine tests, the caloric one was carried out with much energy and the patient's condition was decidedly worse afterwards.

The noise machine was a very valuable instrument, but Dr. Grant asked how much allowance should be made for its over-action. He was certain that its use in one ear affected the opposite one, so that a patient who had some hearing might appear to have none during the action of the test; if, however, the patient heard while the test was in progress, it was positive evidence of great value. In the detection of simulated deafness it was most useful.

He reminded the meeting of his method of compression of the vertebral arteries in the sub-occipital region, for making a diagnosis between congestion and anæmia of the labyrinth, the symptoms of which were often nearly identical.

In reference to the 'fistula symptom' test he described a case in which the stapes was dislocated and suction in the meatus produced frightful mixed nystagmus and forced movements away from the observer. He had seen this same occurrence in another case in which it was certain that the stapes was not dislocated but where there was ankylosis of the malleus and incus. The mechanism of the joint, as described by Helmholtz, was therefore destroyed, and suction of the membrane and ossicles acted directly through them on the stapes. He thought when this symptom occurred it might be explained by the presence of a malleo-incudal[1] ankylosis.

In regard to deafness from exposure to noises (professional) he had

[1] Dr. Dundas Grant regrets that owing to a 'lapsus linguæ' he spoke of the incudo-stapedial joint. The joint concerned is the malleo-incudal.

an idea that an obstructive disease might act to some extent as a protective and asked whether there was any evidence for or against this.

Replies

Professor ALEXANDER (Wien) verweist bezüglich der Fragen des klassischen Falles Ménières auf den Wortlaut seines Referates. Es war infolge des Zeitmangels leider nicht möglich, diesen Teil *in extenso* vorzutragen. Die Nomenklatur ' seröse Labyrinthitis ' ist vor allem als *klinischer* Begriff wertvoll, der Name, den Alexander als erster angegeben hat, ist gut brauchbar. Dass *anatomisch* in Fällen von ' Labyr. serosa ' mitunter auch Zeichen eitriger Entzündung beobachtet werden, soll nicht geleugnet werden. Alexander hat aus diesem Grunde die Labyrinthitis serosa auch nicht in sein Referat der nicht eitrigen Labyrintherkrankungen einbezogen.

Professor VON EICKEN (Giessen). Auf die Fragen von Herrn Dundas Grant habe ich zu bemerken, dass nach Reinkings Untersuchungen an zwei Schlossern eine Mittelohraffektion wie es scheint das innere Ohr vor akustischen Schädigungen schützt.

Marx hat durch experimentelle Untersuchungen bewiesen, dass man mit Radium das Sinnesepithel zerstören kann. Röntgenstrahlen vermögen ebenfalls das Sinnesepithel zu zerstören, was Brünings bei einem Patienten mit Mittelohrcarcinom fand. Der Tumor griff auf die Dura über, konnte somit nicht total entfernt werden und wurde deshalb mit grossen Röntgendosen bestrahlt. Ich stimme Herrn Grant darin durchaus bei, dass wir in dem Chinin ein sehr gutes Mittel gegen Schwindelanfälle besitzen. Sicher ist ferner, dass wir kalte komprimierte Luft sehr gut für den Nachweis calorischer Erregbarkeit verwenden können.

Sehr bemerkenswert ist die Beobachtung von Herrn Grant, der starken Kompressionsnystagmus bei einem Individuum mit normalem Trommelfell fand. Ob wir aus diesem Symptom auf eine Ankylose zwischen Hammer und Ambos schliessen dürfen, muss aber wohl erst ein autoptischer Befund beweisen.

Das Auftreten des Nystagmus bei Kompression und Aspiration nach Luxation und Extraktion des Stapes ist leicht verständlich und schon mehrfach beobachtet worden.

SECTION XVI

OTOLOGY

DEMONSTRATION

HISTOLOGICAL SPECIMEN OF INFLUENZAL LABYRINTHITIS

By Priv. Docent Dr. F. R. NAGER, Zurich

The sections which I wish to demonstrate were cut from the temporal bones of a child who had died of *influenza meningitis*. Patient, a boy five years of age, developed an acute pharyngitis with meningeal symptoms. On the second day there was definitive meningitis, on the third day diminished hearing, on the fourth day complete bilateral deafness; but examination showed no evidence of middle-ear inflammation.

The duration of illness was six and a half weeks, during which time there was never any sign of otitis media beyond a slight redness of the drum membrane of transient character. Repeated lumbar punctures gave always a pure culture of the influenza bacillus.

Histological examination showed slight inflammation of the middle ears, especially in the immediate neighbourhood of the round and oval windows.

In both labyrinths is found the characteristic histological picture of subacute otitis interna in various stages: fluid pus, vascular granulation tissue, and obliterative changes with new-formed fibrous tissue and bone. The vestibule and semicircular canals are more affected than the cochlea. Side by side we see a process of proliferation and *obliteration* with new-formed bone and a destructive invasion of the bone which has led to an *extension* of the inflammation outwards from the labyrinth. Thus we find erosion towards the posterior fossa and also in the outer wall of the lateral semicircular canal (at the site of election for *invasion* from the middle ear into the labyrinth). There has also been a destruction of the membrane of the round window and of the ligament of the stapes; in one spot the foot-plate of the stapes shows a perforation.

The right membranous cochlea is not well preserved, but the left shows very clearly endo- and peri-lymphatic inflammation. While the hair cells and tectorial membrane are missing, the supporting structures can in places be recognized. The nerve fibres and cells of the spiral

ganglion show a dense round-celled infiltration and commencing fibrous degeneration.

In the internal meatus the nerve shows dense infiltration and degeneration and is surrounded by purulent exudate.

The *clinical importance* of this observation arises from the demonstration of the fact that we may have a primary purulent meningitis caused by the influenza bacillus just as we have from the meningococcus.

Further, that this may attack the *labyrinth* and cause *deafness* similar to that occurring in cerebro-spinal meningitis.

In view of such a demonstration influenza meningitis must now be added to the present list of possible causes of deafmutism.

It offers an explanation of cases of deafness reported after influenza without a concurrent otitis media. From a histological standpoint the case is of great interest from the presence of the fistula formation from within (so-called retrograde labyrinth fistula) at various points. Again, it is very remarkable to note the relatively good condition of the ductus cochlearis in spite of the presence of such a destructive process in other parts of the labyrinth.

In reply to the President's question about the histological method used in the slides demonstrated, Dr. Nager said that he generally followed the celloidin method used by Professor Siebenmann (vide *Anatomie und Pathogenese der Taubstummheit*, Bergmann, Wiesbaden).

SECTION XVI

OTOLOGY

DEMONSTRATION

MICROSCOPIC SPECIMENS ILLUSTRATING SOME OF THE NON-SUPPURATIVE DISEASES OF THE LABYRINTH

By Dr. J. S. FRASER, Edinburgh

Fracture of the cranial base, involving the labyrinth. Horizontal section of right ear. (There was a flow of blood and cerebro-spinal fluid from the right ear, as the result of a fall.)

There is a fracture through the outer wall of the labyrinth at the ampullary end of the external canal.

There is blood beneath the mucosa of the middle ear.

Hæmorrhage is present in the endolymph and perilymph spaces of the external and superior canals; also in the air-cells internal to the canals.

Blood is also present in the scalæ of the cochlea.

The fracture passes inwards to an air-cell above the internal meatus, but does not reach the posterior surface of the petrous. Fracture through the promontory and scala tympani of the cochlea.

The line of fracture passes inwards to the narrow canal, which contains the nerve, to the crista of the posterior canal.

All scalæ of the cochlea contain blood.

Hæmorrhage is also present in the vestibule, and in the posterior canal.

There are small hæmorrhages in the internal meatus and in the narrow canal mentioned above, i.e. canal for nerve to crista of posterior semicircular canal.

Neuritis of the seventh and eighth nerves. Patient aged 65. On the morning following a long day of exposure to cold the patient complained of deafness, giddiness, noises in the ear, and facial paralysis; external objects seemed to rotate. Examination two months later showed nerve deafness on the left side; sense of taste retained by anterior part of tongue on side of lesion. Patient died five months after onset of illness.

Small cell infiltration is present beneath epineurium of seventh nerve.

Hæmorrhage around the vestibular ganglion.

The vessels in the internal meatus appear thickened.

The labyrinth itself is healthy.

Senile deafness. Patient aged 80 years. Examination revealed nerve deafness.

The section shows apparently normal conditions of the two parts of the eighth nerve.
Corti's organ is well preserved in all coils.
Possibly in the basal coil there is some diminution of the ganglion cells and of the nerve fibres in Rosenthal's canal and bony spiral lamina.

Tumour involving eighth nerve. Patient aged 25; mentally dull; double optic neuritis; death from coma. Post-mortem, cranial surface of dura covered with small pedunculated tumours which had eroded the inner surface of the cranial bones, which appeared as if pitted by small-pox. In angle between pons and cerebellum there was a tumour mass about the size of a walnut on right side.

A tumour composed of dense fibrous tissue has greatly eroded the internal meatus; no osteoclasts are seen.
Hæmorrhage is present in the tumour.
The scala tympani of the cochlea is invaded by the tumour in all coils.
Ganglion cells have disappeared and are replaced by fibrous tissue.
Hæmorrhage in posterior canal.

Tumour of eighth nerve on right side. Patient aged 75; complained of giddiness; she was unable to walk without assistance. Examination, three years before death, showed that the middle tuning forks were faintly heard by the right ear; low tones and high tones lost; marked arterio-sclerosis (alcoholism). Post-mortem, tumour of eighth nerve on right side.

The tumour has eaten out the internal meatus.
Atrophy of eighth nerve.
Scala tympani of cochlea contains delicate fibrous tissue (tumour ?).
Scala tympani of basal coil filled with delicate connective tissue in which there is some new bone formation.
The smooth end of the posterior canal shows increase of connective tissue in perilymph space.
(The left ear of this patient showed an old labyrinthitis.)

The patient was a deafmute. Parents stated that the patient—a boy of 13—had always been deaf. Functional examination showed that the higher tuning forks were heard (?). Vestibular response to cold syringing active on both sides. The patient died of meningitis following chronic suppurative otitis media and extradural abscess on left side.

Left Ear

Reissner's membrane is vertical in all coils in left ear.
Malformation of Corti's organ.
In lower part of middle coil membrana tectoria is attached to stria vascularis.
Cochlear nerve seems well developed.

Right Ear

Reissner's membrane sunken in all coils in right ear.

Mal-development of Corti's organ in all coils. In upper part of basal coil membrana tectoria attached to stria, and above this a small mass of homogeneous material.

The cochlear nerve and ganglion seem well developed.

The internal meatus shows a layer of pus on the floor between the epineurium and the dura.

SECTION XVI

OTOLOGY

INDEPENDENT PAPER

TWO CASES OF LOSS OF CALORIC VESTIBULAR REACTION, WITH OPERATIVE FINDINGS

By EDWARD BRADFORD DENCH, M.D., New York

The following two cases are rather interesting as showing a sudden ablation in caloric reaction, together with the subsequent labyrinthine findings:

Case I. A woman, 24 years of age, presented at the New York Eye and Ear Infirmary, with the following symptoms: There had been a discharge from the left ear at intervals, since childhood, with great impairment of hearing. Two weeks before coming to the clinic the patient complained of severe pain in the ear, with rumbling noises, which continued up to the time of coming under observation. The patient also complained of dizziness, nausea, and vomiting. For the first week the patient vomited about twice daily, but during the last week this had disappeared. The patient was unsteady when standing, and had a tendency to fall backward and to the left side. The patient had headache at the onset of the present trouble. The right ear had been negative, as far as the patient's history is concerned. An examination of the drum membranes showed that there had been some previous trouble in the right ear. Examination of the left ear showed the canal somewhat narrowed, a slight purulent discharge, the membrana tympani apparently destroyed, and the normal situation of the membrana tympani occupied by a mass of granulation tissue. Slight tenderness anterior to the mastoid tip. On physical examination the patient appears to be a fairly well-developed adult female, but poorly nourished. There is slight spontaneous nystagmus on looking to the right. The patient is unsteady and falls backward and to the left side, with eyes closed. Gait unsteady, no ataxic symptoms of arms or legs. Face normal. Knee-jerks active, but not exaggerated. Babinski sign not present. Heart and lungs negative. The caloric test, on admission, showed an active labyrinth on each side. The caloric test on the following day showed no reaction either to the hot or cold water test upon the left side. The caloric test on the right side was normal. The patient

at this time falls toward the right when walking and jumping, and feels dizzy.

Owing to the fact that the caloric reaction had changed so rapidly in twenty-four hours, that the patient was extremely dizzy, and that the temperature had risen from $99\frac{1}{5}^\circ$ to $100\frac{4}{5}^\circ$ immediately after admission, together with the sudden loss of caloric reaction, the patient was immediately placed upon the operating table. The ordinary radical operation was first performed. A fistula was found in the horizontal semicircular canal, and the oval window was found open. This apparently explained the labyrinthine symptoms. Consequently, a complete labyrinthine extirpation was performed, according to the Neumann method—that is, by a free exposure of the dura in the middle cranial fossa and a free exposure of the lateral sinus posteriorly. The facial ridge was then lowered to its extreme limit. The bony structures were then removed in Trautmann's triangle and the labyrinth entered posteriorly in the usual way. This drained the labyrinth posteriorly. Anteriorly, the lip of bone separating the oval and round windows was removed, thus draining the first and second turns of the cochlea. Free drainage being established, the ordinary meatal flap was formed, the posterior wound sutured, and the wound dressed in the ordinary way. This patient made an uneventful recovery.

Case II was that of a boy, 14 years of age, who was admitted to my service at the New York Eye and Ear Infirmary, with a history of discharge from the right ear, of six months' duration. Two months after the appearance of this discharge he came to the clinic, and two months subsequently the discharge ceased. Three days before his admission to the hospital, and six months after the first appearance of the discharge, the patient complained of acute pain in the right ear and over the sternomastoid muscle. There had been no discharge from the right ear during the present attack. On account of the pain the patient had not been able to sleep for three nights. The patient also complained of headache and dizziness. No symptoms referable to the left ear. A physical examination showed granulation tissue in the right tympanic cavity, springing from the upper and posterior quadrant. Upon the removal of this granulation tissue it was evident that the ear had been the seat of a previous chronic suppurative process. There was a cicatricial membrane covering the tympanum and lying at a lower level than the normal drum membrane. This cicatricial membrane was reddened and somewhat bulging posteriorly. The temperature, at the time of the first examination, was $99\frac{4}{5}^\circ$. The patient seemed seriously ill and was therefore admitted to the hospital. The caloric test, made at the time of the patient's admission, showed that both labyrinths were active. The granulation tissue was removed from the right tympanic cavity immediately upon the patient's admission to the hospital. Following this slight operation, the temperature began to rise until in twelve hours it had reached 104°. It remained practically at this level for eight hours. On examining the

patient eight hours after the operation I found him to be a well-nourished boy, complaining of severe pain in the right side of the neck, and crying out with the almost characteristic meningeal cry. There was no stiffness of the neck, no muscular rigidity in any part of the body, the Babinski and Kernig signs were negative. There was slight nystagmus to the affected side. A careful caloric test showed that the right labyrinth was absolutely dead. This, in contradistinction to the examination made twenty-four hours before, which showed an active labyrinth on this side. This total absence of caloric reaction of the right labyrinth, together with the restlessness of the patient, made me believe the case to be one of meningitis following a labyrinth suppuration, probably excited by the removal of the granulation tissue from the right ear. The high temperature, however, and the absence of all other symptoms of meningitis—such as the absence of the Babinski and Kernig signs, the clear sensorium—and, in fact, no other symptoms aside from the restlessness and crying out of the patient, decided me to make a guarded diagnosis. A spinal puncture at this time showed that the cerebro-spinal fluid was clear, and was not evacuated under pressure ; the differential count of the fluid was normal, its reaction was slightly alkaline, and the carbohydrate test was normal ; the globulin test was also negative. A blood culture made at this time showed after eighteen hours two colonies of streptococci. Three or four hours later the plates showed two or three more colonies.

It was evident, therefore, that we had to deal, in this case, with a general streptococcic infection, and not with a meningitis. A radical operation was performed, and neither the sinus nor dura was exposed at the time of the operation. There was no fistula in any of the semicircular canals, and the oval and round windows were closed. Owing to the negative findings, as far as a meningitis of labyrinthine origin was concerned, and remembering the negative findings of the cerebro-spinal fluid—also bearing in mind the high temperature and the streptococcæmia—I decided to expose the lateral sinus. The sinus lay fully $\frac{1}{2}$ inch behind the limit of the radical operation, and the bone separating the sinus groove from the radical cavity was hard and dense. Nevertheless, when the sinus groove was opened there was an escape of fetid gas. The sinus was found collapsed and separated from the posterior surface of the petrous pyramid by a space of fully $\frac{3}{8}$ inch. No free pus was present in this space, although the sinus was covered with an exudate of coagulated fibrin. The sinus was rapidly exposed backward toward the knee and downward to just above the bulb. It was thrombosed throughout this entire area. The internal jugular was then exposed from the level of the omohyoid muscle to the base of the skull. It contained fluid blood. The facial vein was tied off between two ligatures at its junction with the jugular. Just above the level of the facial vein the internal jugular appeared thickened and white in colour. In other words, it seemed to be occupied by a clot at this level. The jugular was removed from just above the omohyoid to a point just below the base of the skull. The neck wound was closed in the usual way.

The lateral sinus was opened at a point midway between the knee and the bulb. The sinus wall was very much thickened, but after the lumen of the vessel had been entered and a curette introduced downward, free hæmorrhage occurred from below. Hæmorrhage was controlled in this direction by packing, and the torcular end of the sinus was curetted, with the result that free hæmorrhage occurred. This hæmorrhage was controlled by firm packing. The patient's temperature before operation was 105°. It fell within the next twelve hours to 99½°, and the following day it did not rise above 102⅖°. At noon the next day it was 101⅕°. It then gradually began to rise, and at midnight on the second day after the operation it was 102⅗°. At this time the patient began to complain of some pain in the right side. The physical signs showed that he was suffering from either a beginning pneumonia or from pleurisy with effusion. The fact that he had simple crepitation over this side did not render the diagnosis positive at this stage. Lumbar puncture was made on the third day after the operation. The spinal puncture showed a normal cerebro-spinal fluid. It was clear in appearance, the globulin test was negative, and Fehling's solution reduced. The cellular count was negative except for a few blood-cells. A small amount of albumin was present in the cerebro-spinal fluid. We assumed, therefore, that the patient was not suffering from meningitis, but that the rise in temperature was entirely due to the chest complication. On the following day, that is, the fifth day after the first operation, the patient's temperature rose at noon to 105⅘°. A lumbar puncture showed a clear fluid, but a cultivation of the fluid the following day showed long chains of streptococci. The next day a lumbar puncture showed that albumin was present, the globulins were in excess, and pus, blood, and streptococci were present in the cerebro-spinal fluid, thus clearly demonstrating that the patient had a meningitis. The patient was immediately taken to the operating room, and an exenteration of the labyrinth performed, according to the Neumann method, the steps of the operation being the same as those detailed in the previous case In this case the superior petrosal sinus crossed the field of operation, but was exceedingly large, and, consequently, it was impossible to open the cerebellar dura at the internal auditory meatus, as advised by Neumann, in these cases. The entire sigmoid sinus groove was filled with a sloughing sinus wall. The bulb was then thoroughly exposed and curetted, and the patient returned to bed. The temperature dropped after this operation to 102°, but rose in eight hours to 103°. It then fell to 101⅘°, and the patient was very comfortable. The pulse was good, and the patient simply complained of pain in the chest. Suddenly severe dyspnœa developed, the patient became cyanotic, and he died within half an hour of a sudden pulmonary embolus.

These two cases are of interest in that they show a sudden ablation of labyrinthine function—that is, the labyrinth, tested at intervals of twenty-four hours, showed a complete loss of caloric reaction. This seemed sufficient in each case to warrant the complete labyrinth opera-

tion, and the findings at the operation clearly demonstrated the wisdom of such a procedure.

The complete cure in the first case was evidently due to prompt interference. The second case was of unusual interest, in that we had to do with a sinus thrombosis, apparently of long duration, as judged by the operative findings. I do not think there is any question but that the sudden involvement of the labyrinth, in this case, was secondary to the sinus thrombosis. When we bear in mind the close relation which exists between the aqueductus vestibuli, the aqueductus cochleæ, and the lateral sinus, it is not difficult to understand why a thrombosis of the lateral sinus should cause a sudden ablation of labyrinthine function. The aqueductus cochleæ, as we know, lies just above the jugular bulb. The aqueductus vestibuli occupies the posterior surface of the petrous portion of the temporal bone, just in front of the descending portion of the sigmoid sinus. It seems to me beyond question, therefore, in the second case, that infection of the labyrinth occurred secondarily to involvement of the sinus, and that the sudden ablation of labyrinthine function was dependent upon the sinus thrombosis, and prompt interference in this second case would probably have been followed by results quite as satisfactory as those obtained in the first case, had not pulmonary embolism occurred and caused a fatal termination.

SECTION XVI

OTOLOGY

INDEPENDENT PAPER

PRÉVENTION DES AFFECTIONS SUPPURÉES DU LABYRINTHE

PAR M. LE DR. TRÉTRÔP, D'ANVERS (BELGIQUE)

LA question des pyolabyrinthites étant depuis le dernier Congrès otologique de Budapest[1] toujours à l'ordre du jour, il n'est pas sans intérêt d'examiner succinctement quels sont les moyens d'éviter l'infection du labyrinthe. Cette infection, en effet, l'une des plus graves de l'appareil auditif, outre les symptômes extrêmement pénibles et douloureux qu'elle détermine, compromet ou détruit la fonction auditive, quand elle ne s'attaque pas à la vie elle-même du sujet malade.

Et d'abord est-il en notre pouvoir de prévenir les infections du labyrinthe et sa suppuration consécutive ?

En d'autres termes pouvons-nous nous opposer efficacement aux causes multiples d'infection de l'oreille interne ?

Quelles sont les causes habituelles de cette infection ?

On peut les diviser en causes *directes* et en causes *indirectes*.

Les causes directes sont le traumatisme. Les causes indirectes sont les infections générales entraînant l'inflammation labyrinthique par voie sanguine et les inflammations de voisinage.

Causes directes. — Le traumatisme. S'il est des traumatismes dont la prophylaxie échappe à nos moyens d'action tels ceux qui résultent d'accidents, de méfaits, de tentatives de suicide, de rixes ou de faits de guerre, plaies par instruments tranchants ou piquants, armes à feu, etc., il en est d'autres que nous pouvons prévoir tels les traumatismes par introduction de corps étrangers dans l'oreille.

On devrait dès l'école enseigner aux enfants que rien n'est plus dangereux pour l'oreille elle-même et pour le contenu de la boîte crânienne que l'introduction de corps étrangers dans l'oreille, porte-plumes, épingle

[1] Dr. Schmiegelow, *Sur les indications du traitement opératoire des labyrinthites suppuratives secondaires et leur traitement* (XVI[e] Congrès intern. de Méd., Budapest, 1909), sect. xvi.

Dr. Trétrôp, *Les pyolabyrinthites* (XVI[e] Congrès intern. de Méd., Budapest, 1909, sect. xvi).

à cheveux, voire même cure-oreilles : appareils détestables. On ne peut toucher au conduit de l'oreille qu'avec de petits écouvillons d'ouate bien matelassés pour éviter à l'organe le contact de tout corps dur. Les coups, les giffles sur l'oreille, le lancement de projectiles telle la neige durcie en hiver, qui a fait suppurer pas mal d'oreilles moyennes, devraient être sévèrement interdits et même punis. Ceci est un côté de la prophylaxie.

Il y a lieu de parler aussi des traumatismes opératoires. En dehors de l'ouverture voulue du labyrinthe pour faire la cure d'une pyolabyrinthite, il convient, au cours des diverses opérations portant sur l'oreille moyenne d'être des plus circonspects dans le respect intégral de la paroi labyrinthique.

Le simple enlèvement de fongosités ou de polypes insérés sur la paroi labyrinthique, enlèvement fait par arrachement au lieu d'y procéder par section, dans une oreille moyenne suppurant et non-désinfectée, peut être suivi d'une pyolabyrinthite mortelle. Tout otologiste expérimenté en a vu des exemples.

La prophylaxie de ces accidents consiste à ne jamais enlever les polypes ou fongosités avant d'avoir fait un traitement antiseptique pour diminuer l'infection et atténuer la virulence des germes microbiens et lors de la libération de la caisse à procéder par sectionnement, sans faire la moindre traction avant section complète. Il est cependant des malades en imminence ou même en puissance d'infection labyrinthique au moment trop tardif des soins, qui, malgré ces précautions, n'échapperont pas à la pyolabyrinthite.

Je ne citerai pas la lésion de la paroi labyrinthique au cours d'une myringotomie. C'est une maladresse qu'il faut savoir éviter en faisant tenir convenablement le malade. L'ablation accidentelle de l'étrier au cours du curettage de l'oreille moyenne est un accident heureusement rare ; l'effraction de la saillie vers l'aditus du canal semi-circulaire externe, au cours d'une attico-antrectomie est à éviter par les procédés de protection connus.

Il est à remarquer, ainsi que je l'exposai dans mon rapport sur les pylolabyrinthites [1] à Budapest en 1909, que ces traumatismes ont ceci de particulier que leur gravité est ordinairement en raison inverse de leur importance : une perforation microscopique exposant à une infection parfois excessivement sévère.

En cas d'ouverture accidentelle, il semblerait donc indiqué d'ouvrir plutôt largement, ou tout au moins de se tenir prêt à procéder à cette ouverture à la moindre alerte.

Causes indirectes ou de voisinage. — Les pyolabyrinthites résultant des infections diverses : tuberculose, syphilis, infection scarlatineuse, diphtéritique, typhique, érysipélateuse, rubéolique, variolique, etc., comportent les mesures prophylactiques pour éviter ces maladies et je ne m'y arrêterai pas davantage.

[1] Dr. Trétrôp, loc. cit.

Mais parmi les causes indirectes, l'otite moyenne purulente occupe la première place. Les statistiques ne sont point d'accord sur le pourcentage exact de pyolabyrinthites par rapport aux affections purulentes de l'oreille moyenne, on peut toutefois estimer à au moins 1 pour cent sa fréquence. Les individus jeunes et le sexe masculin y sont plus exposés, l'affection est ordinairement unilatérale et affecte de préférence le côté droit.

Parmi les otites moyennes purulentes qui occasionnent le plus fréquemment la pyolabyrinthite, il faut citer en première ligne les otites moyennes purulentes anciennes négligées avec ou sans cholestéatome. S'il est plutôt rare qu'une otite moyenne purulente, soignée dès ses débuts par un spécialiste, aboutisse à une complication aussi grave, il arrive au contraire trop fréquemment que dans les otites moyennes purulentes non-traitées, à la moindre imprudence, cette complication soit imminente et que souvent encore le spécialiste arrive trop tard.

Que faire au point de vue prophylactique ? Répandre de plus en plus dans le public[1] — et encore une fois ici l'instituteur à l'école peut être un de nos plus utiles auxiliaires — la notion de la nécessité de faire soigner les moindres inflammations auriculaires, enseigner aux mères dans les crèches que, loin d'être salutaire, l'otorrhée compromet la santé et la vie de leurs nourrissons, insister auprès des adultes sur la nécessité de poursuivre un traitement complet jusqu'à guérison ou de rester sous surveillance médicale, lorsque l'assèchement définitif de l'oreille moyenne ne peut être obtenu.

Telles sont dans leurs grandes lignes les principales règles de prophylaxie de la complication la plus redoutable de l'inflammation auriculaire. Sans doute, elles ne nous apprennent, à nous, spécialistes, rien de bien neuf, mais il est de ces vérités qu'il est utile et nécessaire de redire de temps à autre, ne fût-ce que pour nous remémorer un côté du rôle de préservation sociale que nous pouvons si utilement remplir et dont nous avons le droit d'être fiers, car il tend une fois de plus au relèvement constant de la durée moyenne de l'existence humaine.

[1] Dr. Trétrôp, *L'art d'éviter les maladies contagieuses, mis à la portée de tous* (Paris, Baillière, 1905).

SECTION XVI

OTOLOGY

INDEPENDENT PAPER

BEITRÄGE ZUR LEHRE VON DEN ACUSTICUS-ERKRANKUNGEN BEI LUES

Von Primararzt Dr. E. VÁLY, Budapest

Von den Labyrintherkrankungen infektiösen Ursprunges kommen die meningialen und die mittelohrischen Ursprunges häufiger vor als die *hämatogenen*.

Die hämatogene Otitis interna kommt bei sekundären und tertiären Lues vor, und zwar viel häufiger bei hereditärer, als bei aquirierter. Hierzu kommen noch als paraluetische Erkrankungen der Tabes, der Paralysis.

Die luetische Erkrankung des Acusticus selber ist sehr schwer von ähnlichen Erkrankungen des Labyrinths und des Gehirns zu unterscheiden. Die Symptome sind für die Lokalisation nicht immer charakteristisch.

Der *Stamm* des Acusticus kann, wie die anderen Gehirnnerven, luetisch erkranken. *Zentral* als Teilerscheinung eines Gehirnbasisgummas (Neuritis acustica gummosa), dies ist die seltenere Form ; oder *peripher* infolge einer gummösen Erkrankung des Felsenbeines. Es kann entstehen als *selbständige* gummöse Neuritis oder *sekundär* infolge einer luetischen Periostitis des Schädelknochens.

Der periphere Teil des Acusticus kann bei Lues des inneren Ohres infolge verschiedener schädlicher Einflüsse ebenfalls erkranken, ohne dass auch das Labyrinth daran teilnimmt. Es ist also möglich, dass der N. cochlearis und der N. vestibularis einzeln oder beide gleichzeitig erkranken, ohne dass das Labyrinth selber krank ist. Es scheint also berechtigt anzunehmen, dass es eine separate Krankheit, *Neuritis acustica syphilitica*, gibt.

Es ist noch nicht entschieden, wie *häufig* die Acusticuserkrankung bei Luetischen auftritt, wie auch das nicht, wie *oft* die sogenannte nervöse Schwerhörigkeit luetisch ist (Busch [1]). Wahrscheinlich ist, dass die luetische Acusticuserkrankung viel häufiger ist als wir es früher dachten.

[1] Busch, *Beiträge zur Anatomie* etc. *des Ohres*, 1909.

Früher wurden nur die Fälle mit auffallender Schwerhörigkeit genau untersucht, obwohl es bekannt ist, dass die luetische Neuritis acustica manchmal ohne Symptome beginnt; sie verursacht nicht einmal subjektive Geräusche, und ist häufig auch mit einer Labyrintherkrankung zu verwechseln. So sammelt Gradenigo von 24 Autoren ca. 1400 Fälle von Ohrsyphilis, darunter 7 % des inneren Ohres. Nach O. Beck [1] ist bei 32 % der Acusticus-Schwerhörigen Lues nachgewiesen. Nach Bürkner [2] erkrankt der Acusticus bei 31 % der Luetischen, nach Brül bei 34 %. Alle diese Zahlen stammen aus der Zeit vor Wassermann. Nach Busch ist in 52 % der Acusticuserkrankungen Wassermann +. Habermann fand bei 42 hereditär luetischen Kindern in 4 Fällen Acusticusaffektion. O. Mayer [3] fand bei 20 % der akuten Luesfälle Acusticuserkrankungen.

In meiner Spitalpraxis fand ich in den letzten $2\frac{1}{2}$ Jahren, wie es meine Kasuistik beweist, bei 160 Acusticusaffektionen $39\frac{1}{3}$ % Wassermann +.

Die grosse Zahl der Acusticuslähmungen bei Lues kann auch beweisen, dass dieser Nerv *minder widerstandsfähig gegenüber den spezifischen Leiden ist, als die anderen* (Rosenstein).

Wie ich schon erwähnte, kann sich das Leiden auf dem Stamm des Acusticus beschränken, oder sich auf die benachbarten Gehirnnerven ausdehnen. Es kann der zentrale oder periphere Teil des Nerven erkranken oder beide zugleich. Am peripheren Teil wieder, kann entweder nur der N. cochlearis oder der N. vestibularis erkranken, oder beide gleichzeitig.

Im Nervenstamme entsteht interstitielle Entzündung, welche ins innere Ohr fortschreitend, auch das Endorgan des Acusticus angreift; später entsteht im Nerv auch kleinzellige Infiltration, interstitielle Proliferation; danu Hämorrhagien, Degeneration und Vernichtung der Nervenzellen und Fibrillen tritt auf; dann Kalkablagerung, Periostitis und Ostitis an den benachbarten Knochen, hauptsächlich an der Kapsel des Labyrinthknochens, Degeneration des Endothels der Blutgefässe, dann Arteritis und Arteriosclerosis (Heubner [4]); wenn die Gehirnnerven primär, oder wie häufiger sekundär erkranken, beginnt das Leiden immer am Perineurium, greift dann auf die Gefässe der Nerven über, und so entsteht in den Nerven nicht nur Drucksteigerung, sondern auch Ernährungsstörung mit allen ihren Folgen.

Nach Schwartze und Gerber [5] ist die luetische Acusticuserkrankung in der Regel bilateral; wenn sie nur einseitig ist, so steht sie stets mit einer vorgeschrittenen Blutgefässerkrankung in Verbindung. Von den 34 Fällen meiner Kasuistik (sekundären und tertiären) waren 23 Fälle bilateral, das ist 67,9 %.

Es ist charakteristisch für die luetische Acusticuserkrankung, dass

[1] O. Beck, *Zeitschrift für Neurologie*, 1911; *Monatschrift f. Ohrenheilkunde*, 1912.
[2] Bürkner-Schwarze, *Handbuch der Ohrenheilkunde*, 1892.
[3] O. Mayer, *Wiener klinische Wochenschrift*, 1911.
[4] Heubner, *Path. Anat. des Ohres*, 1892.
[5] Gerber, *Syphilis des Ohres*, etc., 1910.

der R. cochlearis und vestibularis nicht immer gleichzeitig erkranken. Die primären Erkrankungen treffen mehr den R. cochlearis ; diese sind auch meistens bilateral. Die sekundären Erkrankungen verteilen sich gleichmässig auf beide Zweige. Isolierte Vestibularerkrankung ist im sekundären Stadium sehr selten (*Bárány, Bondy und Neumann beschrieben* 1–1 *Fälle*).

Die häufigere Erkrankung des R. cochlearis erklärt Wittmaack[1] damit, dass, während die anderen Nerven durch gefässreiche Bindegewebe ziehen, der R. cochlearis in einem stark fibrösen, beziehungsweise knöchernen Gewebe liegt, welches selber nicht nur gefässarm ist, sondern wo auch diese wenigen Gefässe selbst zuerst erkranken. Hier tritt bei Fortschritt des Prozesses eine vollständige *Cochleitis* auf ; dann, infolge von Knochenbildung periostale und osteale Verdickungen oder aber es treten die regressiven Bildungen in den Vordergrund, wodurch die Wand der Schnecke teilweise oder ganz zugrunde geht.

Bei aquirierter Lues tritt nach früherer Annahme (Moos) die Acusticusaffektion nur in tertiärem Stadium auf ; neuerdings wurden aber immer mehr Fälle vom Anfange des sekundären Stadiums publiziert. So trat bei 6 Fällen Habermanns die Acusticusaffektion 3–10 Wochen nach dem Erscheinen der Roseola auf. *In meiner Kasuistik sind* 4 *Fälle wo die Acusticuserkrankung am Anfange des sekundären Stadiums beim Erscheinen der Roseola auftrat* (*Fall No. I–IV*).

In der Literatur sind auch Fälle publiziert, in welchen die Acusticuserkrankung schon vor dem Exanthem in der 3. Woche der Infektion auftrat (O. Mayer, Wechselmann), Politzer diagnostizierte sogar einen am 7. Tage der primären Infektion. Bei hereditären Lues kann die latente Krankheit vom Anfange des Fötallebens bis auch zu 25 Jahren später jederzeit auftreten, sowohl in den Hirnhäuten, wie auch im inneren Ohre ; im letzteren Falle nach abgelaufenen infektiösen oder katarrhalischen Erkrankungen. Neuerdings tritt sogar die Auffassung auf, dass auch in Fällen von aquirierter Lues die Acusticuserkrankung nur dann manifest wird, wenn vorhergehende Erkrankungen des Mittelohres oder des Labyrinthes das Terrain vorbereiteten.

Die Symptome sind je nach der Stelle der Erkrankung verschieden. Wie ich schon erwähnte, kann die Krankheit uni- oder bilateral sein, im letzteren Falle wieder die *Intensität* auf beiden Seiten so verschieden, dass wir z. B. an der linken Seite nur eine Schwerhörigkeit minderen Grades finden, während an der anderen schon vollständige Taubheit eingetreten ist.

Die Acusticuserkrankungen bei kongenitaler und bei aquirierter Lues sind in Betreff *der Schnelligkeit und der Intensität des Prozesses* verschieden. Die Hauptklagen sind bei beiden : subjektive Geräusche, Hyperästhesia acustica, fortschreitende Schwerhörigkeit, Gleichgewichtsstörung und Schmerzen der Occipitalgegend. Die von Roosa und Freitag als charakteristisch beschriebene *Diplacusis* beobachtete ich selber in einem Falle,

[1] Wittmaack, *Zeitschrift f. Ohrenheilkunde*, 1892.

No. XXVIII. Bei *aquirierter Lues* tritt die Schwerhörigkeit oft momentan auf, und verstärkt sich schnell. Bei hereditärer Lues beginnt hingegen die Schwerhörigkeit meist erst im 6.–10. Lebensjahre und die Taubheit tritt langsam ein ; manchmal jedoch, besonders bei Kindern, als *Typus apoplectiformis* sehr schnell, so dass der Kranke manchmal in einer Nacht vollständig taub wird (Urbantschitsch). Als Begleiterscheinung der hereditären Acusticuserkrankung können wir katarrhalische oder eitrige Mittelohrentzündungen finden ; auch Augen- oder Zahnleiden (*Hutchinson'schen Symptom*). Infolge *der Lähmung des R. cochlearis* verengt sich die Skala der hohen Töne, die Dauer der Knochenleitung wird kürzer (Schwabach), Rinne +, Weber auf die gesunde Seite lateralisiert. Bei totaler Erkrankung der Schnecke wird die Taubheit vollständig ; bei partieller können einige Töne zurückbleiben, aber niemals die hohen.

Die Lähmung des R. vestibularis verursacht Gleichgewichtsstörung, die aber später paralysiert wird (*latentes Stadium des Vestibulardefektes*) ; die Gleichgewichtsstörung kann jedoch durch Gehirnlues oder durch andere Erkrankungen der Gehirngefässe verursacht werden ; kann der Schwindel vollständig fehlen sowohl bei Lues des Acusticus, wie auch des Labyrinthes. Ob der Schwindel von der Lues des Gehirnes oder des Labyrinthes herstammt, können wir nicht in allen Fällen beurteilen.[1]

Meiner Ansicht nach sind wir berechtigt, luetische Neuritis acustica anzunehmen, wenn bei Syphilitischen bei scheinbar gesundem Mittelohre schnell progrediierende Gehörabnahme, subjektive Geräusche, Gesichtschmerzen und Gleichgewichtsstörungen auftreten.

Je intensiver diese Symptome sind, um so sicherer ist unsere Diagnose. Die Sicherheit wird erhöht durch die gleichzeitige Erkrankung der übrigen Gehirnnerven, besonders des Opticus, und der übrigen zu seiner Sphäre gehörenden Nerven, wie der Facialis, Oculomotorius, Abducens, Trigeminus (Jansen, Lessner) ; es wird die Beurteilung der intracraniellen Acusticuslähmung erschwert wenn *Labyrinthlues* fehlt. Dies kann verursacht werden durch Gehirntumor, umschriebene Meningitis basilaris, ständigen Druck auf dem Acusticus infolge von Periostitis des Felsenbeines.

Die bei *Tabes* auftretende Schwerhörigkeit ist zentralen Ursprunges, verursacht durch Atrophie des Acusticus. Diese und die *hysterische Schwerhörigkeit* ist manchmal von der durch das Labyrinth verursachte schwer zu unterscheiden.

Nachdem die Acusticus-Lues meistens am Ende des sekundären oder am Anfange des tertiären Stadiums auftritt, wird natürlich die Diagnose erschwert durch mangelhafte Anamnese, wie auch durch die Erkennung der am anderen Teile des Körpers eventuell vorhandenen *spätluetischen Symptome*. Solche späte Ohrsymptome sind die Hyperostose und Exostose des Felsenbeines und Labyrinthes, deren naturgemässe Folge die Verengerung der Labyrinthhöhle ist, solche sind : die Verknöcherung des Labyrinthes, die Stenose des Meatus auditorius internus mit ständigem

[1] Rosenstein, *Archiv f. Ohrenheilkunde*, Bd. lxv.

Druck auf den Acusticus. Erleichtert wird die Diagnose, wenn wir auch bei mangelhaften Anamnese Keratitis, Iritis, Exkavation der oberen Schneidezähne, Schwerhörigkeit, Defekte am Septum, Haut- oder Schleimhautkrankheiten nachweisen können, oder in der Familie Aborte vorhergegangen sind etc. Manchmal vergehen von der Infektion bis zur Acusticuserkrankung 10–25 Jahre, so dass das *späte Acusticusleiden das einzige Symptom der luetischen Infektion bildet.* Bei solchen schwerdiagnostizierbaren Fällen hilft uns die Wassermann'sche Reaktion. Wassermann + zeigt sicher, dass im Organismus latente oder manifeste Syphilis besteht. In Fällen unklarer Aetiologie bewegt uns häufig gerade die positive Reaktion dazu, im Organismus nach Lues zu forschen, um dadurch die latente Lues zu entdecken. *Das zeigt aber die positive Wassermann-Reaktion nicht, ob zwischen der Lues und der Schwerhörigkeit Zusammenhang ist; sie bestätigt nur unsere Annahme, dass wir es mit einem Luetischen zu tun haben, und so auch der Acusticus luetisch erkrankt sein kann.* Es ist eine Tatsache, dass wir mittels der Wassermann'schen Reaktion zwischen den Schwerhörigen immer mehr Luetische finden (Zange), und dass die *Lues eine viel grössere Rolle als ätiologisches Moment bei nervösen Schwerhörigkeit spielt, als wir bisher angenommen haben.*

So wurde die Wassermann'sche Reaktion in unklaren Fällen ein wichtiger Teil der funktionellen Untersuchungsmethoden.

Die Prognose ist durchschnittlich *ungünstig.* Die Virulenz der Infektion und die körperliche Kondition des Individuums scheint den Charakter und die Intensität des Leidens wesentlich zu beeinflussen. Nach Jansen werden die Aussichten auf Erhaltung des Gehörs sehr ungünstig, wenn wir innerhalb 8 Tagen nach Auftreten der Acusticuserkrankung die antiluetische Kur nicht einleiten können; nach ihm progrediiert die Schwerhörigkeit immer mehr, obgleich manchmal auch eine kleine oder grössere Pause eintreten kann und die subjektiven Klagen auf kurze Zeit aufhören. Noch schwerer ist die Erkrankung und deren Prognose im tertiären Stadium. Das ist auch Politzers Ansicht. Auch K. Beck[1] hofft nur in akuten Fällen mittels spezifischer Behandlung Erfolge zu erreichen.

Schwartzes Erfahrungen widersprechen diesen Ansichten. Nach ihm können je nach Verschiedenheit der Fälle auch Veraltete heilen, manchmal bessert sich auch die vollständige Taubheit, anderseits wieder heilt nicht einmal die Schwerhörigkeit minderen Grades. Auch die Besserung der hereditären Taubheit ist nur scheinbar. Voss hält die frühluetischen Acusticuserkrankungen heilbar, er empfiehlt energische und je früher eingeleitete antiluetische Behandlung und hofft sogar bei Erkrankungen im tertiären Stadium eine gewisse Besserung zu erreichen.

Im Punkte der Therapie gibt es zwei durchaus entgegengesetzte Ansichten. Beide stehen auf derselben prinzipiellen Basis, nämlich, dass bei allgemeiner Lues die Quecksilber und Jodbehandlung so lange zu forcieren ist, bis die Spirochaeten vollständig zugrunde gehen, d. h. bis von positiver Wassermann eine negative wird.

[1] *Archiv f. Ohrenheilkunde,* 1912.

Medikamentöse Behandlung ist auch dann einzuleiten, wenn die Zeichen der Nervendegeneration schon zu erkennen sind, um zu versuchen, wenn auch nur ein Minimum vom Gehör zu retten. Besonders bei acquirierter Lues ist diese Ansicht berechtigt, wo im sekundären Stadium, manchmal besonders bei nur einseitiger Schwerhörigkeit — auch im tertiären Stadium Besserung zu erwarten ist.

Politzer empfiehlt bei luetischer Schwerhörigkeit Pilocarpin, während die übrigen Autoren dieses Mittel nur in chronischen Fällen verwenden. Ist das Pilocarpin wirkungslos, gebraucht auch Politzer Quecksilber und Jod.

Ueber den Erfolg der *Salvarsanbehandlung* sind wir noch nicht im Klaren.

Im Anfange wurde Salvarsan bei allen luetischen Labyrinth-Erkrankungen als indiziert betrachtet, später wähnten Finger, Urbantschitsch, Alexander, Pogány[1] und andere nicht nur spirillotrope, sondern *neurotrope* Wirkung des Mittels zu beobachten, gegen welche besonders der Acusticus empfindlich schien. Krankheitsfälle bewiesen, dass nach Salvarsanbehandlung nicht nur an der Haut, sondern auch im inneren Ohre entzündliche Reaktionen auftreten können, seröse Infiltration der Gewebe, welche auf dem im engen Knochenkanal verlaufenden Acusticus Druck ausüben und so dem Herxheimer'schen Symptom analoge Reaktion hervorrufen. Nach Ehrlich[2] ist aber der erkrankte Nerv in diesen Fällen nur im latenten Stadium. Diese temporäre entzündliche Reaktion kann sich nach O. Beck[3] derart steigern, dass der N. acusticus seine Funktion zeitweise vollständig einstellt ; wenn aber auch der funktionelle Defekt der Nerven mindergradig ist, vergeht er langsamer als z. B. das Oedem der Haut. Die Prognose dieser Neuritiden ist günstig. Auch Frey meint, dass das Salvarsan noch niemals eine beständige Verschlechterung am Acusticus verursachte. Alexander[4] empfieht in akuten Fällen kein Salvarsan ; besonders dann nicht, wenn schon vorher ein anderes Ohrenleiden bestand. Er verordnet das Salvarsan in inveterierten Fällen, auch in hereditären, hauptsächlich in mit Schwindel labyrinthären Ursprunges kombinierten. O. Beck hält das Salvarsan kontraindiziert in allen Fällen wo die Acusticuserkrankung das einzige Symptom ist. Der richtige Mittelweg ist unbedingt der, das Salvarsan in grösseren Dosen mit Quecksilber und Jod kombiniert zu verordnen, wie das Perkins, Fabry[5], Arzt[6], und Voss empfehlen.

Nach meiner Kasuistik verwendete ich das Salvarsan nur in 6 Fällen (VI, XV, XVIII, XXI, XXVII und XXXI). Die subjektiven Geräusche wurden bei allen gebessert, beziehungsweise geheilt ; das Gehör verbesserte sich jedoch nur in Fall No. XVI ; in allen übrigen verschlechterte es sich früher oder später. Im Falle No. XXVII zeigte sich Anfangs Besserung, nach 2 Wochen trat aber schnell vollständige Taubheit ein.

[1] Pogány, *Gyogyaszat*, 1911.
[2] Ehrlich, *Berl. klin. Woch.*, 1910.
[3] Beck, *Archiv f. Ohrenheilkunde*, 1912.
[4] Alexander, *Heilkunde*, 1911.
[5] Fabry, *Zeitschr. f. Neurologie*, 1912.
[6] Arzt, *Archiv f. Ohrenheilkunde*, 1910.

Mit Erfolg verwendete ich aber im Gegenteile die anderen Antiluetica, hauptsächlich wenn die Intensität der Erkrankung auf beiden Ohren verschieden war. In den Fällen XVI, XXIII, XXIV, XXV, XXVII, XXXIV meiner Kasuistik sahen wir auf Quecksilber und Jodbehandlung Besserung auftreten. Pilocarpin-Medikation war nur im Falle No. XXXII von Erfolg.

Endresultate. — 1. Es scheint, dass Acusticusaffektionen auch im sekundären Stadium der Lues häufiger sind, als bisher angenommen wurde.

2. Es ist noch nicht entschieden, ob diese Acusticusaffektion luetischen Ursprunges ist, *sicherlich* kommt sie bei leutischen Individuen ziemlich häufig vor.

3. Die medikamentöse Behandlung ist nicht nur im sekundären, sondern auch im tertiären Stadium indiziert, besonders wenn der Grad der Schwerhörigkeit auf beiden Ohren ungleich ist. Merkur und Jod kommen hier hauptsächlich in Betracht.

4. Salvarsan können wir anwenden bei veralteten Acusticusläh mungen, wenn Lues vorhanden ist, besonders wenn der N. vestibularis auch erkrankt ist.

5. Salvarsan ist in grösseren Dosen zu verabreichen und mit Merkur und Jod zu kombinieren.

6. In allen von mir beobachteten Fällen blieben die subjektiven Symptome nach Salvarsanbehandlung aus ; das Gehör verbesserte sich jedoch nur in einem Falle.

Kasuistik

Vorliegende Studie wurde mit Beachtung von 160 Fällen bereitet, welche *Acusticusaffektionen* ich von Januar 1911 bis Juni 1913, also beinahe $2\frac{1}{2}$ Jahre teilweise selber auf meiner Spitalsabteilung sammelte (106 Fälle), teilweise (54 Fälle) von den Ohrenärzten Herren Dr. Rejtö und Dr. Pogány liebenswürdigst überlassen wurden.

Nach unseren Uebereinkommen diagnostizierten wir Acusticuserkrankung nur wenn (1) das Trommelfell vollständig oder beinahe gesund war, (2) die Knochenleitung verkürzt, (3) Rinne positiv, (4) C_4 verkürzt, (5) der Kranke die Flüsterstimme von weniger als 5 Meter hörte. Andererseits schliessen wir aus der Statistik Intoxikationen, traumatische Fälle, die Berufsschwerhörige und die scheinbar infolge von Infektionskrankheiten auftretenden Fälle. Leugnete der Kranke Lues oder die Anamnesis war unsicher, wendeten wir immer Wassermann'sche Reaktion an, wenn die Gehörsuntersuchung Acusticuserkrankung zeigte ; aber auch, wenn die vom Kranken eingestandene Lues nicht manifest war. Die Mehrzahl der Fälle gehörte natürlich ins Tertiärstadium.

I. Frau A. S., 25 Jahre. Vor 6 Wochen Luesinfektion ; starkes Ohrensausen, neuerdings rapid zunehmende Schwerhörigkeit. Gehörsuntersuchung : R. und L. Flüstern 2 m, Konversation auf Zimmerweite,

Rinne +, Weber auf die linke Seite lateralisiert, Knochenleitung und C_4 verkürzt. Wassermann + + +. Fall steht unter Behandlung.

II. Frau I. T., 32 Jahre. Seit 2 Wochen manifeste Lues. Wassermann + +. Gehör: Flüstern R. und L. 1 m, gewöhnliches Sprechen R. 3 m, L. 1 m. Rinne +, Weber unbestimmt, Knochenleitung verkürzt, C_4 sehr verkürzt.

III. H. K., 23 Jahre. Acquirierte vor 8 Wochen Lues. Neuerdings Ohrensausen und rapide Abnahme des Gehörs. Gehör: Flüstern R. und L., Konversation 30 cm, Rinne +, Weber nicht untersuchbar, Knochenleitung und C_4 verkürzt.

IV. Frau H. A., 41 Jahre. Bekam schon 2 Monate nach Aquirierung der Lues wegen hochgradiger Schwerhörigkeit in einem anderen Institute Pilocarpininjektion. Neuerdings rapide Abnahme des Gehörs, Rinne +, Knochenleitung und C_4 kaum. Steht unter antiluetischer Behandlung.

V. A. Cs., 49 Jahre. Hört seit einem Jahre auf beiden Ohren schlecht. Lues vor 21 Jahren. Linkerseits Trommelfell wenig getrübt. Hört Uhr auf 10 cm, Flüstern ad Concham, Konversation 2 m, Rinne +, Weber unbestimmt, Knochenleitung kaum, C_4 kurz. Rechtes Ohr beinahe normal. Wassermann + +.

VI. Herr T. K., 24 Jahre. Seit 23 Jahren beiderseitiges Ohrensausen und Schwerhörigkeit; neuerdings Schwindel, Erbrechen, Kopfschmerz und spontane Nystagmus.

Rechts und links: Flüstern 10 cm., Konversation 2 m, Rinne +, Weber nach rechts, Schwabach verkürzt; hohe und tiefe Töne verkürzt. Wassermann + + +. Auf Salvarsan keine Besserung, sogar sowohl beim Gehör, wie bei den subjektiven Klagen, Verschlechterung.

VII. Frl. I. T., 21 Jahre. Hört L. seit 2 Jahren schlecht; zunehmende subjektive Geräusche. Lues nicht nachweisbar. Trommelfell normal. Gehör: Flüstern 10 cm. Konversation 1 m, Rinne +, Weber unbestimmt, Knochenleitung und C_4 verkürzt. Wassermann −. Therapie: Jodbehandlung.

VIII. K. M., Ingenieur, 47 Jahre. Im Kindesalter beiderseitige Ohreneiterung; jetzt Narben am Trommelfell; hört seit einem Jahre am linken Ohr sehr schwer; hochgradiger Schwindel, welcher periodisch auftritt, starkes subjektives Geräusch. Gehör: L. Flüstern 0, Konversation 50 cm, Rinne +, Knochenleitung und C_4 verkürzt. R. Flüstern 50 cm, Konversation 1 m, Rinne +, Weber lateralisiert, Knochenleitung ziemlich lang, C_4 verkürzt. Wassermann −. Therapie: Jod und Pilocarpin.

IX. Frl. L. G., 20 Jahre, seit 1 Jahr Lues. Gehör: auf beiden Seiten Flüstern 0, Rinne −, Knochenleitung und C_4 verkürzt, Weber unsicher. Wassermann + +.

X. Frl. H. E., 36 Jahre. Gehör auf beiden Seiten herabgesetzt, Periostitis luetica ossium nasi. Wassermann + + +.

XI. Frl. I. F., 36 Jahre. Flüstern auf beiden Seiten 25 cm, Rinne +, Knochenleitung und C_4 verkürzt. Im rechten äusseren Gehörgange 2 Gummata. Wassermann + +.

XII. H. H., 34 Jahre. Gehör beiderseitig herabgesetzt, rechts starkes Ohrengeräusch. Wassermann + +. Therapie: Pilocarpin wirkungslos, bessert nur die subjektiven Geräusche.

XIII. Frl. I. Sz., 24 Jahre. Subjektive Geräusche. Gehör: Flüstern rechts 50 cm, links 10 cm. Wassermann + + +.

XIV. H. S., 56 Jahre. Flüstern rechts 1 m, links 25 cm, Rinne +, Knochenleitung und C_4 verkürzt. Wassermann + +.

XV. Frl. I. I., 40 Jahre. Starke subjektive Geräusche, beiderseitige Schwerhörigkeit, Knochenleitung und C_4 kurz, Rinne +. Wassermann + + +. Therapie: auf Salvarsan hören die subjektiven Symptome vollständig auf. Gehör bessert sich ein wenig.

XVI. Herr K. L., 40 Jahre. Starke subjektive Geräusche. Einseitiges Schwerhören. Anchylosis stapedis. Calcificatio m. tympani. Wassermann +.

XVII. Herr K. M., 26 Jahre. Wassermann + + +. Rinne +, Knochenleitung und C_4 verkürzt. Steht unter Behandlung.

XVIII. Herr L. M., 30 Jahre. Vor 5 Jahren Lues. Flüstern: rechts und links 0, Knochenleitung und C_4 sehr kurz, Rinne +. Wassermann + +. Therapie: auf Salvarsan Abnahme der subjektiven Geräusche.

XIX. Herr M. S., 41 Jahre. Vor 15 Jahren Lues. Seit 10 Jahren subjektive Geräusche und herabgesetztes Gehör in beiden Ohren. Rinne +, Knochenleitung und C_4 sehr kurz. Wassermann +. Merkurbehandlung.

XX. Herr O. L., 26 Jahre. Seit 3 Jahren taub und leidet seitdem oft an Schwindel. Haemeralopie. R. Pupille weit, L. verengt. Lichtgefühl besteht. Ohrenspiegeluntersuchung negativ. Gehör: R. und L. Flüstern und Konversation 0, Rinne −, Weber unsicher, Knochenleitung und C_4 0. Rechts horizontal-rotatorischer Nystagmus. L. horizontaler Nystagmus, der sich auf calorische Reaktion kaum verstärkt. Wassermann +. Therapie: auf Pilocarpin-Injektionen keine Besserung.

XXI. Herr B. N., 28 Jahre. Vor 1 Jahre Luesinfektion. Erhielt dann Salvarsan, wonach er bemerkte, dass er L. schlechter hört. Inunktionskur. Gehör: Flüstern auf $2\frac{1}{2}$ m, Weber L., Rinne rechts −, links +, Knochenleitung und C_4 verkürzt.

XXII. Frl. N. N., 26 Jahre. Vor 3 Jahren Lues; gegenwärtig Gumma im äusseren Gehörgange. Hört seit Jahren auf beiden Ohren schlecht, was auffallend zunimmt. Gehör: Flüstern auf beiden Seiten 50 cm, Konversation 2 m, Rinne +, Weber unbestimmt, Knochenleitung und C_4 sehr kurz. Wassermann + +. Therapie: Merkurbehandlung. Gehör bessert sich, Gumma auch; steht unter Behandlung.

XXIII. Herr A. V., 30 Jahre. Voriges Jahr Luesinfektion. Starkes Ohrensausen und Herabsetzung des Gehöres. Gehör: R. und L. Flüstern ad Concham, Rinne +, Knochenleitung und C_4 sehr kurz. Auf Quecksilber-Behandlung Besserung.

XXIV. Herr N. J., 29 Jahre. Vor 2 Jahren Lues. Starkes Ohrensausen und Herabsetzung des Gehöres. Gehör: R. und L. Flüstern ad Concham, Rinne +, Knochenleitung und C_4 sehr kurz. Wassermann + + +. Therapie: Quecksilber-Behandlung; Ohrensausen nimmt ab, teilweise bessert sich das Gehör.

XXV. Frl. I. S., 44 Jahre. Seit 4 Jahren progressives Verschlechtern des Gehöres. Ohrengeräusch und Schwindel. Gehör: R. und L. Flüstern 0, Konversation $\frac{1}{2}$ m, Rinne +, Knochenleitung und C_4 sehr kurz. Wassermann + +. Therapie: steht unter Quecksilber-Behandlung. Bisher Besserung des Gehöres und der subjektiven Symptome.

XXVI. Frau F. W., 34 Jahre. Vor 6 Jahren Lues, progressive Schwerhörigkeit. Gehör: R. und L. Flüstern 1 m, Konversation 2 m. Rinne +, Knochenleitung und C_4 sehr kurz. Wassermann + +.

Therapie: auf Quecksilber-Behandlung wenig Besserung; auch Pilocarpin bessert nicht.

XXVII. Herr S. V., 39 Jahre. Im Herbst 1911 wegen allgemeiner Lues und bilateraler Acusticusaffektion Salvarsan; 2 Wochen später besserte sich das Gehör, wurde danach schnell total taub.

XXVIII. Herr I. L., 36 Jahre. Rechts Diplacusis. Wassermann + +. Gehör sehr herabgesetzt.

XXIX. Herr L. K., 32 Jahre. Seit Jahren bilaterales progredierendes Schwerhören. Tabes. Wassermann + +. Pilocarpin-Injektionen ohne Erfolg.

XXX. A. P., 41 Jahre. Im März 1912 Lues acquiriert. Steht seit Mai d. J. unter ohrenärztlicher Behandlung; er erhielt Pilocarpin-Injektionen. Gehör verschlechtert sich progredierend und schnell. Rinne +, Knochenleitung und C_4 kaum. Neuerdings versuchen wir Quecksilber, Erfolg noch unbestimmt.

XXXI. Frl. R. F., 27 Jahre. Vor Jahren Lues, Gehör herabgesetzt. Ende Dez. 1910 Salvarsan. 4 Monate später beiderseitig auffallend schnelle Abnahme des Gehörs. (Flüstern 0, Rinne +, Knochenleitung und C_4 sehr kurz.)

XXXII. Herr J. K., 36 Jahre. Diagnosis: Effectio N. acustici l. untr. Flüstern: R. 20 cm, L. ad Concham. Wassermann + +. Antiluetische Behandlung erfolglos. Auf Pilocarpin Flüstern R. 6 m, L. 1 m.

XXXIII. A. K., Schlosser, 30 Jahre. Vor 2 Jahren Luesinfektion, nachdem nimmt das Gehör immer mehr ab. Gehör: R. und L. Flüstern 20 cm, Konversation 5 m, Weber nach L., Rinne +, Knochenleitung und C_4 sehr kurz. Wassermann +. Auf antiluetische Kur bedeutende Besserung.

XXXIV. Herr F. M., 28 Jahre. Vor 4 Jahren Luesinfektion. Gehör: R. und L. Flüstern 20 cm, Konversation 2 m, Weber unbestimmt, Rinne R. +, L. −, Knochenleitung und C_4 sehr kurz. Auf antiluetische Behandlung Besserung.

Meine Statistik enthält auch noch 126 hier nicht detaillierte Fälle: 80 Männer und 48 Frauen. *Endresultat*: von 160 Acusticusaffektionen 63 mal Wassermann +, d. i. Lues in 39,3% der Fälle *bewiesen; das bedeutet aber nicht, dass alle diese Acusticusaffektionen luetischer Natur waren.*

DISCUSSION

Professor MYGIND (Copenhagen). There are only two points I wish to say a few words about. The first is that, according to my experience, the deafness caused by hereditary syphilis is generally not absolute, though as a rule very considerable. The other is that hereditary syphilis does not, according to my experience, play any important part as a cause of ' nervous ' deafness.

SECTION XVI

OTOLOGY

L'ACOUMÉTRIE MILLIMÉTRIQUE ET SES AVANTAGES EN PRATIQUE COURANTE

PAR M. LE DR. TRÉTRÔP, D'ANVERS (BELGIQUE)

LE but de cette communication n'est pas d'établir que l'acoumétrie millimétrique est scientifiquement supérieure aux diverses méthodes qui ont été proposées et plus ou moins suivies, mais bien de faire voir une fois de plus la valeur pratique de ce mode de mensuration de l'ouïe et l'aide efficace qu'elle apporte journellement au spécialiste pour apprécier le degré de surdité d'un malade. La feuille acoumétrique dressée lisiblement aussi bien pour le malade que pour le médecin permet de comparer les valeurs trouvées à celles d'une ouïe moyenne normale, d'apprécier à intervalles déterminés le gain obtenu par le traitement, de le faire constater au malade lui-même et d'obtenir ainsi à la fois les indications sur la voie à suivre et l'encouragement nécessaire tant pour le malade que pour le médecin à persister dans l'application d'un traitement souvent long et fastidieux pour les deux parties en cause.

Il y aura bientôt treize ans que j'emploie journellement ou presque journellement cet appareil.

L'acoumètre millimétrique fut en effet présenté par moi à la Société de Médecine d'Anvers en décembre 1901 et en juin suivant à la Société belge d'oto-rhino-laryngologie à Bruxelles.[1]

Vous rappellerai-je en deux mots le principe de cet appareil. D'une part, des sons transmis par deux microphones, d'autre part des téléphones récepteurs dont l'intensité est graduée par une bobine d'induction à chariot. La graduation de la bobine donne en millimètres la valeur auditive de chacune des oreilles d'un sujet donné.

Pour la description détaillée, je ne puis mieux faire que de prier les intéressés de la lire dans ce remarquable traité de technique oto-rhino-laryngologique, 2e édition, de notre savant et sympathique collègue le Dr Escat, de Toulouse.[2]

[1] Dr. Trétrôp, 'Table électrique munie d'un acoumètre millimétrique' (*Ann. de la Soc. de Méd. d'Anvers*, décembre 1901. *Soc. belge d'oto-rhino-laryng.*, juin 1902).

[2] Dr. Escat, *Technique oto-rhino-laryngologique*, Deuxième édition, p. 200, Paris. Maloine, 1911.

Les sons utilisés dans l'acoumètre millimétrique sont celui de la montre, du diapason, de la musique et de la voix humaine, c'est-à-dire l'échelle de sons nécessaire pour entendre à peu près tout ce qu'il faut dans la vie normale.

Le diapason est la base essentielle de cette acoumétrie. Plusieurs diapasons peuvent y être utilisés, mais en pratique un seul donnant le la suffit. Les autres sons viennent en ordre subsidiaire, bien qu'ils soient aussi très utiles.

C'est dans toutes les acoumétries que j'ai pratiquées la perception du diapason qui disparaît la dernière. Il n'est pas rare au cours du traitement d'une surdité considérable, n'ayant donné des millimètres de perception que pour le diapason seul, les autres sons n'étant pas perçus au début, de voir réapparaître la perception, faible d'abord, plus prononcée ensuite, de la voix humaine, de la musique et de la montre. Parfois, il subsiste des lacunes, l'un ou l'autre son n'étant point perçu. Mais à une perception acoumétrique plus variée ou plus intense, correspond toujours une amélioration de l'ouïe dans la vie sociale.

Les valeurs millimétriques trouvées à un premier examen sont inscrites dans la première colonne de la feuille acoumétrique ; les colonnes suivantes servent aux mensurations effectuées tous les mois ou toutes les six semaines au cours d'un traitement et aux mesures prises à intervalles irréguliers en cas de soins interrompus. En note, on inscrit les valeurs normales.

Un seul coup d'œil sur la feuille acoumétrique permet de juger à la fois de la valeur auditive de chacune des oreilles par rapport à l'ouïe normale et des progrès réalisés depuis le début du traitement ou des pertes subies depuis que tout traitement avait cessé. La même feuille permet en cas de doute de savoir si une ouïe mesurée antérieurement a perdu ou est restée stationnaire.

Une acoumétrie millimétrique — et ceci a son importance pratique — réclame 10 à 15 minutes tout au plus. Et qu'on n'objecte pas qu'il faut le silence absolu et l'éloignement de tout bruit pour y recourir. On peut faire de l'acoumétrie millimétrique, tout comme de la téléphonie, dans les endroits les plus bruyants moyennant certains dispositifs courants. Il suffit notamment de garnir les téléphones des bourrelets pneumatiques en caoutchouc usités en téléphonie.

L'acoumétrie millimétrique ne remplace pas les épreuves courantes de diagnostic : Rinné, Weber, Schwabach, etc., celles-ci doivent toujours nécessairement être faites.

En pratique, l'acoumétrie millimétrique dans toute sa simplicité nous dit *immédiatement sans renseignements du malade*, quelle est l'oreille qui entend le moins et quelle est la valeur de l'ouïe par rapport à l'ouïe normale. N'est ce pas suffisant, après avoir relevé soigneusement les commémoratifs, examiné le conduit auditif, le nez et le naso-pharynx pour avoir rapidement de bons éléments de diagnostic et pouvoir commencer le traitement ? Si l'on veut recourir à des procédés plus précis, sans être pour

cela toujours pratiquement meilleurs, on peut toujours le faire ultérieurement.

*Valeur millimétrique de l'acuité auditive de M*_____

mesurée à l'acoumètre spécial du docteur Trétrôp.

Date :					Observations
Oreille droite :					
Montre . . .					
Diapason . .					
Musique . .					
Voix humaine					
Oreille gauche :					
Montre . . .					
Diapason . .					
Musique . .					
Voix humaine					

Conductibilité cranio-tympanique { oreille droite _____ / oreille gauche _____

Épreuve de Rinné { oreille droite _____ / oreille gauche _____

Épreuve de Weber { oreille gauche _____ / oreille droite _____

Je puis produire de très nombreuses feuilles acoumétriques et j'en ai publié un certain nombre[1] démontrant que dans la pratique courante l'acoumétrie millimétrique fournit une aide précieuse d'appréciation pour le malade et pour le médecin, suffisamment exacte pour servir de base à la bonne direction du traitement.

Le premier modèle d'acoumètre millimétrique fut construit par moi et depuis j'ai mis entièrement et gracieusement à la disposition des constructeurs toutes les données pour reproduire mon appareil, n'ayant

[1] Dr. Trétrôp, ' Surdité datant de dix ans ramenée à une audition moyenne par le procédé de Delstanche,' *Soc. de méd. d'Anvers*, mars 1902 ; ' Du traitement de la surdité par la méthode de Babinsky,' *Ann. de la Soc. de méd d'Anvers*, mai 1904 ; ' De la chirurgie conservatrice de l'ouïe ' (ibid.) ; ' Résultats personnels du traitement des vertiges, des bourdonnements et de la surdité par la méthode de Babinsky,' *Congrès ann. de la Soc. belge d'oto-rhino-laryng.*, Bruxelles, 1904, et *Ann. des malad. de l'oreille et du larynx*, Paris, décembre 1904 ; ' Essai d'acoumétrie métrique,' *VII^e Congrès international d'otologie*, Bordeaux, 1904 ; ' De l'acoumétrie millimétrique,' *Soc. franç. d'oto-rhino-laryng.*, Paris, mai 1907, etc. etc.

qu'un but : que mes collègues puissent utiliser dans leur pratique courante, avec le minimum de dépenses — ceux qui savent faire du travail d'atelier peuvent le construire eux-mêmes—un appareil de mesure de l'ouïe honnête, simple, fonctionnant régulièrement des mois et des années sans surveillance et des plus pratiques.

Cet appareil, après une expérience très étendue de treize années, je puis, mieux qu'aux premiers jours, le recommander à mes estimés collègues en toute confiance.

SECTION XVI

OTOLOGY

DISCUSSION No. 5

(Jointly with Section XV, Rhinology and Laryngology, Discussion No. 4)

THE SPECIAL TREATMENT OF THE THROAT, NOSE AND EAR DURING THE ACTIVE STAGES OF CERTAIN INFECTIOUS FEVERS, NAMELY—SCARLET FEVER, MEASLES, GERMAN MEASLES, MUMPS, INFLUENZA, TYPHOID FEVER, WHOOPING-COUGH, SMALL-POX, CHICKEN-POX, ERYSIPELAS, POLIOMYELITIS AND CEREBRO-SPINAL MENINGITIS (DIPHTHERIA EXCLUDED)

(For Report by E. W. Goodall, M.D., see Part I, pp. 135–47)

Rapport sur le Traitement des Oto-Rhino-Laryngopathies au cours des Maladies infectieuses et des Fièvres éruptives

Par M. le Dr. V. DELSAUX, de Bruxelles

Lorsque nous avons à nous occuper des soins de l'oreille, du nez ou de la gorge concomitants ou consécutifs aux maladies infectieuses ou aux fièvres éruptives, nous sommes frappés par le manque presque absolu de mesures préventives ou curatrices dirigées contre les complications buccales, otitiques, laryngées ou nasales, pourtant si terribles parfois, de ces maladies.

Les traités de médecine générale sont à peu près muets à cet égard, et nos livres de spécialités, nos journaux, s'ils relatent de très nombreux faits de ce genre, renferment bien peu de données prophylactiques.

Il est donc rationnel et urgent de condenser en quelques pages les principales indications du traitement préventif et curatif des affections auriculaires nasales, laryngées, etc., qui sont symptômes ou complications de fièvres éruptives et des maladies infectieuses.

Sans doute, il serait désirable de faire de chacune des maladies en question un chapitre spécial, de localiser dans un casier particulier les indications de leurs diverses périodes ; mais je ne pense pas qu'en posant la question comme il l'a fait, le Comité organisateur des XV^e^ et XVI^e^ sections du Congrès international de Médecine ait eu l'intention de mettre d'un seul coup la chose au point dans tous ses détails.

C'est donc une revue d'ensemble que je vais présenter à mes confrères, leur demandant de compléter, par leurs remarques personnelles, les grandes lignes de mon rapport. De leur coopération naîtra, je pense, une solution meilleure et plus complète que celle résultant d'une impression personnelle.

Qu'il me soit permis de faire remarquer tout d'abord qu'actuellement on ne peut plus mettre en doute la contagiosité pour aucune des maladies que nous envisageons ici. Qu'elle soit directe ou médiate, qu'elle se fasse par l'air inspiré, par les aliments ou par contact, la contagion est nettement établie pour la grippe, l'érysipèle, les fièvres éruptives, la méningite cérébro-spinale, etc. De là découlent des mesures prophylactiques d'ordre général et mesures d'hygiène individuelle.

I. — Mesures d'Hygiène générale

Il faut nécessairement isoler tout malade atteint de l'une quelconque des maladies en question. En réalisant cet isolement, on s'efforcera autant que possible de le rendre individuel, c'est-à-dire que chaque malade possède un local particulier, mais avec un service de soignage commun à plusieurs malades, puisqu'il serait difficile de faire autrement. Les infirmières seront stylées méticuleusement au point de vue de la contagiosité.

On conçoit, en effet, que les agents spécifiques ou infectieux varient de virulence d'un individu à un autre, du début d'une maladie à sa période d'état ou à celle de déclin, etc.

Autant que faire se pourra, on veillera à ce que l'atmosphère des chambres de malades soit elle-même purifiée par l'installation de moyens de ventilation et de chauffage en rapport avec la science moderne. Pourquoi ne ferait-on pas pour les salles de malades ce qu'actuellement on réalise pour les bureaux des compagnies d'assurances, pour les banques, pour certains locaux scolaires même ? Ne pourrait-on y faire circuler un courant d'ozone qui aurait l'immense avantage de tuer bien des germes, d'atténuer tout au moins leur virulence au lieu de les diffuser au dehors avec toute leur activité néfaste ?

Ce qui vient d'être exposé comme un desideratum au point de vue de l'air respirable doit être complété par une propreté minutieuse des objets de couchage, du matériel d'alimentation, de celui de soignage proprement dit, du mobilier, etc. La désinfection par l'étuve à formol et vapeur d'eau sous pression semble constituer le meilleur moyen de stérilisation pour les mêmes objets qui servent à soigner le malade.

Pour la désinfection des objets de couchage, cette même méthode est pratiquement applicable. Il doit être entendu que le local, le mobilier, les literies seront désinfectés chaque fois qu'un malade doit succéder à un autre.

En ce qui concerne le personnel infirmier, il faut que, bénévolement, il se soumette à des mesures rigoureuses de propreté du corps, des vêtements, des mains, à des soins méticuleux de la bouche, du nez, qu'une fois par jour au moins les infirmières prennent un grand bain, qu'elles changent quotidiennement et complètement de vêtements, etc. C'est la meilleure façon de pratiquer pour elles-mêmes l'hygiène individuelle et d'éviter la contagion de malade à malade par l'intermédiaire du personnel qui leur donne des soins.

II. — Mesures d'Hygiène spéciale

Tout malade reconnu atteint de l'une quelconque des maladies qui nous occupent doit être l'objet de soins attentifs et répétés de la bouche, de la gorge, du nez et, s'il y a lieu, des oreilles et des voies respiratoires supérieures.

1° *Soins de la bouche.* — On fera immédiatement panser les dents malades, autant qu'il sera possible ; on fera brosser les dents plusieurs fois par jour à l'aide d'une brosse douce et d'une solution boro-oxygénée ; on prescrira au malade de se rincer la bouche et de se gargariser fréquemment, par exemple, avec la solution suivante :

Solution boriquée à 40 °/₀₀, 800 centimètres cubes.
Eau oxygénée, 200 centimètres cubes.
Alcoolat de menthe, 5 grammes.
Alcoolat de cochléaria, 5 grammes.

Si l'on a affaire aux angines du début de la scarlatine, on redoublera de vigilance ; on touchera les ulcérations soit à la solution de chlorure ou de sulfate de zinc à 1 pour 30, soit à l'aide de l'une des solutions suivantes :

Camphre et acide phénique āā 50 centigrammes.
Glycérine et alcool à 80° āā 10 grammes.

Ou bien :

Iode métallique, 1 gramme.
Iodure de potassium, 4 grammes.
Glycérine, 30 grammes.
Huile essentielle de menthe, 10 gouttes.

Après ces attouchements, on fera faire des lavages de la bouche à la solution boriquée et oxygénée et, si le malade est en état d'adynamie, on pratiquera ces lavages à la seringue, la tête étant penchée en avant au-dessus d'un récipient approprié.

Il est de pratique courante également de toucher les ulcères au bleu de méthylène. Mais cette manière de faire, outre qu'elle n'est pas toujours

fidèle, présente l'inconvénient d'être d'un maniement difficile et de tacher les objets de toilette, les couvertures et draps de lit, etc.

Au moment où la spirillose buccale vient d'être rapprochée de celle de l'angine de Vincent et même des spirochètes de la syphilis, il semble que l'on doive attendre de bons effets de l'attouchement des ulcères amygdaliens avec le salvarsan ou le néo-salvarsan en nature. Toutefois, il faudra surveiller les doses.

Enfin, il n'est pas sans intérêt de mettre en parallèle avec ce dernier moyen la thérapeutique buccale par les ferments et notamment par le ferment lactique. Cet agent est, on le sait, souverain dans certaines formes de pyorrhée alvéolaire.

Les nombreux cas d'angine simple, d'amygdalites folliculaires ou même pultacées que j'ai soignés avec succès de cette façon m'engagent à recommander les insufflations de lactéol en poudre ou de l'un quelconque des ferments similaires, dans les cas d'angines graves ou banales des maladies infectieuses.

Ces ferments, outre qu'ils n'ont pas l'inconvénient des solutions caustiques, soi-disant antisepsiques ou modificatrices, ont le grand avantage, lorsqu'ils sont déglutis par le malade, de prolonger leur action bienfaisante dans le tube digestif. C'est une raison de plus d'en vulgariser l'emploi. Chez les typhiques, notamment, on humectera la *langue* avec un tampon d'ouate monté sur une pince à pansements, imbibé de mélange à parties égales d'eau de Vichy et de glycérine ; puis on raclera la langue prudemment, de la base à la pointe, avec un gratte-langue. On aura soin de nettoyer de la même façon les sillons jugo-gingivaux.

On soignera particulièrement les *lèvres* de tous les patients atteints de maladies infectieuses. Une bonne formule est la suivante :

Baume du Pérou, 1 gramme.
Cire blanche, 1 gramme.
Beurre de cacao, 5 grammes.
Glycérine, q. s. *ad consist.*,

pour un bâton cosmétique que l'on trempera un instant dans l'eau bouillie tiède avant de l'appliquer sur les lèvres.

On examinera chaque jour la bouche des malades ; on se rendra compte de l'état des ganglions du cou ; on surveillera l'évolution des amygdalites et l'on sera prêt à ouvrir les abcès para ou péri-amygdaliens, s'ils se produisent.

Quant aux soins adjuvants, et j'entends par là les enveloppements humides du cou, les pansements à l'ouate sous feuille de gutta-percha, les compresses thermophores, etc., ils procurent aux malades un soulagement réel en cas d'entreprise des ganglions sous-angulo-maxillaires. Qu'il me soit permis d'insister sur le mode d'application de ces pansements.

En général, on se contente d'entourer le cou du malade d'un collier de hauteur appropriée, d'application plus ou moins exacte. Cela est tout

à fait insuffisant et, sinon inutile, du moins fastidieux pour le patient. En effet, au bout de fort peu de temps, ce pansement s'élargit, s'abaisse et tombe au bas du cou.

Ce qu'il faut faire, c'est un pansement humide ou ouaté exact, recouvrant surtout les régions carotidiennes supérieures et sous-angulo-maxillaires. Et pour le réaliser, pour le rendre efficace, il faut de toute nécessité le maintenir à l'aide d'une bande passée au-dessus de la tête, d'une part, sous le menton et autour du cou, de l'autre. Ainsi appliqué, le pansement tient en place, reste humide s'il a été fait de la balnéation et est en tout cas utile, puisqu'il soulage.

Comme liquide destiné à humecter le pansement, je prescris volontiers l'eau glycérinée à 20 pour cent. Elle a l'avantage de ne pas se dessécher aussi rapidement que l'eau pure et ne laisse pas d'enduit pulvérulent comme les solutions boriques, boratées ou alunées, dont l'action topique spéciale est bien aléatoire.

2° *Soins du nez.* — Dans toutes les maladies infectieuses, il est indispensable, voire même urgent, de prescrire des soins du nez. Voyons quels ils peuvent, quels ils doivent être :

Étant donné que la température du corps s'élève considérablement dans la plupart d'entre elles, il se produit de ce fait un dessèchement de la muqueuse des voies aériennes supérieures. Il en résulte soit que des croûtes se forment par accumulation de mucus ou de muco-pus, soit que des ulcérations se produisent par grattage ou par expulsion desdites croûtes.

Pendant la première période des fièvres éruptives, il est encore possible de faire faire aux malades des irrigations nasales ; mais plus tard, lorsqu'ils sont tombés dans l'adynamie ou l'inconscience, cela n'est plus réalisable.

Si l'on prescrit des irrigations nasales, celles-ci seront faites *sans pression aucune*. Ce seront des lavages plutôt que des douches. On emploiera pour ce faire soit le syphon de Weber, soit la pipette nasale de Depierris, soit le bain nasal de Fraenkel, mais non l'irrigateur d'Esmarch, l'enema, la seringue qui sont des appareils dangereux à cause de la pression qu'ils produisent dans les fosses nasales et le cavum.

Nous savons, par trop d'expériences malheureuses, quels sont les méfaits de la douche nasale et des appareils similaires ; nous connaissons, hélas ! les complications sinuso-nasales et auriculaires qui sont leur fait. N'utilisons jamais ces appareils chez les malades infectieux, banissons-les de notre arsenal instrumental.

Mais, me dira-t-on, quel liquide faut-il employer pour les lavages du nez ? Le plus simple, répondrai-je, celui qui nous est indiqué par la thérapeutique naturiste, le sérum artificiel, tiède. On fera passer un quart, un demi-litre, un litre dans les fosses nasales, matin et soir, et l'on aura soin de recommander au malade de ne pas se moucher après cette opération, afin d'éviter de projeter du liquide vers les trompes d'Eustache et les caisses tympaniques.

Les irrigations nasales ne seront qu'un moyen d'exception, car il est préférable d'appliquer à la désinfection du nez ou plutôt à l'entretien de sa perméabilité l'application de pommades, l'instillation d'huiles médicamenteuses, les pulvérisations des mêmes agents.

Ici encore, le médecin se méfiera de la tendance à prescrire des pommades ou solutions dites ' antiseptiques '. J'ignore si l'on a pu se convaincre de la possibilité de faire de fosses nasales septiques des cavités stériles : je n'ai rien trouvé dans la littérature médicale qui pût m'éclairer à cet égard. Mais ce que la pratique de plus de vingt-cinq ans m'a enseigné, c'est que si l'on obtient du malade ou de son entourage que soit appliqués régulièrement plusieurs fois par jour un onguent légèrement antisepsique, des pulvérisations ou des instillations d'huiles médicamenteuses, on voit les fosses nasales se déterger, le pus ou le muco-pus disparaître en quelques jours, pour faire place à de la sécrétion muqueuse, claire, plus ou moins abondante.

Voici ci-après quelques formules qui viendront à point plutôt aux praticiens généraux qu'à nos confrères en spécialité. Elles ont en leur faveur : l'innocuité, la non-accumulation de principes thérapeutiques, leur conservation de longue durée, leur application facile :

Acide borique finement pulvérisé, 2 grammes.
Huile essentielle de menthe Mitcham, 2 gouttes.
Vaseline Cheesebrough, 30 grammes.
(Pour un onguent à mettre en tube d'étain.)

En appliquer gros comme un pois toutes les deux heures ou toutes les heures, à l'entrée de chaque narine ; aspirer fortement par le nez jusqu'à ce que la pommade arrive dans l'arrière-nez. Dès lors, ne pas renâcler pour l'expulser ; la laisser agir là le plus longtemps possible ; ne pas se moucher.

Évidemment quand il s'agit d'un fébricitant ou d'un adynamique, on n'en peut réclamer d'aide et il faut se servir, chez eux la plupart du temps, des instillations ou des pulvérisations :

Résorcine ou aristol, de 25 à 50 centigrammes.
Huile essentielle de menthe ou huile de caryophille, 2 gouttes.
Huile de paraffine, 20 à 30 grammes.

En mettre 5 centimètres cubes dans un pulvérisateur *ad hoc* et faire toutes les heures ou toutes les deux heures des pulvérisations répétées dans les fosses nasales.

La même solution peut s'employer en instillations à l'aide d'un compte-gouttes.

Quand des fissures se produisent à l'entrée des narines, des ulcérations à l'endroit d'élection de la cloison (zone hémorragipare d'Escat), on emploiera avec avantage l'onguent suivant :

Précipté blanc, 25 à 50 centigrammes.
Vaseline jaune, 30 grammes.
Huile d'eucalyptus, 3 gouttes.

En applications quatre à cinq fois par jour à l'entrée des narines, sur les fissures, les raghades, ou en introduire dans la fosse nasale où siège l'épistaxis.

On remarquera que ni l'ergotinine ni l'adrénaline n'entrent dans la composition de ces pommades. Certes, ces médicaments ont leurs indications ; mais leur emploi continu pendant plusieurs jours peut avoir des inconvénients qu'il y a lieu d'envisager sérieusement. A mon sens, il est préférable d'associer à la thérapeutique locale l'administration d'une potion au chlorure de calcium qui s'est montrée efficace dans bien des cas d'épistaxis que j'ai eu à soigner, chez des typhiques, notamment.

Dans ces dernières années, plusieurs articles ont paru dans la presse médico-spéciale, sur les méfaits du menthol en oto-rhino-laryngologie. Je me rallie complètement aux conclusions de leurs auteurs et c'est là le motif de son exclusion des préparations que je préconise.

Prescrit à des doses beaucoup trop fortes, le menthol s'acidifie en vieillissant, devient caustique, irritant. L'huile essentielle de menthe, fabriquée par distillation de la plante mère et non, chimiquement, de toutes pièces, n'a pas les mêmes inconvénients, à condition que l'on se serve de l'essence de qualité supérieure.

D'autre part, on recourra avec avantage, dans les cas d'érysipèle dus comme l'on sait, la plupart du temps, à l'infection lymphatique par a voie de fissures nasales, à l'onguent à l'Ichtyol ou au naphtalan 3 à 4 grammes pour 30 grammes de vaseline jaune ou de lanoline.

Il est notoire que la méningite cérébro-spinale se propage par voie nasale. Conséquemment, il est utile, voire même indispensable, de recourir, dans les cas de l'espèce, à l'un des topiques dont j'ai donné plus haut quelques formules.

Les malades grippés, les coquelucheux verront l'intensité de leur maladie, le nombre de leurs crises diminuer, la durée de l'infection s'abréger si l'on a soin de leur prescrire des soins du nez par des moyens appropriés.

Qu'il me soit permis de faire à ce sujet une réflexion générale : Des recherches ont été faites dans les salles de tuberculeux sur la présence, dans les fosses nasales des malades, des infirmiers, du personnel médical, du bacille de Koch. Elles ont abouti à la conclusion, facile à prévoir d'ailleurs, qu'il faut s'inquiéter de la possibilité de la contagion par voie nasale.

Si l'on a trouvé, d'autre part, le microbe de Weichselbaum dans le nez des méningitiques cérébro-spinaux, si le bacille de Pfeiffer a été décelé dans le mucus nasal des grippés, il est probable que l'on trouverait aussi les microbes pathogènes de la fièvre typhoïde, de la coqueluche, etc., dans les fosses nasales des malades qui en sont atteints, comme aussi dans celles des infirmiers et des médecins qui les soignent. Il y aurait là une belle étude à faire et je la signale à l'attention de mes collègues en spécialité aussi bien qu'à celle des médecins praticiens.

Si, par raisonnement, on peut accepter la proposition que je viens de faire, on doit aussi admettre que dans la plupart des maladies infectieuses, sinon dans toutes, et dans les fièvres éruptives, il est urgent et

indispensable d'instituer une thérapeutique nasale appropriée, rationnelle, qui sera en même temps curative pour le malade et prophylactique pour son entourage et pour les personnes qui le soignent.

Je n'ai rien de particulier à dire des moyens destinés à prévenir les complications de la rhinite du côté des cavités annexielles de la face ; la thérapeutique prophylactique des sinusites se confond avec le traitement des rhinites.

Pourtant je dois insister sur la nécessité d'empêcher les malades de se moucher bruyamment, avec force, en bouchant les deux narines à la fois. C'est une pratique détestable et dangereuse pour l'oreille et les sinus de la face. De toute urgence, il faut enseigner aux malades qu'ils ne doivent moucher que successivement les deux fosses nasales, sans y mettre un trop grand effort, de crainte de chasser vers les oreilles ou les cavités annexes du nez, les sécrétions nasales et leurs microbes infectieux.

3° *Soins du cavum.* — Ils se confondent, en somme, avec les soins du nez. Car s'il est loisible, à la première période des maladies graves, infectieuses ou des fièvres éruptives, de réclamer du malade sa collaboration active, cela devient aléatoire ou impossible dans la suite.

Certains topiques ont été préconisés dans les maladies qui nous occupent pour compléter l'action des lavages, des onguents, des instillations, des pulvérisations. A moins d'indications précises, péremptoires, je suis d'avis que l'on doit y renoncer. Comment, en effet, aller badigeonner le naso-pharynx d'un typhique, d'un méningitique, d'une façon vraiment efficace ? Ou bien on tente de le faire sous conduite du miroir et l'on reconnaît bientôt que la chose est malaisée ou impossible ; ou bien, de parti pris, on se passe du miroir et l'on pénètre de force et quand même, brutalement, derrière le voile du palais. Alors le tampon va de droite à gauche, de bas en haut et, en même temps qu'il cautérise ou porte l'agent modificateur, il meurtrit la muqueuse, fait saigner, ouvre des voies nouvelles à l'infection. En général, abstenons-nous de ces moyens brutaux ; comptons sur la thérapeutique nasale de douceur, et réservons les cautérisations aux cas d'ulcères du haut pharynx. N'allons pas, surtout, en pleine évolution de maladie infectieuse, sous prétexte d'obstruction rétro-nasale, porter la curette dans le cavum. Soyons médecins, soyons prudents, soyons humains !

4° *Soins des oreilles.* — Deux ordres de lésions se produisent d'habitude, du côté de l'organe auditif, en cas de maladies infectieuses, contagieuses, ou de fièvres éruptives, ce sont : les otites moyennes, complications d'angines, d'amygdalites, de rhinites ; ce sont, d'autre part, les lésions de l'oreille interne par les toxines fabriquées par le malade lui-même au cours de l'infection.

(*a*) *Oreille moyenne.* — La pathogénie même des complications tympaniques nous reporte tout d'abord vers les soins du cavum, du nez et de la bouche précédemment décrits. Mais si l'infection, devenant plus intense, menace de faire se rompre le tympan, il faut, sans hésiter, pratiquer la paracentèse précoce de la membrane. Ce faisant, on peut espérer

raccourcir la période de suppuration otitique. Les travaux de Körner, notamment, nous ont démontré la haute valeur thérapeutique de l'ouverture chirurgicale hâtive du tympan.

Que si la rupture s'est faite d'elle-même, il faut s'assurer que le drainage de la caisse peut se faire efficacement, et, dans le cas contraire, sans hésiter, il faut agrandir l'ouverture du tympan.

Voici donc écartées, par la parencentèse ou par l'appoint d'une incision libératrice, les complications de l'otite purulente. Deux méthodes thérapeutiques s'offrent au médecin : 1° celle, d'application facile, souvent efficace, des lavages détersifs renouvelés plusieurs fois dans la journée, avec, entre eux, le drainage à la gaze ou à l'ouate ; 2° celle du drainage systématique, traitement à sec de l'otite purulente, sans lavages quels qu'ils soient, mais avec surveillance quotidienne ou biquotidienne par le médecin.

Il est difficile de prendre position pour l'une ou l'autre méthode : il faut se guider sur les indications de chaque cas particulier, sur l'abondance de la sécrétion, la nature de l'agent infectieux, les symptômes de réaction locale, la possibilité ou l'imminence de complications mastoïdiennes ou autres, etc.

Si l'on se range du côté des partisans des irrigations d'oreilles, on les pratiquera à l'aide de solutions boriquées tièdes, à 30 pour mille de sérum artificiel, d'eau oxygénée, de perhydrol, etc. On emploiera, pour ce faire, la seringue stérilisable, le bock à irrigation continue, la canule à double courant, etc., maniés avec douceur, sans pression forte ; la quantité de liquide à injecter variera aussi suivant les indications ; elle sera, en général, de 1 demi-litre, au moins.

Après l'irrigation, on asséchera le fond du conduit, sous otoscopie, à l'aide d'un tampon d'ouate parfaitement stérile ; puis on insufflera l'une des poudres usitées en pareil cas, depuis l'acide borique jusqu'à l'aristol, l'europhène, le dermatol, etc., et on placera dans le conduit et à fond un tampon d'ouate hydrophile stérilisée ou mieux de gaze neutre stérile.

Ce qui importe, c'est de ne pas ajouter de nouveaux germes pathogènes à ceux qui ont infecté l'oreille. Ici comme dans n'importe quel domaine de la chirurgie, l'asepsie la plus complète est de rigueur.

Si l'on s'en tient aux pansements secs de l'otite, on agira comme je viens de l'indiquer plus haut, en ayant soin, préalablement, d'assécher au moyen de tampons stériles le fond du conduit et le tympan.

En tout cas, mais surtout s'il s'agit de scarlatine, on veillera à l'éventualité de complications osseuses de l'otite moyenne. On examinera chaque jour la sensibilité de la mastoïde à la pression, et dès qu'apparaîtront des signes indubitables d'antrite ou de mastoïdite, on fera l'ouverture chirurgicale de l'apophyse.

S'il s'agit de maladie infectieuse chez un ancien otorrhéique, on surveillera minutieusement l'oreille moyenne et, dès que du pus apparaîtra dans la caisse, on utilisera les instillations médicamenteuses d'alcool boriqué ou picriqué après détersion au tampon ; on redoublera de vigilance chez les scarlatineux.

N'oublions pas que si, d'après les travaux les plus récents sur l'étiologie de la scarlatine, il semble que l'on ne doive plus rapporter au streptocoque la cause de la maladie (Grünbaum, 1904, Catacuzène, Bernhardt, plus tard ; Levaditi, Landsteiner et Praseck, de l'Institut Pasteur de Paris, tout récemment), il n'en est pas moins vrai que l'agent pathogène nous est encore complètement inconnu à l'heure actuelle. D'autre part, n'oublions pas non plus que le streptocoque est le plus acharné, le plus virulent de tous les microbes qui envahissent l'oreille ; que c'est lui qui détermine les complications osseuses les plus graves ; que c'est lui, enfin, qui a une action élective spéciale nécrotisante sur le labyrinthe et qu'ainsi il cause des lésions extrêmement dangereuses, sinon irrémédiables du côté de l'encéphale et du cervelet. Apprenons à bien connaître notre ennemi, à le dépister, à en combattre la dangereuse puissance partout où il s'est installé.

Prendre des mesures prophylactiques autres que celles que je viens d'indiquer contre les infections mastoïdiennes et labyrinthiques, c'est se faire illusion, j'en suis certain. Envisageons donc à chaque instant la possibilité de devoir intervenir ; soyons prêts à le faire ; agissons dès que se montre le danger.

(*b*) *Oreille interne.* — Il doit être entendu que je n'envisage ici que l'action des toxines sur le labyrinthe acoustique ou statique, puisque j'ai rattaché à l'otite moyenne purulente l'envahissement du labyrinthe par le pus.

Dans ces dix dernières années, on en est arrivé à concevoir l'action néfaste sur l'oreille interne des toxines variolique, grippale, rubéolique, typhique, morbilleuse, etc., et on l'a rapprochée des intoxications syphilitique, diphtérique, alcoolique, tabagique et médicamenteuse.

S'il est vrai que le nerf auditif est, de tous les nerfs crâniens, le plus sensible aux intoxications (Politzer), on admettra sans peine que les délicats ramuscules de l'organe de Corti, les cellules de la membrane basale, soient fâcheusement influencées par les toxines quelles qu'elles soient.

Ceci nous amène à nous demander : 1° Quels moyens nous pourrions éventuellement mettre en œuvre pour diminuer la production des toxines ou leur degré de virulence ; 2° si nous devons nous reconnaître impuissants à cet égard, par quels moyens nous pourrons faciliter leur élimination et amoindrir ainsi leurs ravages.

Reportons-nous un instant à la doctrine humorale que sont venues éclaircir les théories pastoriennes, et voyons ce que faisaient nos pères médecins, influencés qu'ils étaient, ou plutôt dirigés dans leur thérapeutique par ces doctrines : ils faisaient boire abondamment leurs malades, ils les purgeaient, ils les saignaient, ils leur faisaient prendre des sudorifiques. Dans quel but ? Pour modifier les ' humeurs peccantes ', ce que nous appelons maintenant les poisons éliminés par les microbes. Et ainsi, empiriquement, l'expérience leur servant de guide, ils guérissaient autant de malades que les polypharmaceutes et les polypragmatiques qui leur succédèrent. Ils avaient mille fois raison de purger, de laver le sang de leurs malades, de le débarrasser des principes mauvais ; ils fai-

saient moins bien en les saignant et en leur enlevant ainsi, avec une certaine quantité de toxines, un nombre considérable de phagocytes.

Vu à la lueur de la science moderne, tout cela nous apparaît avec une netteté suffisante pour que, nous aussi, nous prescrivions, au cours des maladies infectieuses et des fièvres éruptives, des boissons abondantes, des sudorifiques, des purgatifs salins, pour que nous fassions à nos infectieux, en passe de faire de la sclérose de l'oreille interne, des infusions hypodermiques de sérum artificiel. En outre, nous nous garderons, à moins d'indications particulières, de leur donner des antithermiques, car la fièvre elle-même, en activant la transpiration cutanée, en augmentant l'évaporation pulmonaire, est une façon pour l'organisme d'éliminer ses toxines. Je crois qu'il y a dans l'hypodermoclyse une voie thérapeutique nouvelle à suivre. Mais, auparavant, médecins généraux et spécialistes de l'oreille auront à étudier ensemble les indications et les contre-indications générales de cette façon de procéder.

Mieux vaut prévenir que guérir, dit le proverbe. Cela est vrai aussi pour la sclérose de l'oreille interne, et je suis persuadé qu'en agissant comme je viens de l'indiquer, on s'épargnera souvent la peine d'imaginer des traitements empiriques teintés de modernisme scientifique qui n'ont d'effet réel que sur la situation monétaire de leurs auteurs.

Je ne crois pas utile d'envisager les moyens de prévenir les périchondrites et les nécroses du pavillon de l'oreille au cours des maladies qui font l'objet du présent rapport. Ce sont fort heureusement des raretés, même, en cas d'érysipèle, la plus grave des infections streptococciques.

5° *Soins du larynx et des voies respiratoires supérieures.* — La fièvre typhoïde, la grippe et la coqueluche sont les causes les plus habituelles des infections laryngées et trachéo-bronchiques ; l'érysipèle du larynx est exceptionnel ; l'œdème de la glotte au cours de la variole est aussi une rareté.

Encore une fois, les voies respiratoires supérieures ne s'infectent, au cours de ces affections, que par continuité de tissus et conséquemment d'infection, ou bien par contamination de haut en bas par les sécrétions buccales, nasales et naso-pharyngiennes. Conséquemment, les symptômes laryngés et trachéo-bronchiques des maladies infectieuses, de la grippe et de la coqueluche seront d'autant moins fréquents que l'on aura mieux soigné la bouche, le nez et le naso-pharynx.

Toutefois, là ne doivent pas se borner nos soins de thérapeutique préventive. On rencontre souvent, au cours de la fièvre typhoïde, de la sécheresse du larynx et de la trachée, constatable au laryngoscope, allant même parfois jusqu'à la siccité presque complète. Des ulcérations surviennent ensuite, qui compliquent encore l'état précaire du malade ; la nécrose peut atteindre l'épiglotte, les cartilages squelettiques du larynx et même la trachée.

Sans accorder aux inhalations salines chaudes et aux pulvérisations une valeur antiseptique quelconque, je pense qu'elles sont pourtant indiquées dans certains cas. Leur application est relativement facile

puisqu'il suffit de les amener dans le voisinage de la bouche ou du nez du patient, d'entretenir là une atmosphère humide, pour pallier dans certaine mesure aux complications citées plus haut. Certes, les inhalations et pulvérisations sont impuissantes dans la laryngite infectieuse, streptococcique, de la grippe ou de la fièvre typhoïde. C'est alors aux lavages intralaryngiens, aux injections intratrachéales d'huiles médicamenteuses qu'il faut avoir recours.

A ce propos, malgré l'opinion de certains de nos confrères français, j'estime que l'on ne peut faire de besogne utile qu'en pratiquant les injections intratrachéales sous la conduite de la vue. Si la laryngoscopie indirecte est difficile ou impossible chez les typhiques, on aura recours à la laryngoscopie directe qui permettra d'appliquer à bon escient l'injection intralaryngée ou intratrachéale.

Par contre, la laryngo-trachéite grippale ou la localisation trachéale de la coqueluche bénéficieront certainement des injections directes, intralarygiennes ou intratrachéales.

Enfin, a-t-on raison de croire à de la périchondrite ou à de la chondrite du larynx ou de la trachée, on se tiendra prêt à intervenir par voie externe ou par les voies naturelles suivant les indications de chaque cas.

Ayant ainsi exposé, sommairement pourtant, les principaux moyens prophylactiques et thérapeutiques à appliquer aux oto-rhino-laryngopathies compliquant les maladies infectieuses et les fièvres éruptives, il me reste à formuler quelques brèves conclusions. Les voici :

1° Le médecin qui soigne ces affections a pour devoir, actuellement, de compléter l'examen de ses malades par une enquête s'adressant au nez, à la gorge, aux oreilles et aux voies respiratoires supérieures. Il faudra donc qu'en attendant que les principes de l'oto-rhino-laryngologie soient pratiqués par tous les médecins, parce que faisant partie des examens universitaires, il recoure aux lumières de ses confrères spécialistes.

2° Lorsque sera établi le diagnostic de complications du côté de l'oreille, du nez, etc., des soins spéciaux et attentifs seront donnés à ces organes par un personnel infirmier spécialement stylé à cet effet. A défaut de soigneurs compétents, ou transitoirement, un médecin ou un élève médecin rompu à ces pratiques seront chargés de ces soins.

3° La surveillance du traitement des oto-rhino-laryngopathies doit être confiée à des médecins compétents.

4° Il y a lieu de prendre d'urgence des mesures spéciales afin d'éviter la propagation par le larynx, le nez, la gorge et les voies respiratoires supérieures des maladies infectieuses.

5° Les médecins généraux, unissant leurs efforts à ceux de leurs confrères spécialistes, doivent étudier ensemble quels sont les moyens prophylactiques à diriger contre les oto-rhino-laryngopathies compliquant les maladies infectieuses et les fièvres éruptives. Ils se mettront d'accord, notamment, sur la façon de prévenir l'intoxication de l'oreille interne par les toxines produites au cours ou au décours de ces maladies.

SECTION XVI

OTOLOGY

DISCUSSION No. 5

(Jointly with Section XV, Rhinology and Laryngology, Discussion No. 4)

THE SPECIAL TREATMENT OF THE THROAT, NOSE AND EAR DURING THE ACTIVE STAGES OF CERTAIN INFECTIOUS FEVERS, NAMELY—SCARLET FEVER, MEASLES, GERMAN MEASLES, MUMPS, INFLUENZA, TYPHOID FEVER, WHOOPING-COUGH, SMALL-POX, CHICKEN-POX, ERYSIPELAS, POLIOMYELITIS AND CEREBRO-SPINAL MENINGITIS (DIPHTHERIA EXCLUDED)

Additional Remarks by E. W. GOODALL, M.D., London

The Larynx

This organ is occasionally primarily affected in small-pox, erysipelas, influenza, measles, and whooping-cough; very rarely in any of the other diseases. Usually the affection is a laryngitis, involving more or less the whole interior of the larynx; but in small-pox it consists of an invasion of the organ with the eruption. This occurs in severe confluent cases, and may result in ulceration. Very rarely the same event is seen in chicken-pox.

The larynx may be involved secondarily in scarlet fever, erysipelas, influenza, typhoid fever, measles and whooping-cough. In scarlet fever and erysipelas it is affected by direct extension of the inflammatory process from the fauces. In scarlet fever, influenza, typhoid fever, and measles, the inflammation may go on to ulceration. The parts most commonly affected are the true vocal cords, especially their posterior extremities, and the mucous membrane immediately below them.

I have stated that the larynx is only occasionally attacked in these diseases. The infrequency of attack is shown by the following figures: Amongst 9,934 cases of scarlet fever treated in the Eastern Hospital during the ten years 1900 to 1909, only 24, or 0·24 per cent. developed a laryngeal complication; and amongst 1,316 cases of measles treated in the same hospital during the two years 1911 and 1912, 62 showed laryngeal symptoms, or 4·7 per cent. In whooping-cough the Asylums Boards Hospital Statistics show an incidence in the case of laryngitis of 0·5 per cent. Laryngitis is not tabulated amongst the complications of

typhoid fever in these statistics, because it is so rare. At the Eastern Hospital out of 247 consecutive autopsies of typhoid fever cases, in only 13 was the larynx affected, 5·2 per cent. The incidence in all cases is certainly below 1 per cent. Of the common acute infectious diseases, therefore, with the exception of diphtheria, measles is the one in which the larynx is most often involved.

In measles, if the patient recovers, the laryngeal complication usually also recovers, and any permanent defect rarely remains. But in those cases of scarlet and enteric fever in which the larynx is invaded, the patient is lucky if he escapes with his life, as usually they are of great severity. And if he does not die, the treatment of the laryngeal ulceration is often very troublesome, and the organ may be so hopelessly damaged, that he is condemned to wear a tracheotomy cannula for the rest of his life.

In respect of the treatment of the laryngeal complication my practice is as follows : Directly the patient develops laryngeal symptoms, I cause the air around him to be moistened with steam. I do not consider it necessary in every case to add any drug to the water from which the steam is generated ; but when ulceration is present, eucalyptus, creasote, or benzoin are useful. If the patient becomes worse is spite of the steam, the question arises—which operation should be performed, tracheotomy or intubation. If there is laryngeal ulceration, or if there are strong reasons for suspecting ulceration, tracheotomy should be performed. Intubation of an ulcerated larynx will often aggravate the ulceration. In the early laryngitis of measles, if you can tide the patient over the period of two to four days till the rash comes out, you will often save him from operation ; for as the cutaneous efflorescence makes its appearance, the laryngitis subsides naturally. It should be remembered that laryngeal symptoms arising late in an attack of measles not infrequently indicate the onset of diphtheria, and therefore a dose of antitoxic serum should be given. Intubation is to be preferred in the laryngitis of measles, should operative measures be required. But if the patient is constantly coughing out the tube, intubation should not be persisted in, but recourse had to tracheotomy. Otherwise troublesome ulceration of the larynx is certain to follow. As laryngeal symptoms in scarlet and typhoid fever are almost invariably due to ulceration, it is wise to perform tracheotomy straight away, and not to try intubation. If an intubated patient cannot do without the tube at the end of 12 or 14 days, even though he has been wearing it comfortably during that period, I usually submit him to tracheotomy. For in my experience, prolonged intubation leads to ulceration, and to a retained tube or cannula. Since I have followed these rules, I have had very little trouble with cases of this nature. Lastly, I should say that in the acute infectious diseases, intubation is not an operation for private practice. It is always necessary to have a medical man within there or four minutes' call, in case the tube should be coughed out.

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DISCUSSION

Mr. Arthur Cheatle (London). I wish to make a few remarks with regard to middle-ear infection in specific fevers. When the upper respiratory tract is infected by a specific fever, especially scarlet fever, measles, and influenza, I am strongly opposed to the use of irritating antiseptics and fluids of any sort; in my opinion hot soothing vapours should alone be used.

Directions should be given that the nose should not be violently blown and the nostrils should not be compressed during the act.

If the middle-ear tract becomes implicated an early and free incision should be made in the membrane. If the antrum shares in the infection the subsequent course of the disease and the treatment indicated depend on the type of bone present. If it is a cellular one pus forms in the mastoid with all its attendant dangers and requires early operation. If the diploetic infantile type exists, the dense outer antral wall prevents pus from penetrating to the surface, and the dense layer of bone between the central cavity and the diploetic mastoid prevents extension downwards, and several things may happen.

(1) Drainage through the membrane may be sufficient and cure may result without further operation.

(2) Intracranial, especially if the antrum is small, or labyrinthine complications may result, or

(3) Local destruction of the lining membrane of the antrum and attic takes place, caries of the wall occurs, especially of the cells which always line the inner antral wall, and of the ossicles ; later cholesteatoma forms and local osteosclerosis. In other words a *chronic* middle-ear suppuration is established. I may say that if mastoid cells are present but are surrounded by dense bone the same things may occur as in the pure infantile type.

It is exceedingly rare for us to see an acute suppuration of the middle ear become chronic apart from tuberculosis, the reason being that it becomes chronic when we aural surgeons do not see the initial acute infection, that is, during the specific fevers when the patients are in quarantine either at the fever hospitals or in their own homes. I can assure Dr. Goodall that hundreds leave the fever hospitals with chronic discharge from the ear or ears. No patient should leave the care of the hospitals or their private doctors with any discharge at all from the ear. The antrum should at least be opened. X-rays will be of great service in determining the type of bone present. I think a great deal of loss of health, hearing, and life would be prevented if specialists in ear, nose, and throat diseases were attached to the fever hospitals and if they were called in early when the cases are being treated in their own homes.

Professeur JACQUES (Nancy). Notre distingué rapporteur, le Dr Delsaux, a fait, à mon avis, de très justes réserves sur l'emploi des antiseptiques forts dans la thérapeutique des infections des muqueuses des premières voies respiratoires et digestives. Et dans les antiseptiques forts j'englobe l'eau oxygénée chirurgicale, dont on a fait, il me semble, grand abus et que je considère comme un médicament plus nuisible par son action irritante, quotidiennement vérifiée, que précieux par son pouvoir désinfectant, très discutable sur des surfaces recouvertes d'enduit muqueux coagulable par les acides.

Dr. DUNDAS GRANT (London) thought that nasal douches with aqueous solutions were apt to be dangerous, and should be avoided if possible. The same did not apply so much to sprays ; he was in favour of the introduction of oily solutions, such as liquid vaseline containing a small quantity of an antiseptic such as eucalyptol and very little menthol. The use of cocaine to diminish turgescences of the mucous membrane of the interior of the nose was of questionable safety ; he recommended in preference, as a substitute for it, a soft ointment containing in the half ounce, 20 grains of anæsthesin and 20 minims of a solution of adrenalin (1 per 1,000), either watery or oily. A weak spray of adrenalin might with discretion be used from time to time.

In regard to the ear its examination in young children was sometimes

imperfect and difficult, because the examiner did not realize that the auricle in them should be drawn down instead of up. He considered the routine introduction of carbolized glycerine into the ear a most valuable prophylactic, and Sir Robert Woods, of Dublin, as the result of laborious daily examinations and comparisons of results, found that a combination of opium, cocaine, belladonna, and creolin in a gelato-glycerine aural suppository was the most efficacious. Dr. Grant was in favour of the early opening of the mastoid cells, but he had met with cases in which the mastoid wound refused to close and in some of which the radical mastoid operation was called for. He quoted Dr. Knyvett Gordon as an advocate of the somewhat extreme measure of performing the radical mastoid operation in cases in which the suppuration persisted without doing the simple one first. He thought that few would care to adopt such a routine practice. Dr. Grant had observed no reference in Dr. Goodall's report to cases answering to Professor Killian's description of the almost malignant infection of the sinuses of the face, presenting the appearance of a circumscribed erysipelas, the sinusitic nature of which was apt to be unrecognized. No doubt it must be a very rare condition.

In regard to vaccines he considered that a great deal depended upon the expertness of the preparer and administrator. He considered Dr. Goodall's paper was of the utmost value as showing how the matter with which we are dealing presented itself to the epidemiologist, thus assisting us in finding out the *via media* in which safety lay.

Dr. Bronner (Bradford) used a nasal spray when there was much discharge from the nares, which was often of a 'crusty' nature. The de Vilbis nasal douche, which was really a very coarse spray, was most useful. In ordinary cases of rhinitis without much secretion *oily* sprays were the most useful, with menthol. These were also most useful from a prophylactic point of view. All persons exposed to infection should use a menthol and paroleine spray daily.

Weak solutions of cocaine increased the penetrating powers of drugs through epithelium, as had been proved by experiments on the cornea. Bendelak Hewetson was the first to advise the use of glycerine of carbolic ear drops in cases of threatened middle-ear suppuration.

Dr. Bronner had seen one case of acute œdema of the larynx after measles, which was at once relieved by injections of antidiphtheritic serum.

He had recently seen most interesting specimens by Professor Manasse of Strassburg, showing that in cases of fatal ear disease after scarlet fever the lining mucous membrane of the mastoid was always partially necrosed, much more so than in ordinary cases. This explained why bone complications were so common after scarlet fever.

Professor F. Pick (Prag). Es ist hier naturgemäss vorwiegend die Rede von lokaler Behandlung und Prophylaxe, ich möchte aber auf eine

interne Prophylaxe hinweisen, die vielleicht aussichtsvoll ist. Wir haben in Prag viel Abdominaltyphus gesehen — an einem Tage lagen im Spital gleichzeitig 222 Fälle; und wenngleich selten — selten im Vergleiche zu den relativ häufigen Larynxulzerationen, die die Sektion zeigt — sehen wir nach Typhus Perichondritiden und Stenosen, die eine längere Dilatationsbehandlung notwendig machen. Nun hat man in letzterer Zeit, um die Typhusbazillen im Urin unschädlich zu machen, die Darreichung von Urotropin vorgeschlagen, ein Mittel, welches auch bei infektiösen Katarrhen der Nase und der Bronchien (Influenza) oft gut wirkt. Redner hat auch den Eindruck, dass wenn man so Urotropin von der 2. bis 3. Woche des Abdominaltyphus gibt, diese Komplikationen von den oberen Luftwegen aus seltener werden, und er empfiehlt weitere Anwendung dieses gewiss unschädlichen Mittels.

Dr. DAN MCKENZIE (London) said that he ranged himself most decidedly upon the side of conservatism with regard to the treatment of enlarged tonsils and adenoids during the acute stages of the fevers. Experience of operating during the febrile attack upon those conditions the speaker had none, but his knowledge of operations upon adenoids in other septic states had filled his mind with caution with regard to the subject.

On the other hand, in connexion with acute otitis his opinion was strongly radical—perhaps even virulently radical. Early incision of the membrane is most efficacious. He strongly supported Mr. Scott's suggestion of daily inspection of the ear.

With regard to indications for opening the mastoid he was equally radical, holding not only that the antrum should be opened promptly when indications were decidedly present, but that it should also be opened when indications were doubtful.

In the matter of nasal douching and other nasal applications it was obvious that success and freedom from danger in this therapeutic measure was governed by the individual case. In the case of a nervous child, for example, the use of sprays, &c., may do more harm than good, so that it should not be adopted as a routine practice.

Of all cleansing throat sprays none had given him so much satisfaction as hydrogen peroxide frequently employed.

Dr. KŒNIG (Paris) has very few cases of sinus or middle-ear infection as complications of ordinary acute coryza, since in such cases he uses the following treatment which in the great majority aborts the coryza in one or two sittings. He first sprays both nostrils thoroughly with a weak solution of cocaine so as to make the nose absolutely free, the cocaine is then cleared out by a tepid alkaline spray, and when the patient has gently blown out the liquid which might remain, dry lactic acid bacillus powder is then blown all over the pituitary membrane. The patient continues at home to snuff up the powder, and is instructed to blow his

nose very little and with very little violence. This treatment is non-irritating, is devascularizing, and gives the speaker better results than any treatment he has ever used. No local antiseptic treatment should be used at the same time, for experiments have shown that antiseptics destroy the lactic acid bacillus.

Mr. Douglas Harmer (London) stated that sensitized streptococcal vaccines will sometimes produce a rapid cure in an acute septic infection, and showed charts of three cases: (1) A man with sepsis after an operation on the tonsils, in whom temperature fell to normal in three days. (2) A man with acute cellulitis of neck requiring incisions and tracheotomy, who gradually recovered in seven days. (3) A patient with mastoid disease who had had operations on the mastoid, lateral sinus, and cerebellum, and was then given vaccines, after which a rapid recovery ensued.

Dr. P. Watson-Williams (Bristol). Adenoids should not be removed in acute stages of infective fevers or even soon after. But as I and Professor Walker Hall have proved that adenoids are lymphoid hypertrophies in response to infection by cocci, their early removal before the onset of infectious fevers is a valuable safeguard. Intubation has great value in acute respiratory conditions, inasmuch as it often overcomes the necessity for a tracheotomy wound. I have had to resort to intubation in typhoid fever.

For many years I have insisted on the use of coarse sprays for nasal purposes and of the danger of douching. Protargol, weak iodine solutions, or biniodide of mercury (1 : 6000) can be used to thoroughly wash out. But avoid unnecessary and frequent use of cocaine, it tends to inhibit the resistance to mucosal infection. For the ear, glycerine of carbolic acid drops, early incision; and for mastoiditis relatively early Schwartze operation. But over-hasty interference is deprecated, as in generally healthy childhood the possibility of spontaneous recovery is far greater than in adults.

Mr. Herbert Tilley (London) condemned the use of nasal douches as being very dangerous, but a coarse nasal spray was of inestimable advantage in cleansing the nasal passages of obstructive and septic secretions. Such a spray will be of more value if the nasal cavities are first rendered patent by the use of a 3 per cent. solution of cocaine.

By thus cleansing and keeping free the nasal cavities, we shall render less likely infection of the nasal sinuses as well as of the ear.

With regard to the latter, the speaker was in the habit of ordering in any case with threatening ear symptoms, that five drops of a 5 per cent. solution of carbolic acid in glycerine should be poured into the auditory meatus. By this means we can relieve pain, and should a middle-ear abscess burst through the membrane or a paracentesis be performed, the discharge will flow into an aseptic passage, and the discharge

which is due to a monococcal infection will be likely to remain so, and will be more amenable to treatment than if secondary infection of the middle ear takes place from an unclean meatus.

Mr. SYDNEY SCOTT (London) showed on the screen sections of the temporal bone removed from a patient who had died of scarlet fever. He was indebted to Dr. Goodall for the bone. During life there were no local signs of mastoiditis, only otorrhœa. The temporal bone was of the infantile type described by the President. The sections through the antrum showed the engorged mucosa and the presence of masses of long-chain streptococci—the so-called streptococcus conglomeratus of Körte—which was probably the streptococcus pyogenes, specified by Gordon. He advocated the daily routine inspection of the tympanic membrane in fever patients, without awaiting more obvious symptoms indicative of operative treatment. He considered it advantageous to make inspections of the nose, and to remove crusts which formed. He found the coarse jet sprays most useful, as advocated by Mr. Tilley.

He asked Dr. Goodall whether he had any comparative statistics to show whether acute rheumatism occurred more frequently after faucial infections when treated with coagulating antiseptics, such as mercuric perchloride, than when treated say with alkaline sprays.

Dr. TRÉTRÔP (Anvers). Le traitement des oto-rhino-laryngopathies au cours des maladies infectieuses comporte aussi l'emploi si utile des vaccins microbiens. Pour ne citer ici que deux d'entre elles : pour la fièvre typhoïde le savant bactériologiste londonien Wright est l'auteur de vaccins très efficaces. Ceux de Chantemesse et Vidal en France vous sont aussi bien connus. Pour la coqueluche notre collègue et ami Bordet, directeur de l'Institut Pasteur de Bruxelles, a trouvé à la fois le microbe et le vaccin spécifique. Ces vaccins microbiens absolument indiqués dans des maladies infectieuses nettement définies agissent énergiquement sur la phagocytose, qui est renforcée à un haut degré ; ils ont une action à la fois curative et prophylactique, puisqu'ils réduisent le nombre de microbes spécifiques et diminuent à la fois la quantité de toxines qui empoisonnent le malade et la quantité de microbes susceptibles d'être rejetés au dehors et d'aller contaminer des individus sains. Au cours de très nombreux examens bactériologiques j'ai décelé maintes espèces pathogènes dans le nez et les voies respiratoires supérieures, et en ce qui concerne les fosses nasales j'aurai l'occasion de vous dire quelques mots dans ma communication de cet après-midi : les infections par voie nasale.

Professor MYGIND (Copenhagen). Dr. Goodall was inclined to believe that patients with otitis media contracted during infectious diseases generally left the fever hospital with the ear disease contracted there healed, or in a condition which did not demand surgical measures later on.

My experience does not at all support this view. In the Copenhagen Commune Hospital I often see in my department cases which have been

discharged from the fever hospital, which also is under the government of the Copenhagen Commune, and which are admitted on account of chronic suppuration of the middle ear or of subperiosteal abscess with osseous mastoiditis or of intracranial complications. Many of these patients have lost their lives, the surgical treatment having come too late. I think, therefore, that the treatment of ear diseases in fever hospitals is very frequently quite insufficient, and that many patients leave the fever hospitals with a mastoid process which really ought to have been opened in the hospital. I feel convinced that thousands of lives are lost each year all over the world, because they have not had their mastoid process opened during their stay in a fever hospital.

I must therefore repeat what I said last year at Liverpool at the meeting of the British Medical Association, that every fever hospital ought to have a competent aural surgeon attached to it. I was then supported in my view by all the *confrères* who took part in the discussion, and I am glad to hear that such a competent man as our President has said exactly the same at this meeting.

Dr. Maurice (Paris). A l'appui de la paracentèse précoce, je citerai, parmi beaucoup d'autres, un cas absolument typique. Un confrère m'appelle un soir pour voir un enfant de 2½ ans qui depuis trois ou quatre jours présentait une température qui oscillait entre 39 et 40°, sans qu'il puisse en déterminer la cause. Il craignait un début de méningite et me priait d'examiner l'oreille. Je trouve à l'otoscopie deux tympans un peu plus rosés que normalement, mais non bombés ; à tout hasard et devant l'innocuité de l'intervention je fais une double paracentèse qui n'amena que du sang. La température, qui à ce moment atteignait 39°,5, descendit d'un degré d'heure en heure ; deux heures et demie plus tard elle atteignait 37° pour ne plus se relever ; l'enfant dès le lendemain jouait et mangeait comme de coutume. Comme conclusion, dans la médecine infantile, l'examen otoscopique devrait être pratiqué dans tous les cas où l'on se trouve en face d'une hyperthermie dont on ignore la cause.

Professeur Segura (Buenos Ayres). Au sujet de cet important rapport, je veux insister sur la nécessité pour les médecins non spécialistes de posséder des connaissances générales et spéciales pour traiter les maladies infectieuses, ceci pour éviter les complications fréquentes du côté des oreilles, du nez et de la gorge, etc. Je suis convaincu qu'une bonne partie des complications produites par des traitements maladroits seraient évitées en possédant des connaissances spéciales même rudimentaires. Nous savons tous que les médecins ont de la tendance à employer des solutions caustiques plus irritantes qu'utiles ; pour ma part je me permets de conseiller, d'une façon générale, soit en instillation dans le nez soit en pulvérisation dans la gorge, etc., des solutions alcalines dans lesquelles on peut ajouter parfois de la cocaïne et de l'adrénaline pour bénéficier des effets heureux de la vaso-construction.

Dr. LAUTMANN (Paris). Les faibles solutions de protargol et collargol (1 à 2 %) administrées avec la seringue de Marfan doivent être appliquées chaque fois qu'il y a une maladie infectieuse de n'importe quelle origine, y compris la simple rhinite aiguë. Pour plus de détails voir *Maladies de l'oreille*, etc., n° 2, 1913.

REPLY

Dr. DELSAUX (Bruxelles). Je dois tout d'abord remercier Messieurs les Présidents et mes collègues des deux Sections réunies d'avoir bien voulu compléter par leurs remarques personnelles les grandes lignes de mon rapport. Les observations qui ont été présentées concordent, pour la plupart, avec celles que j'ai exprimées. Il en est pourtant qui présentent quelques divergences. Je suis plutôt étonné de retrouver ici des défenseurs de la douche nasale et les arguments qu'ils donnent en sa faveur ne m'ont pas convaincu, je dois l'avouer. Il fut un temps où dans la presse médico-spéciale on ne lui attribuait que des inconvénients et aucune utilité. Je crois qu'entre ces deux opinions il y a place pour un troisième avis, plus tolérant : *In medio virtus*. Mais je suis persuadé, toutefois, que les cas où la douche nasale est utile, au cours des maladies infectieuses, sont excessivement rares.

En ce qui concerne l'association de l'adrénaline et de la cocaïne aux 'sprays', pommades, etc., je suis obligé de faire également des réserves. Ces médicaments, l'adrénaline surtout, sont d'introduction relativement récente dans la thérapeutique et nous ignorons encore leurs effets éloignés. Si nous ajoutons à cela que ces médicaments sont confiés aux mains de personnes relativement peu au courant des doses thérapeutiques, qu'elles ont une propension à donner les soins indiqués pendant plusieurs jours de suite, sans interruption, nous pourrons nous convaincre qu'il faut faire sur ce point certaines réserves.

De l'ensemble de la discussion il semble bien résulter que tous nous sommes d'accord pour réclamer des administrations compétentes que des oto-laryngologistes soient attachés aux hôpitaux ou aux séances où sont hospitalisés les malades infectieux. Je crois bien interpréter notre désir à tous en priant MM. les Présidents de nos deux Sections de rédiger un vœu en ce sens.

Following the close of the discussion the subjoined resolution was put to the meeting and carried unanimously :

'That it would be greatly to the advantage of the community if experts in Otology and Laryngology were attached to the special hospitals for the treatment of epidemic diseases.'

The resolution was subsequently handed to Dr. Herringham, the General Secretary, by Mr. Arthur Cheatle and Mr. Sydney Scott, and by him transmitted to the Permanent Committee of the International Congress.

SECTION XVI

OTOLOGY

EPIDIASCOPIC DEMONSTRATION OF VARIOUS MICROSCOPIC PREPARATIONS

Von Professor WERNER KÜMMEL, Heidelberg

Verfasser zeigt mittelst des Projektionsapparates eine Reihe photographischer Aufnahmen von mikroskopischen Präparaten, z. T. Farbenphotographien nach dem Lumière'schen Verfahren.

Die ersten stammen von einem Fall von tympanogener Labyrinthitis, der an eitriger Meningitis zugrunde gegangen ist; der Einbruch war durch Perforation der Fussplatte des Steigbügels erfolgt. Auf zwei Aufnahmen der Schnecke sieht man, wie an den Wänden der Scala tympani und vestibuli Mikrokokken (nach Weigert gefärbt) in dichten Massen angesammelt sind, und auch im Ganglion spirale massenhaft auftreten. Die Zahl der Mikrokokken ist wohl durch postmortale Vermehrung so gross geworden.

Zwei weitere Diapositive zeigen bei einem anderen, gleichfalls an Meningitis verstorbenen Patienten die Infektion des lateralen Bogengangs von einem wahrscheinlich bei einer früheren 'Radikaloperation' operativ entstandenen Defekte aus.

Drei weitere Diapositive zeigen die Veränderungen im Labyrinth bei einem Soldaten, der in der vierten Woche einer Erkrankung an epidemischer Zerebrospinalmeningitis verstorben, und bereits am zweiten Tage vollständig ertaubt war. Vorher hatte der Patient gut gehört. In der untersten Schneckenwindung findet sich ein entzündliches, grösstenteils schon organisiertes Exsudat, die Gebilde des Ductus cochlearis sind hier fast ganz zugrunde gegangen. In den beiden oberen Schneckenwindungen kein Exsudat, das Cortische Organ relativ gut erhalten. Ebenso sind die Nervenfasern des N. cochlearis und seiner in der Lamina spir. ossea, auch der der untersten Windung, verlaufenden Fasern (nach Wittmaack durch sekundäre Osmierung gefärbt) auffallend gut erhalten. In der basalen Schneckenwindung und im Vestibulum findet sich bereits beginnende Verknöcherung.

Eine Mikrophotographie der Schnecke von einem Kinde, das in der zweiten Woche einer Pneumokokkenmeningitis gestorben ist, zeigt ausgesprochene entzündliche Infiltration des Nervenstammes im inneren Gehörgange, bei relativ geringen Veränderungen im Duct. cochlearis.

Nichtinfektiöse Veränderungen am menschlichen Labyrinth zeigt zunächst das Mikrophotogramm der Schnecke von einem Patienten, der ganz kurze Zeit nach der Exstirpation eines sehr grossen Tumors der gleichseitigen Kleinhirnhemisphäre gestorben war. Hier fanden sich kolossale Hämorrhagien im N. cochlearis und seiner Scheide, sowie in der Scala tympani, z. T. auch der Scala vestibuli, der ganzen Schnecke. Sie sind nach Meinung des Vortragenden vielleicht durch die mechanische Zerrung bei der Exstirpation zu Stande gekommen, aber auch dadurch schwer zu erklären.

Ein Mikrophotogramm der Schnecke von einem Patienten, der an Urämie bei chronischer Nephritis zugrunde gegangen war, ist dadurch interessant, dass der Cochlearis gut erhalten, der N. facialis aber stark degeneriert ist : gewöhnlich ist ja das Umgekehrte der Fall. Eine Autochromaufnahme zeigt die völlig normale Beschaffenheit auch der Gebilde des Duct. cochlearis.

Die Schnecke eines Falles von chronischer Mittelohreiterung, bei dem nachträglich nach Heilung der ' Radikaloperation ' ein Erysipel aufgetreten war und zum Tode geführt hat, zeigt weitgehende degenerative Atrophie des N. cochlearis und der Gebilde des Duct. cochlearis (Siebenmanns ' Cholesteatomtaubheit ').

Vollständige degenerative Atrophie des Cortischen Organs und des N. cochlearis zeigt ein Fall, der ohne Operation an einem grossen Tumor des Kleinhirn-Brückenwinkels zugrunde gegangen war.

Weiter werden mittelst des Episkopes farbige Zeichnungen vorgeführt, welche nach Präparaten aus einer Arbeit von K. Beck über experimentelle Schädigungen des Gehörnerven durch Bakterientoxine und andere Gifte angefertigt sind. Vor allem wird darauf aufmerksam gemacht, wie die Veränderungen, die sich an einem Teil dieser Tiere fanden, eine sehr verschiedene Deutung erfahren müssen. Die meisten der Präparate stammen von Tieren, die nach der Vergiftung durch ' intravitale Durchspülung ' getötet wurden : von der Aorta, bezw. dem Herzen aus, wurde in Narkose und unter künstlicher Atmung das gesammte Blut mit Ringer'scher Salzlösung ausgespült, und die Salzlösung dann durch Einfliessenlassen der Fixierungsflüssigkeit in das Gefässsystem ersetzt. Auf diese Weise kommen die Organe in ganz unveränderter Form zur Fixierung. Bei gewissen Vergiftungen zeigen sich dann zumeist nur feinere Veränderungen an den Nissl'schen Granula der Nervenzellen, an den Fasern des Cochlearis, seltener des Vestibularis und Facialis. Viel gröbere Veränderungen finden sich aber bei Präparaten, die nicht in dieser Weise, sondern durch Einlegen der herausgenommenen Organe in die Fixierungsflüssigkeit gewonnen wurden : hier hat die langsam eindringende Fixierungsflüssigkeit nachträgliche Veränderungen an Nervengebilden verursacht, deren Vitalität bereits durch den Einfluss des Giftes geschädigt, aber noch nicht erloschen war. Diese (Wittmaacks ' agonale ') Veränderungen sind durchaus nicht zu verwechseln mit den eigentlich ' postmortalen ', die an gesunden und kranken Organen durch die postmortale

Autolyse verursacht werden und ganz andern Charakter zeigen. Die Kenntnis dieser Verschiedenheit ist deshalb so wichtig, weil nur mit ihrer Berücksichtigung die pathologisch-anatomischen Befunde beim Menschen richtig gedeutet werden können. Beispiele dieser verschiedenen Veränderungen am Gehörorgan von Meerschweinchen und Kaninchen werden auf den Zeichnungen dargestellt.

Schliesslich werden noch zwei Diapositive von Meerschweinchenschnecken gezeigt, bei denen durch langandauernde Einwirkung derselben Pfeife (a^3) in einem Falle, bei leiserem Ton, eine Veränderung der bekannten Art im Cortischen Organ nur an dessen unterstem Teil, im anderen, bei Einwirkung eines lauteren Tones von gleicher Tonhöhe, aber eine viel weiter aufwärts reichende Degeneration erzielt wurde. Diese letzteren Präparate entstammen einer Untersuchungsreihe von H. Marx.

DISCUSSION

Dr. J. Gordon Wilson (Chicago). Professor Kümmel's demonstration specially interests me because some years ago I tried the effect of injecting organic arsenic compounds into animals to see the results on the vestibular nerve. The white rat was taken because it is peculiarly suitable for showing vestibular irritation. Apart from the physiological phenomena shown, which clearly point to labyrinthine irritation, the pathological findings of increase of plasma cells along the nerve point to local irritation by drugs belonging to this group.

[Monday Afternoon, August 11]

SECTION XVI

OTOLOGY

EPIDIASCOPIC DEMONSTRATION OF MICRO- AND MACRO-SCOPIC PREPARATIONS

By Professor GUSTAV ALEXANDER, Vienna

A. Macroscopic Specimens

Sections through the ear with cholesteatoma of the middle ear and labyrinth.

Macerated temporal bones showing chronic labyrinth suppuration with caries of the labyrinth capsule. Labyrinth sequestrum.

Exostosis of lateral labyrinth wall.

Congenital deformities of the middle ear and labyrinth.

B. Microscopic Specimens

Microscopic sections of the different stages of acute and chronic labyrinth suppuration.

Labyrinthitis ossificans Alexandri.

Congenital deafness with defective sinus septa of the cochlea (Alexander).

Macula neglecta of the human labyrinth.

Abnormalities in the histological structure of the normal ear-labyrinth of man.

Corpora amylacea in the nervus octavus.

Congenital deafness.

Otosclerosis.

Circumscribed chronic labyrinth suppuration.

Atrophy of Corti's organ in consequence of arterio-sclerosis.

Angiosarcoma.

Acusticus tumour.

C. Models

Model of the osseous and membranous labyrinth magnified 20 times.

Series of 12 models of the embryological development of the membranous labyrinth (guinea-pig).

Models of the embryological development of the labyrinth of echidna aculeata.

D. Devices and Instruments

Registering device of the statics of the human body.

Turning chair for clinical examination of the vestibular apparatus.

Instruments for the operation of labyrinth resection (after Alexander).

SECTION XVI

OTOLOGY

DEMONSTRATION

VARIOUS HISTOLOGICAL SPECIMENS EXEMPLIFYING PATHOLOGICAL CONDITIONS OF THE LABYRINTH

By Dr. J. S. FRASER, Edinburgh

1. Acute suppurative otitis media. Fistula into external canal. Acute purulent labyrinthitis.

There is a fistula into the external canal.
Coagulated lymph and pus in the external canal, posterior canal, and crus commune.
The internal auditory meatus shows meningitis.

2. Acute suppurative otitis media six months before death. Latent labyrinth suppuration. The patient was insane.

The scalæ of the cochlea are filled with coagulated fluid.
There is new bone formation especially in the scala tympani.
A fistula is seen from the tympanic cavity into the basal coil, which is almost entirely obliterated by new bone formation.
The internal meatus is almost entirely filled with new-formed fibrous tissue.
The seventh nerve appears comparatively healthy, but the cochlear nerve is replaced by fibrous tissue.
Fibrous thickening of the submucous tissue of the tympanic cavity and membrane.
The niche of the round window is obliterated.
The stapes is present in the oval window.
The vestibule is filled with new-formed fibrous tissue.
There is a fistula inwards from the vestibule to the posterior fossa ; above this an abscess cavity is seen (extra-dural abscess).

3. Chronic middle-ear suppuration.

An *exostosis* from the upper surface of the promontory blocks the oval window.

Chronic suppurative otitis media. Para-labyrinthitis.

Pus in the air-cells between the posterior canal and the posterior fossa.
The labyrinth is healthy.
The mucosa of the tympanic cavity is greatly thickened and engorged.

4. Chronic suppurative otitis media. Acute fibrino-purulent labyrinthitis.

The basal coil of the cochlea is filled with fibrino-purulent exudate.
The cochlear canal is dilated.
The internal meatus shows meningitis and hæmorrhage.
The oval window is empty.
The lower part of the vestibule contains pus and fibrinous exudation, as does also the posterior canal.

5. Chronic suppurative otitis media. Acute purulent labyrinthitis three weeks before death.

The attic cavity is lined by cholesteatoma.
A polypus projects from the inner wall in the region of the superior canal.
Both ends of the superior canal show peri-labyrinthitis ; the ' cartilage' bone is replaced by granulation tissue.
The endosteal bone of the ampullary end is necrotic in the superior canal while in the smooth end it has disappeared entirely.
The Eustachian tube is lined by mucous membrane, and the tympanic cavity lined by cholesteatoma.
The cochlea shows labyrinthitis apparently of somewhat older date than that in the vestibule and canals. The cochlear duct is markedly dilated and the other two scalæ are filled with vascular fibrous tissue. The bony capsule of the cochlea is almost healthy.
The vestibule and the external and posterior canals contain pus.
Marked peri-labyrinthitis is present in these situations. In the internal meatus the nerves are embedded in granulation tissue.

6. Chronic suppurative otitis media. Acute labyrinthitis five weeks before death. (Cerebellar abscess.)

The tympanic cavity is almost entirely obliterated.
Perforation of tympanic membrane.
Facial nerve embedded in fibrous tissue.
Vestibule filled with granulation tissue.
Pus in posterior canal.
Slight peri-labyrinthitis.
A large polypus projects into meatus from the region of the external semicircular canal. In the centre the polypus contains pus.
The polypus can be traced right through the labyrinth to the posterior fossa.
Necrosis of endosteal bone lining posterior canal.

7. Chronic suppurative otitis media. Purulent labyrinthitis six months before death. (Septic thrombosis of lateral sinus. Cerebellar abscess.)

The hollow spaces of the cochlea are replaced by new-formed fibrous tissue and bone.
Internal meatus filled with fibrous tissue, which shows recent hæmorrhages.
Old organized clot in jugular bulb.
The stapes is displaced into vestibule.
Vestibule filled with fibrous tissue and new bone.
It should be noted that in the centre of the vestibule pus is still present.
The jugular bulb contains organized thrombus.

8. Chronic suppurative otitis media. Old labyrinthitis. (Temporo-sphenoidal abscess.)

There is cholesteatoma on inner wall of tympanic cavity.
Cochlea shows new bone formation in scala vestibuli.
Vestibule shows dilatation of utricle and saccule, and some new bone formation in peri-lymph space.
External canal shows new connective tissue in peri-lymph space.

9. The patient was aged 75 years, and was totally deaf in the left ear. The case was regarded as one of senile deafness (?) but post-mortem examination showed that there was a tumour of the acoustic nerve on the right side, and microscopical examination showed an old labyrinthitis in the left ear.

Horizontal section of left ear shows new bone formation in the scala vestibuli of basal and middle coils.
Serous exudation in air-cells.
Well-marked corpora amylacea in eighth nerve.
There is new bone formation in scala tympani of basal coil.
Opening of aqueduct of cochlea is blocked.
New formation of fibrous tissue and bone in smooth end of external canal.

10. Chronic suppurative otitis media. Old labyrinthitis. (Recent septic thrombosis of sigmoid sinus.)

There is cholesteatoma on inner wall of tympanic cavity.
Cochlea entirely replaced by new bone.
Atrophy of eighth nerve.
Dilatation of utricle and saccule.
Fistula of external canal (old).
Peri-lymph space of vestibule and canals shows new fibrous tissue.
The section shows cholesteatoma in niche of oval window.
Foot plate of stapes replaced by fibrous tissue ?
New bone formation in peri-lymph space of vestibule.
Cochlea entirely replaced by new bone.

11. Tuberculous otitis media and interna. Child, age 9 months (breast fed). Death from tuberculous meningitis and general tuberculosis.

There is a fistula into scala vestibuli of basal coil ; basal coil itself filled with new fibrous tissue and bone.
Mucosa in region of round window is thickened and contains giant cells.
Middle coil of cochlea shows dilatation of scala media, and new fibrous tissue and bone in the other two scalæ.
The internal meatus shows meningitis.
There is also a fistula through promontory and oval window.
The vestibule contains caseous pus in the centre, and outside this new fibrous tissue and bone.

12. Age, 9 months (a twin). Tuberculous otitis media at age of 6 weeks. Labyrinthitis.

There is caries of bone of promontory.
Fistula into apical and basal coils.

All scalæ of cochlea filled with granulation tissue—probably tuberculous.
Tuberculous extra-dural abscess above cochlea.
Tuberculous disease of bone below internal meatus.
Escape of pus-cells into aqueduct of cochlea.
Superior and external canals contain pus and granulation tissue.
Nucleus of labyrinth replaced by tuberculous granulation tissue.
Ductus endo-lymphaticus contains pus.
There is extensive necrosis of outer part of labyrinth : the facial nerve is absent.

SECTION XVI

OTOLOGY

DEMONSTRATION

EXTRA MACULÆ FOUND IN THE AMPULLÆ OF SOME BIRDS

By URBAN PRITCHARD, Emeritus Professor of Aural Surgery, King's College, London

Last year at the International Otological Congress at Boston, U.S.A., I showed some microscopical sections of the bird's labyrinth in which I had recently discovered extra nerve terminations in the ampulla, and since then I have further confirmed these observations in other specimens.

Over and above the nerve epithelium found on the crista acustica of the ampulla, there are in some birds, such as the nightingale and probably other migratory birds, large areas of similar nerve terminations lining the walls of the ampulla and the surfaces of certain fan-shaped bodies described by Dr. Albert Gray in his valuable work on the labyrinth.[1]

The existence of these extra static maculæ has not, I believe, been demonstrated before, although Dr. Gray evidently suspected it from his macroscopical observations. Unfortunately I have not examined the ampulla of a sufficient number of birds to justify me in stating more than that these maculæ exist in some birds, together with the fact that they have so far not been found in any of the mammals or the other lower vertebrates (reptiles, amphibians, or fishes).

Considering the importance of an extra acute sense of orientation to birds [2] for flying, especially to the migratory birds, it is natural that they should require much larger areas of nerve terminations in the semicircular canals.

It is worthy of note that the cilia of the nerve cells of these extra maculæ are identical with those of the crista acustica, and differ markedly from the cilia of the maculæ of the saccule and utricle of the vestibule; and again, that they differ still more from the cilia of the cells of Corti in the cochlea.

[1] Albert Gray, *Labyrinth of Animals*, ii. 98.

[2] Dan McKenzie, 'The Semicircular Canals and Sense of Position or Orientation,' *Proceedings of the Royal Society of Medicine*, 1912, p. 141.

The cilia of the cells of Corti are very short and bristle-shaped, being arranged in a row of four to each cell of Corti.

The cilia of the vestibule are of two alternating forms, one bristle-shaped and the other thorn-shaped,[1] and are considerably larger and longer than those of the organ of Corti.

Lastly, the cilia of the ampullar crista are still longer and wavy, so that the bird's extra ampullar maculæ are easily distinguished from the vestibular maculæ.

Photographic Lantern Slide Demonstration

1. *The crista acustica of the cat.* Showing the whole course of the ampullar nerve with the large ganglion cells at its junction with the eighth nerve in the internal auditory meatus, and its passage throughout in bone until it reaches the crista, and there terminates in the nerve epithelium, which is covered with the long wavy ampullar cilia.

2. *Section of the macula acustica of the saccule of the cat.* Showing the nerve filaments passing from the nerve in the bone to the nerve epithelium, also the stiff vestibular cilia (alternating bristle-shaped and thorn-shaped) with the otolith mass above, the otoliths themselves having been dissolved out in the preparation.

3. *Section of the posterior labyrinth of the nightingale.* Showing the fan-shaped body of Gray in a cavity connected with the ampulla. The surface of this body is covered with ciliated cells, the cilia being long, needle-shaped, and wavy, like those of the crista acustica of birds and mammals.

4. *Another section through the fan-shaped body*, showing the ampullar nerve epithelium with long wavy cilia. These two (Nos. 3 and 4) demonstrate the extra maculæ found connected with the semicircular canals of certain birds, and the fact that these birds have a vastly larger area of nerve terminations than is found in mammals.

[1] Urban Pritchard, 'Termination of Nerves in the Vestibule and Semicircular Canals,' *Journal of Anatomy and Physiology*, 1878.

SECTION XVI

OTOLOGY

DEMONSTRATION

RECONSTRUCTION MODEL OF THE HUMAN LABYRINTH

By Dr. J. K. MILNE DICKIE, Edinburgh, and Dr. J. S. FRASER, Edinburgh

The original block measured about $\frac{3}{4}$ inch in all directions and the model has been enlarged 20 diameters.

The model shows :

1. The inner end of the external auditory meatus with the tympanic membrane.
2. The tympanic cavity with ossicles, also the aditus, antrum, and air-cells which partially surround the labyrinth.
3. The bony capsule of the labyrinth with the carotid canal, jugular bulb, opening of aqueduct of cochlea, saccus endolymphaticus, internal auditory meatus, and seventh and eighth nerves.

Parts (1) and (2) can be separated, antero-posteriorly, from part (3), so that the inner surface of the tympanic membrane with the malleus and incus can be seen, as well as the outer wall of the labyrinth with the tubal part of the tympanic cavity, tensor tympani muscle, promontory, oval and round windows, facial canal and prominence of the external semi-circular canal : the stapes remains *in situ* in the oval window with the tendon of the stapedius attached to it.

Part (3) can be separated into various bits in the transverse vertical direction, so that the perilymphatic spaces of the cochlea, vestibule, and canals can be seen as hollow spaces ; the membranous labyrinth itself has been left as a solid structure.

[Monday Afternoon, August 11]

SECTION XVI

OTOLOGY

DEMONSTRATION

MICROSCOPIC SPECIMENS OF SECTIONS OF THE HUMAN LABYRINTH

By Mr. SYDNEY SCOTT, London

Mr. Sydney Scott gave a screen demonstration of microphotographic slides of sections of the human labyrinth prepared by the paraffin method.

1. A case of acute labyrinthitis, showing disintegration of the membrana secundaria with streptococci *in situ*.
2. Other sections of the cochlea and vestibule from similar cases.
3. A true radial section of the organ of Corti fixed in Flemming's strong solution one hour after death.
4. The labyrinth from a case of bilateral auditory nerve tumour, showing the invasion of the basal coil of the cochlea by the growth.

The sections were prepared by the phloroglucin-nitric acid-paraffin process, and although it was possible to obtain sections of 5 or 6 microns, or even of only 3 microns, as in the case of the streptococcal specimens, yet the process had its drawbacks, for it sometimes failed, and valuable material had in this way been lost. He was struck with the apparently consistent results obtained by others with the celloidin method.

SECTION XVI

OTOLOGY

INDEPENDENT PAPER

NOTE SUR LA TECHNIQUE DES PANSEMENTS MASTOÏDIENS

Par M. le Dr. MAURICE MIGNON, de Nice

Les opérations mastoïdiennes entraînent généralement des suites post-opératoires prolongées, dont les inconvénients sont constitués, dans les cas favorables, plutôt par le pansement que par la plaie elle-même.

Pour arriver à diminuer ces inconvénients, on a recours à différents moyens que l'on peut classer en deux catégories :

1° Les *méthodes opératoires* qui doivent, par une technique s'adaptant à chaque cas, avoir pour but de donner le meilleur résultat possible dans un minimum de temps.

2° Les *méthodes de pansement* qui jouent aussi un rôle considérable et permettent d'obtenir selon leur emploi des résultats variables par des procédés plus ou moins avantageux.

On peut, dans un pansement, considérer *deux parties* : *l'interne*, faite au moyen de gazes variées, de poudres antiseptiques appliquées selon des méthodes diverses, qui ont été étudiées et discutées avec soin ; *l'externe*, composée d'ouate et de bandes qui constituent pour le malade la partie la plus désagréable du pansement. Il nous semble que, dans beaucoup de cas, on pourrait simplifier les pansements et les rendre à la fois plus esthétiques et plus confortables, tout en respectant les principes nécessaires pour leur bonne exécution.

On ne peut nier la gêne causée aux malades par les bandes de pansement qui, entourant la tête, sont particulièrement désagréables à la suite de la chirurgie mastoïdienne ; celle-ci permet souvent aux opérés de circuler peu de temps après l'opération, et même de reprendre leurs occupations, alors que l'aspect du pansement est pour eux la principale cause de souci. Quand l'état général est bon, quand la réaction locale a diminué, on est autorisé à faire le pansement le plus simplement possible, tout en ayant l'obligation de se conformer aux conditions suivantes :

1° Pansement léger, facile à changer, permettant d'éviter la transpiration de la peau voisine de la plaie, celle-ci facilitant l'infection, par la chaleur et l'humidité.

2° Réaliser les meilleures conditions d'esthétique, qui donnent satisfaction aux malades et leur permettent souvent sans inconvénients de reprendre des occupations nécessaires pour quelques-uns.

3° Faire une bonne occlusion et une bonne protection de la plaie en causant le moins de gêne possible au malade.

Ces conditions étant établies, nous aurons souvent des raisons qui peuvent amener à les réaliser autrement que par des bandes entourant la tête. Nous avons été engagés à étudier ce point de technique pour répondre aux désirs de plusieurs malades qui nous avaient sollicité. La première fois, il s'agissait d'une artiste qui, après avoir été opérée pour une mastoïdite, voulait au bout de peu de jours exécuter un engagement important et reparaître sur la scène d'un théâtre, d'une façon régulière, sans attirer l'attention sur l'endroit opéré ; mon premier essai, dont j'expliquerai plus loin la réalisation, me prouva qu'on pouvait facilement simplifier les pansements sans inconvénients. Dans la plupart des cas d'interventions simples (mastoïdites ou évidements avec petite ouverture de la plaie laissée derrière l'oreille), on peut au bout de peu de jours supprimer les bandes. Dans les cas pouvant guérir vite, la simplification du pansement a moins d'importance ; mais lorsque les suites se prolongent, cette modification n'est pas sans intérêt, surtout pour les personnes ayant par leur profession des obligations spéciales.

Par quoi peut-on remplacer les bandes ?

1° Par un pansement au *collodion*, moyen souvent employé, mais qui n'est pas sans inconvénients ; peu solide s'il est fait légèrement, ce pansement devient très difficile à enlever et pénible pour le malade lorsqu'il est fait avec une grande quantité de collodion et doit se répéter quotidiennement.

2° Par une *étoffe caoutchoutée* taillée d'après la forme de la région mastoïdienne et appliquée après avoir été légèrement chauffée ; cette toile spéciale, manquant d'adhésion à certains endroits, se soulève et ne protège plus la plaie, ou si elle est rendue adhérente tout autour de celle-ci, elle favorise d'une façon excessive les sécrétions ; enfin, pour enlever les débris de caoutchouc qui adhèrent autour de la plaie après chaque pansement, il faut employer des essences minérales dont les vapeurs seules, même s'il n'y a pas contact du liquide, sont irritantes pour la plaie.

3° Par une *pièce prothétique* pouvant s'appliquer facilement et rester fixée derrière l'oreille ; ce moyen est celui que nous avons cherché spécialement à réaliser.

Nous avons d'abord employé un petit appareil en caoutchouc vulcanisé que nous avons présenté en 1912 à la Société française d'Otologie,[1] sous le nom de *couvre-mastoïde*. Cette pièce a la forme de la région mastoïdienne et se prolonge par une sorte de croissant qui passe d'arrière en avant au-dessus de l'oreille et la maintient en place sans autre moyen. On peut cependant avec avantage augmenter la fixité de l'appareil en

[1] *Bulletins de la Société française d'Otologie*, mai 1912.

lui ajoutant un simple ruban de caoutchouc qui fait le tour de la tête et se trouve facilement caché par les cheveux.

A la suite de nos premiers essais, nous avons fait faire l'appareil en aluminium, et l'avons employé dans un certain nombre de cas avec toute satisfaction. L'opérateur pourrait lui-même couper dans une plaque d'aluminium la forme nécessaire et l'adapter à chaque cas particulier, pour réaliser le dispositif qui convient le mieux à la plaie et au sujet.

On peut mettre sous l'appareil, en suivant sa périphérie, une petite bande d'ouate qui empêche l'air de passer aussi librement, lorsque l'adhésion sur la région n'est pas complète ; on peut aussi, en suivant le bord de l'appareil, mettre sur sa face externe une petite bande de toile caoutchoutée qui le fixe plus solidement, mais elle n'est pas nécessaire. Pendant la nuit seulement, il sera bon de placer autour de la tête une bande qui sera enlevée chaque matin ; de cette façon on évitera que l'appareil se déplace pendant le sommeil. On pourrait encore ajouter quelques perfectionnements en munissant l'appareil d'un petit ressort qui se placerait autour de la tête et le fixerait complètement ; enfin la surface de l'appareil peut être, à la partie externe, peinte de la couleur des cheveux, pour le rendre presque invisible.

Il est difficile de dire à partir de quel moment l'appareil peut être employé après l'opération ; cela dépend naturellement de chaque cas. D'une façon générale, on peut souvent commencer à l'appliquer au bout de huit à dix jours, lorsque l'inflammation post-opératoire a disparu et que les points de suture sont assez solides.

Cette technique des pansements présente donc des *avantages* réels si l'on emploie l'appareil au moment qui convient. Cette simplicité des pansements est particulièrement pratique pour les femmes, dont les cheveux cachent complètement la plaque et la fixent plus complètement sur la région. Celle-ci, étant en métal, peut être stérilisée à chaque pansement. L'esthétique est donc satisfaite et la protection de la plaie suffisante ; pour les enfants, l'emploi de ce moyen est discutable, car il peut présenter des inconvénients.

SECTION XVI

OTOLOGY

DISCUSSION No. 6

CLIMATIC AND OCCUPATIONAL INFLUENCES IN DISEASES OF THE EAR

(For Reports by Dr. CLARENCE J. BLAKE and Professor GIUSEPPE GRADENIGO see Part I, pp. 309–35)

DISCUSSION

The PRESIDENT. A new cause of deafness is that of aeroplaning, and is one of which more and more will be heard. There are three factors, the noise of the engine, the height, and the direct pressure of air. In my patients it has been difficult to determine the part affected, for they have only come to me to be reassured as to their hearing which had been diminished directly after a flight, and had recovered completely. In one case both ears were affected, one recovering in a few days and the other in a fortnight. I expect the noise is the chief factor, and the deafness labyrinthine. The trouble is recognized by some pilots, for they direct their passengers to wear wool. One can imagine pathological conditions in the ear which would be likely to cause disaster during flight, especially in the case of a pilot. I believe that a pilot's certificate can be obtained without any medical examination at all, and it is therefore possible for a diseased person to have charge of an aeroplane, and to be a danger not only to himself and his passengers, but to those on whom he may fall.

For blocking the ears during gun-fire or in any noise I have found the 'clay fibre' invented by Sir William Dalby to be the best thing; it is a mixture of clay and fibre, so that the mass hangs together; it should be made soft and pliable by manipulating with the fingers, and placed in the ears like a piece of cotton wool.

Dr. BLEGVAD (Kopenhagen). Ich möchte ein paar Worte über die Einwirkung des professionellen Telephonierens auf die Ohren sagen. Prof. Blake sagt, dass bei Personen mit normalen Gehörorganen kein schädlicher Einfluss ausgeübt wird. Darin bin ich mit ihm einverstanden, weil es mit den Erfahrungen übereinstimmt, die ich in 1905 veröffentlicht

habe. Ferner sagt Prof. Blake, dass es sich gezeigt hat, dass das professionelle Telephonieren bei Personen, bei welchen das Gehör herabgesetzt ist, sehr wahrscheinlich eine Verminderung des Gehörs herbeiführen wird. Das stimmt aber nicht ganz mit meinen Erfahrungen überein. Ich bin in den letzten acht Jahren als Ohrenarzt bei der Kopenhagener Telephongesellschaft angestellt und habe in dieser Zeit alle die Damen, die Anstellung als Telephonistinnen suchten, einer genauen Ohrenuntersuchung unterworfen, und jeder Fall von schwererem Gehörleiden als Folge des Telephonierens ist von mir gesehen worden. Ich habe aber keinen Fall gesehen, wo das Gehör wirklich durch das Telephonieren allmählich vermindert worden ist, und ich habe das Wort ergriffen, um zu fragen, ob einige meiner hier gegenwärtigen mehr erfahrenen Kollegen je solche Fälle gesehen haben, also Fälle, wo das Telephonieren eine ganz langsame, progrediente Verminderung des Gehörs hervorgebracht hat, Ich bin doch mit Prof. Blake damit ganz einverstanden, dass nur Personen mit absolut normalen Gehörorganen als Telephonistinnen angestellt werden sollten. Nicht weil das Gehör bei ohrkranken Personen notwendigerweise durch das professionelle Telephonieren vermindert wird, sondern weil es beim Telephonieren vorkommen kann, dass das Gehörorgan von verschiedenen Traumen getroffen werden kann, die das Ohr schädigen können: ein starker Schall, wie er beim 'Läuten' oder bei einem Blitzschlag in der Telephonleitung vorkommt, kann — rein mechanisch — ein atrophisches, narbiges Trommelfell zerreissen und vielleicht ein Labyrinthleiden herbeiführen, obwohl kein solcher Fall mir bekannt ist. Ich möchte aber auch auf diesem Punkte meine mehr erfahrenen Kollegen fragen, ob sie solche Fälle von Labyrinthleiden kennen. Es sind viele Fälle — namentlich in Deutschland — veröffentlicht worden, wo z. B. Blitzschlag schweren Schaden bei Telephonistinnen hervorgebracht hat, auch Taubheit, aber alle diese Fälle sind rein hysterischer Natur oder sind traumatische Neurosen, und das Leiden ist nicht von der Elektrizität herbeigeführt, sondern eine Wirkung des Schreckens beim plötzlichen Knall, des starken Lichtes vom Blitz u.s.w. Ich bin mit Dr. Horn einverstanden, dass ein plötzlicher und unerwarteter Schall schwere, rein nervöse (hysterische) Störungen verursachen kann.

Mit den jetzigen Apparaten sind aber die Gefahren für die Telephonistinnen sehr vermindert worden. Es gibt kein Klingeln, Läuten mehr, und in den grossen Städten gibt es nur ganz wenige oberirdische Leitungen, so dass Blitzschläge beinahe ausgeschlossen sind. Die Verminderung der Schallintensität beim Ueberführen durch die Telephonleitung ist gewiss auch jetzt nicht so gross als wann Dr. Blake in den achtziger Jahren seine ersten Untersuchungen machte.

Dr. DAN MCKENZIE (London). With reference to the relative degree of harmfulness on the part of high and deep tones, I drew attention to the fact that although high-pitched noises might be more injurious than those of low pitch, it should not be forgotten, nevertheless, that

exposure to deep tones also caused ' noise deafness ' (this term he preferred for the nomenclature). Thus he had observed a large amount of deafness among ' beetlers ', men in the manufacture of cotton who worked amidst deep-sounding tones of long wave-lengths.

In general it might be laid down, with regard to noise in general, that painful sounds are harmful, for individuals varied considerably in their reaction to the effects of noise. Generally speaking, sharp rattling or brassy metallic ringing or braying were the most deleterious sounds. It was a frequent experience in the navy that gun-deafness was more common in the men who worked the smaller machine guns, than in those who attended to the giant ordnance, that is to say, apart from the occasional immediate effect of the ' blast ' or concussion upon the membrana tympani.

The effect of middle-ear disease upon the production of noise deafness was also not uniform. Sometimes the existence of a middle-ear obstruction had all the good effect of a plug in the meatus and shielded the cochlea from the blows of loud or penetrating sounds. At other times, middle-ear deafness was associated with hyperacusis, i.e. with an abnormal sensitiveness of the cochlea and tendency to suffer from injury. Thus the advice he had seen given as regards recruits for the navy, that persons with even mild middle-ear deafness should be rejected, was only advisable in certain cases.

With reference to prevention it had been generally agreed that although meatal plugs were of some use, their value was restricted by reason of the fact that much of the noise reached the labyrinth through the bones of the body. For this reason he had been considering whether it was not possible to lessen the vibrations which reached the cochlea through the bones. In the case of soldiers, the earth does not transmit sound-waves so readily as the rigid metal framework of a warship. But efforts should be made by, e.g., providing a sand-bed floor or other material to prevent or hinder the passage of the waves to the cochlea. Moreover, sailors should be instructed to wear suitable plugs, not cotton wool, and to bend the knees and back so as to lessen the transmission of the sound.

There was another side to the subject and that was the influence of the ever-increasing intensity of street-noises in modern cities. Locally, on the ear, the effect may be severe ; generally he was convinced of the tiring influence of noises upon the brain. This could be lessened by the wearing of suitable meatal plugs composed of some plastic material. He then described the calming and quietening effect of meatal plugs upon the mind, particularly in the prevention of train-tiredness.

Professor Karl von Eicken (Giessen). In dem Referat von Blake wurde gesagt, dass ich bei Meerschweinchen den Gehörgang auf einer Seite fest verstopft hätte, um zu untersuchen, ob das innere Ohr der zugestopften Seite durch die Schallwellen nicht geschädigt würden.

Dieses Zitat entspricht nicht ganz den Tatsachen: ich habe, um die Zuleitung des Schalles auf aerotympanalem Wege zu erschweren, einseitig den Ambos operativ entfernt und allerdings auf dem so behandelten Ohr beobachtet, dass die akustischen Schädigungen ausblieben. Hössli ist in der gleichen Weise vorgegangen. Aus diesen Versuchen glaube ich mit Sicherheit schliessen zu dürfen, dass die akustischen Schädigungen auf aerotympanalem Wege und nicht auf dem Wege der Knochenleitung zustande kommen. Deshalb dürften die Vorschläge, die Lokomotivführer und Kesselschmiede vor akustischen Schädigungen dadurch zu schützen, dass man sie auf eine Gummiplatte stellt, keinen Zweck haben.

Die schweren Störungen des inneren Ohrs, die Friedrich bei Marineoffizieren beobachtet hat, während die von Jaehne untersuchten Unteroffiziere des Landheeres weniger schwere Störungen zeigten, erklären sich durch die starken Detonationen und die starken Reflexionen der Schallwellen an den Wänden der Räume, in denen die Detonation erfolgt.

Von grossem Interesse ist die Beobachtung von Reinking bei zwei Schlossern, die nur auf der Seite eine nervöse Schwerhörigkeit aufwiesen, die ein gesundes Mittelohr hatten. Diese Beobachtungen wurden oben von Dr. Dan Mackenzie bestätigt: er fand bei Marineoffizieren, die ein Mittelohrleiden hatten, keine Schädigung des inneren Ohres. Ganz besonders hat mich die Mitteilung Mackenzies interessiert, dass Soldaten der Marine die Detonationen durch kleine Geschütze und Maschinengewehre in viel höherem Masse als schädlich empfanden, als die Detonationen grosser Geschütze. Dies steht in einem gewissen Gegensatz zu den Beobachtungen von Jaehne und Friedrich. Auf den Kriegsschiffen scheinen demnach die Maschinengewehre, die zu einer starken Erschütterung des Bodens keine Veranlassung geben, schwerer zu schädigen, als die grossen Geschütze: auch hier dürften die auf aerotympanalem Wege zugeleiteten Schallwellen die Schädigung bedingen. Die Erschütterung des Bodens allein — das ist durch die Versuche von Beck und Schäfer erwiesen — schädigen das innere Ohr. Deshalb glaube ich auch nicht, dass die Vibration des Aeroplans, die Dr. Horn eben angeschuldigt hat, das Gehör schädigen kann. Gegen akustische Schädigungen schützt uns nur ein fester Verschluss des Gehörganges.

Dr. HERBERT CHILD (Exeter). (*a*) Occupational deafness; loud noises and dust. If it is our duty to the community to protect the boilermakers and workers in dusty trades, much more is it our duty to protect ourselves and all town-dwellers from unnecessary noises, and still more, from street dust, that horrible compound of horse-dung and powdered stone, often flints, which we shall inhale so long as horses foul our streets. This is to be done by our influence on civic and municipal authorities.

(*b*) Concerning the effects of aeroplaning, in addition to the three factors given by Mr. Arthur Cheatle, add a fourth, the aspiration effect of a current of air passing over the aperture of a tube at right angles, as seen in some forms of spray. This leads to an actual *diminution* of pressure.

both in ear and lungs: both dyspnœa and alterations in hearing are experienced.

Dr. Kœnig (Paris) asked Dr. Blegvad if he had seen any improvement in the hearing of women employed in the telephone offices and affected by incipient otosclerosis or chronic deafness from various causes. Those people may hear very well through the telephone and should not be deprived of their means of livelihood. If re-education of the hearing by electrophonoid apparatus, as used by some of their colleagues on the Continent, was to be found of any value, he thought that the telephone would be a more ideal instrument to re-educate the hearing than the electrophonoid instruments, provided all extraneous sudden noises and shocks could be eliminated.

Dr. Maurice (Paris). Les orateurs ont insisté sur l'action nuisible du bruit dans les affections d'oreille. Le mécanisme de cette action est double. 1° Ou bien il s'agit de bruits violents et soudains comme un coup de canon, ce qui provoque des altérations aiguës (rupture du tympan, hémorragies intra-labyrinthiques); 2° ou bien c'est la continuité du son qui fatigue par sa monotonie et sa continuité (employés de téléphones et de chemins de fer, meuniers, etc.). Il s'installe chez ces sujets de la paresse auditive par le fait qu'ils se défendent des sons qui les énervent et les fatiguent. Il faut se pénétrer de cette idée que le son est utile pour entretenir l'ouïe et c'est là-dessus que sont basées les différentes méthodes de rééducation auditive, dont la mienne, mais qu'il devient nuisible lorsque les vibrations sont trop intenses et trop continues, tout comme un médicament peut être excitant à faible dose et déprimant à dose massive, l'alcool par exemple.

Reply

Dr. Blegvad (Copenhagen). In reply to Dr. Kœnig I may say that I am unable to give him the desired information. I never saw a case of beginning otosclerosis amongst the telephone-operators, and I think this is—partly at least—due to the fact that all persons seeking to be appointed as telephone-operators have had their ears carefully examined by me, and no person with diseased ears is admitted.

SECTION XVI

OTOLOGY

INDEPENDENT PAPER

DEUX CAS DE MÉNINGITE OTOGÈNE SUPPURÉE SUIVIS DE GUÉRISON

Par M. le Dr. G. COULET, Chef de Clinique Oto-rhino-laryngologique à la Faculté de Médecine de Nancy (France)

Je désire contribuer à l'étude des infections méningées, dont le point de départ est si souvent à l'appareil auriculaire, en vous rapportant deux cas de méningite otique, suivis de guérison complète.

Le temps n'est pas très éloigné où le diagnostic de méningite une fois posé équivalait à un arrêt de mort. Aujourd'hui encore, les auteurs classiques affirment que ' la guérison est exceptionnelle et que, dans la plupart des cas, la maladie laisse après elle des traces indélébiles (épilepsie, idiotie, etc.) '.

C'est pour apporter notre contribution à la réaction qui s'effectue contre cette opinion pessimiste que nous jugeons utile de citer l'histoire de deux malades de la clinique otologique de la Faculté de Médecine de Nancy, tous deux atteints de méningite suppurée otogène, confirmée par l'examen cytologique du liquide céphalo-rachidien, et guéris, sans aucune trace, par l'action chirurgicale accompagnée de ponctions lombaires.

Je résumerai brièvement l'observation du premier de ces malades que j'ai fait paraître en détail dans la *Revue médicale de l'Est* en 1911, en collaboration avec mon maître, M. le professeur Jacques.

1re *Observation.* — Il s'agit d'un jeune cordonnier, âgé de 20 ans, atteint, depuis son enfance, d'otite moyenne suppurée gauche.

Il nous est envoyé le 10 juillet 1910 avec des phénomènes de méningite bien caractérisés, dont l'apparition a coïncidé avec la cessation brusque de l'écoulement auriculaire. La température est à 39°, le pouls rapide, petit. Nous notons des vomissements, de la raideur de la nuque, de l'obnubilation intellectuelle.

L'examen de l'oreille malade permet de constater la présence d'enduit blanchâtre, recouvrant le tympan et de deux polypes, cachant une large perforation de la membrane.

Rien à la mastoïde. Deux jours après son entrée au service, la cure

radicale est pratiquée sous chloroforme : l'antre, l'aditus et l'attique sont débarrassés des nombreuses formations polypeuses qui l'encombrent. Nous découvrons, au niveau du canal semi-circulaire horizontal, une fistule, mais en aucun endroit la dure-mère n'est mise à nu.

Puis, profitant de l'anesthésie, nous pratiquons une ponction lombaire qui donne issue, à 10 cg. de liquide louche, à reflets verdâtres. L'examen de ce liquide nous apprend qu'il contient des *polynucléaires* assez abondants, *des globules de pus* dégénérés, quelques rares cocci, isolés ou en diplocoques prenant le gram.

L'état du malade s'améliore un peu, puis redevient mauvais les jours suivants, ce qui nous détermine à pratiquer une deuxième ponction lombaire ; elle donne issue à du liquide louche dont l'examen décèle des globules de pus et, comme agents microbiens, des microcoques, tantôt isolés, tantôt associés par diplocoques ou en paquet, et qui prennent le gram.

Le lendemain, la température est descendue à 37° et reste normale à partir de ce jour-là. L'état général du malade s'améliore continuellement et il quitte l'hôpital après un séjour de deux semaines.

Revu pendant les trois mois qui suivent son exeat, il ne présente aucun phénomène pathologique et est incorporé dans un régiment d'infanterie pour y accomplir ses deux ans de service militaire.

Voici donc une otite suppurée chronique, accompagnée d'ostéite des parois antrale et tympanique, avec érosion du labyrinthe et infection de l'espace périlymphatique ; la cure radicale donne issue au pus auriculaire, met un terme à l'infection du labyrinthe, tandis que la ponction réitérée du cul-de-sac lombaire élimine les bactéries colonisant dans l'espace céphalo-rachidien.

2e *Observation*. — Le 19 mars 1913, se présente à la consultation de l'hôpital un homme de 36 ans, journalier à St. Max-Nancy, il avait déjà été examiné à la clinique quinze jours auparavant pour un écoulement d'oreille à droite, nous avions diagnostiqué une otite moyenne aiguë, à tympan largement et spontanément ouvert, et prescrit des lavages qui devaient assurer la désinfection de l'oreille et amener une rapide amélioration.

En examinant à nouveau cette oreille, nous constatons que les principaux signes de l'otite ont rétrocédé et, en particulier, que l'écoulement purulent a cessé.

Mais, depuis deux jours, des phénomènes généraux inquiétants sont survenus ; maux de tête violents, état vertigineux, asthénie profonde. Son médecin note une forte élévation de la température et nous le renvoie avec prière de l'hospitaliser.

L'examen de l'oreille droite nous montre que la perforation de la membrane du tympan est cicatrisée et que l'audition est en partie rétablie. A gauche, nous trouvons un tympan congestionné et un peu tendu, dont l'incision immédiatement pratiquée reste sans effet.

L'état général est franchement mauvais, pâleur de la face, pouls rapide

et petit, température à 39°,5, photophobie, inégalité pupillaire, raideur de la nuque, rachialgie lombaire très accentuée, signe de Kernig, constipation, céphalée occipitale.

L'urine analysée est de composition chimique normale.

Tous ces signes constatés à l'arrivée du malade sont nettement caractérisés à la visite du soir.

Étant donné la bénignité de l'affection auriculaire, nous abandonnons l'hypothèse de méningite d'origine otique pour pencher vers l'idée d'un cas de méningite cérébro-spinale épidémique. (Cette dernière forme était d'autant plus plausible, qu'un cas de méningite de Weichselbaum venait d'être découvert dans le quartier de la ville où habitait notre malade).

Le lendemain, la température ne dépasse pas 37°, mais les signes de contracture s'accentuent encore et, le surlendemain, le thermomètre indique le matin 40°. Une nouvelle ponction lombaire évacue, sous forte pression, 20 cm. de liquide trouble que l'on remplace par 5 cm. d'électrargol. (L'examen microscopique, pratiqué au laboratoire de l'Institut sérothérapique, indiqua une *polynucléose abondante, sans méningocoque*, avec prédominance de lymphocytes dans le liquide de la deuxième ponction.)

Après la deuxième ponction lombaire, les symptômes alarmants s'atténuent progressivement, la température redevient normale cinq jours après l'entrée du malade au service, et il quitte l'hôpital, après un séjour de deux semaines, complètement guéri.

Huit jours après sa sortie de l'hôpital, il vient nous demander un certificat, constatant la non-contagiosité de l'affection pour laquelle nous l'avons traité ; la municipalité de son village, soucieuse de la santé publique, ayant interdit à sa femme de continuer à travailler chez ses concitoyens, par crainte de la dissémination d'une méningite cérébro-spinale épidémique dont son mari n'avait d'ailleurs pas été atteint.

Un mois se passe et, à notre grande surprise, le malade est renvoyé par son médecin avec les mêmes signes de méningite que la première fois (température élévée, pouls fréquent, signes de contracture, céphalée, etc.).

Puis, comme l'état menace de devenir inquiétant et que la température monte à 40°, une rachicentèse est pratiquée qui permet l'élimination de 15 cm. de liquide céphalo-rachidien louche, remplacés par 5 cm. d'électrargol.

Le lendemain, la température est redevenue normale, tous les phénomènes de méningite s'amendent et disparaissent peu à peu et six jours après sa nouvelle entrée au service, le malade reçoit son exeat. (Cette fois, aucun phénomène auriculaire n'a pu être relevé.)

Cet homme a été revu ces jours derniers : il a repris son fatigant travail de dégraisseur : il ne s'est jamais ressenti de la redoutable affection pour laquelle nous l'avons traité.

En résumé, méningite suppurée consécutive à une otite moyenne aiguë, suivie au bout d'un mois d'une rechute grave, guérie à la suite d'une paracentèse du tympan et de trois ponctions lombaires, dont deux suivies d'une instillation d'électrargol.

Ces deux cas montrent la possibilité d'une guérison complète des méningites suppurées ; ils indiquent, en outre, que la ponction lombaire constitue, non seulement, un moyen utile de contribuer à leur diagnostic, mais qu'elle apporte un appoint considérable à leur thérapeutique.

DISCUSSION

Mr. C. E. West (London). Too great pessimism is unjustified in cases of meningitis not secondary to sinus thrombosis or brain abscess. We are in much the same position as the early operators on general peritonitis. The factors to be combated are sepsis, intra-cranial pressure, and destruction of brain tissue. The nature and virulence of the infection are the important factors under the first heading. Direct treatment by vaccines, specific sera, and direct bactericides such as electrargol seems in general of small efficiency. In virulent infections there is usually a general blood infection before death. The destruction of brain tissue can only be influenced by diminished sepsis. The surgical opportunity is the prevention of intra-cranial pressure. When this is due to a meningeal effusion it may be effectively attacked : when caused by intra-ventricular effusion with blocking of the foramina of the floor of the fourth ventricle we seem to be helpless. Drainage of the meninges is possible through the lumbar theca or directly from the base of the brain. The speaker advocates the use of drainage through the internal auditory meatus (translabyrinthine drainage) for this purpose. Its advantages are the slight nature of the extension of the radical operation ; its long-continued efficiency and absence of liability to brain hernia ; and the surgically sound principle of following the route of the septic invasion and drainage of the region where infection is most concentrated. Cases treated in this way get great relief and in some cases recover ; in many cases living from ten to eighteen days after the operation in comparative comfort and often apparently dying of internal hydrocephalus.

To improve results we require : earlier diagnosis, examination of the cerebro-spinal fluid ; efficient drainage ; and increased assistance from the pathologists.

Professor Mygind (Copenhagen). Cases of pyogenic meningitis cured after treatment are by no means very rare. I myself have operated on over 50 patients with this intra-cranial complication and about $\frac{1}{5}$ of these have recovered. I am very doubtful whether lumbar puncture is of much therapeutic value in this disease, the results have, anyhow, in my hands been very unsatisfactory. The most important part of the surgical treatment of pyogenic meningitis is, in my opinion, early and complete removal of the primary focus of the disease in the temporal bone, including the opening of the labyrinth if this part of the ear is found diseased by the functional test. Whether it is necessary to open in all cases the subdural space is still *sub judice*. In my first series of cases I always

performed this operation and drained the subdural space, but I have latterly given it up after having found that it produced very often serious prolapses of the brain, and that I had lost one or two patients through this complication, who I think would have otherwise survived. When, however, there is the least suspicion either of cerebral or of subdural abscess or of an internal pachymeningitis, I always open the dura, and in all cases I expose it freely to make this examination, and also to be able to control the state of the dura during after-treatment and to facilitate an eventual secondary brain operation.

Dr. J. S. Fraser (Edinburgh) asked the opinion of Dr. Coulet as to the possible danger of spreading a localized meningitis by performing lumbar puncture. Dr. Fraser stated that he operated on all cases of otitic meningitis except one in which the patient was *in articulo mortis* at the time of admission. So far he had had no case of recovery but he intended to persist. Dr. Logan Turner of Edinburgh had already reported two cases of cure of purulent meningitis by operation combined with lumbar puncture. In Dr. Fraser's last case—a very hopeless one in which the labyrinth had already been drained—he performed the operation recently described in *The Laryngoscope* by Haynes of America: the operation was found to be technically easy in this instance but was unfortunately not successful. Dr. Stoddart Barr of Glasgow had recommended drainage of the lateral ventricle in these cases combined with lumbar puncture and lavage.

Dr. Jansen (Berlin) glaubt, dass wir an der Diagnose der serösen Meningitis festhalten müssen, und zwar bei dem vestibularen Symptomenkomplex ohne Neigung zur Steigerung und leichten oder mässigen Grades, ohne Funktions- und Reflexverlust, mit rascher Heilung. Die Steigerung der serösen zur—nicht bakteriellen—toxischen, resp. fibrinösen Interna können wir nicht mehr mit Sicherheit von der eitrigen erkennen, wenigstens zumeist nicht. Redner diagnostiziert sie aber in den Fällen mit langsamem Verlust und bei nur verhältnismässig leichtem, jedenfalls aber rasch verschwindendem vestibularem Symptomenbilde, eventuell zusammen mit Ertaubung. Bei dem grössten Teile der Fälle mit schwererem klinischen Bilde, raschem und dauerndem Funktionsverluste ist die Diagnose sehr zweifelhaft, so bei der Mehrzahl der Fälle von Voss. Redner diagnostiziert dann eitrige Interna. Zur Entlastung der Paukenhöhle genügt nicht selten die Paracentese.

Dr. Dan McKenzie (London) wished to bear witness to the satisfactory method of draining the meningeal spaces through the internal auditory meatus. The operation is easy and its results as regards drainage satisfactory in many, perhaps in most cases. As to sacrificing the labyrinth in so doing, the fear of doing so does not often arise. In a large proportion of cases of meningitis the labyrinth can be proved to be diseased, in others

the patient is so ill that immediate operation is called for, and in that case the fear of sacrificing the labyrinth does not trouble us. Our fears are for the patient's life. He agreed to some extent with what Dr. Mygind had said regarding subdural drainage, but chiefly because he believed that there were some cases which no amount of drainage would cure. The last case he had operated on he drained through the labyrinth and also made six or eight small openings through the cranium all over the skull on both sides of the vault and of the base, as well as the translabyrinthine drainage; the patient, however, died. And that brought him to the point that there were many cases in which general and free subdural drainage was unavailing. Many of these died of general sepsis due, no doubt, to absorption from the meningeal spaces. He suggested that we should endeavour to dilute and flush out the meningeal spaces in addition to draining them.

Mr. Sydney Scott (London). Although sharing the responsibility for developing the translabyrinthine operation which he had first proposed in 1907 (see *Archives of Otology*, 1908) he would not go so far as to recommend its adoption for those cases of otitic lepto-meningitis which were not accompanied by labyrinthitis. He had found by histological investigation that otitic lepto-meningitis was not always preceded by labyrinthitis, although labyrinthitis so frequently led to lepto-meningitis (cf. *St. Bartholomew's Hospital Reports*, 1909). In support of this view he mentioned three examples of otitic cerebro-spinal meningitis in adults, which recovered after the Schwartze operation and lumbar puncture alone. In each case the labyrinth was normal (according to symptoms and special tests)—no sera, vaccines, or silver preparations had been employed and translabyrinthine drainage had not been resorted to—yet these patients recovered from bacteriologically proved cerebro-spinal infections. He reserved the translabyrinthine operation for meningitis secondary to labyrinthitis. He had inserted the wire drain in specimen 129 (*Otological Museum Catalogue*). Later experiences showed it was not necessary to introduce the drain beyond the internal meatus, but if it was desired to do so, the wire should curve forwards to avoid injury to the pons. Having regard to the heavy death-roll of meningitis he congratulated the reader of the paper and also Professor Mygind on his 20 per cent. recoveries. Early diagnosis could not be too strongly insisted upon. Headache was often the outstanding symptom, but if sought for, there were generally rigidity of the cervical extensors and sluggishness of the pupillary and tendon reflexes. Twice he had observed a white 'tache cérébrale'—vaso-constrictor type. Recovery from meningitis purulenta was sometimes at the cost of mental derangement. Psychic phenomena were observable after recovery from serous meningitis.

Dr. W. S. Syme (Glasgow). Early diagnosis is all-important, and the examination of the pulse for early signs of increased cerebral pressure

may be helpful. The primary disease should be removed without delay and a lumbar puncture performed. These procedures may be followed by recovery, and it is interesting that the majority of recoveries of otogenous meningitis have followed these simpler measures. In one case in which there was severe frontal headache with sickness and vomiting, the performance of the radical mastoid operation, combined with the removal of a large quantity of turbid cerebro-spinal fluid by lumbar puncture from which a streptococcus was cultivated, was followed by the disappearance of the headache, and the patient ultimately recovered.

Before operation all cases should be carefully examined as to the condition of the labyrinth, and our methods should be adopted according to the result of this examination. In bad cases we should expose the middle and posterior fossæ extensively and open the dura.

Dr. Kœnig (Paris). Dr. Fraser asked Dr. Coulet if he thought that lumbar puncture offered any danger in otogenic meningitis. In that connexion I should like to say that at one of our local meetings in Paris a few years ago Dr. Lermoyez told us that he knew of a case of sudden death following immediately on a lumbar puncture.

Some of the gentlemen spoke of the importance of the early diagnosis of intra-cranial complications of otitis. It is not always easy to make an early diagnosis, as there may be no symptoms other than a most violent headache. I had such a case, where acute suppuration of the right middle ear had completely ceased when the patient came to see me. He kept going about, against my advice, attending to his work and complaining only of a most violent headache. I am sorry I did not open his cranial cavity, for I heard a few days later that he had suddenly died. I did not obtain a post-mortem.

Professor Alexander (Vienna) mentioned, by invitation of the President, many important points in the diagnosis and treatment of suppurative meningitis.

Dr. Jansen (Berlin) glaubt nicht, dass hier, wo man von Drainage spricht, wirklich eine Drainage erreicht wird. Man kann keinen Vergleich mit Abszess oder Peritonitis anstellen. Die eitrige Meningitis ist auch an sich nicht stets hoffnungslos, besonders die labyrinthogene ist es nicht. Durch die frühzeitige Entfernung des induzierenden Herdes (Labyrinth, die Thrombosis, Pachym. interna) kann man die Prognose oft gut gestalten. Bei Pachym. interna ist die Durainzision früh angezeigt. Redner wendet sich gegen die jetzt so häufigen Durainzisionen, die doch nie eine Drainage bewirken. Wenn man aber aus symptomatischen Gründen die Eröffnung der Dura vornehmen will, z. B. wegen enormer Kopfschmerzen, so erscheinen unter Umständen die Durainzisionen, resp. Eröffnung vom Fundus des innern Gehörganges durch die Schnecke, berechtigt. So wie die Inzisionen der Dura jetzt vorgenommen werden, verwirft sie der Redner.

Reply

Dr. Coulet (Nancy). A propos de la question posée par le docteur Fraser, à savoir: si la ponction lombaire est dangereuse, pratiquée au début de la méningite, lorsque le diagnostic est encore hésitant, je répondrai qu'en aucun cas la ponction lombaire n'est dangereuse. Il faut, bien entendu, la pratiquer avec toutes les précautions d'asepsie possibles pour ne pas infecter le liquide céphalo-rachidien et au niveau de la queue de cheval où il est impossible de traumatiser la moelle. Je crois d'autre part à l'utilité incontestable de la ponction lombaire lorsque le diagnostic de méningite est bien établi, parce qu'elle draine un liquide infecté et rempli de micro-organismes colonisant dans les espaces périméningés. Il ne suffit pas de s'attaquer à la cause (en drainant le labyrinthe ou en découvrant les lésions osseuses de la caisse et de l'antre), il faut aussi éliminer le plus possible du liquide infecté contenu dans le canal rachidien.

SECTION XVI

OTOLOGY

INDEPENDENT PAPER

NEW METHOD FOR THE DIRECT DILATATION OF THE EUSTACHIAN TUBE

By Á. V. GYERGYAI, M.D., Privat-Docent of Ear, Nose, and Throat Diseases at the F. J. Royal University of Kolozsvár, (Hungary).

Mr. Chairman and Gentlemen,—My method of direct examination of the naso-pharynx (which is shown by Figs. 1 and 2) allows us to see the opening of the Eustachian tube and to introduce relatively large instruments both into its pharyngeal orifice and into its interior.

This possibility gave me the idea of having dilators made which would correspond to the form of the Eustachian tube, and with which one could dilate an obstructed Eustachian tube better than by other methods.[1]

The tympanum with the antrum and mastoid cells communicates with the free air only through the Eustachian tube. The air in the middle ear is continually absorbed by the mucous membrane and is renewed at the moment of deglutition, yawning, &c., through the Eustachian tube. If the tube is narrow or blocked from any reason this ventilation is prevented. The blocking of the Eustachian tube is a very frequent occurrence, and is a causal factor in most ear diseases and makes their healing slow or imperfect.

The importance of a difference in density between the air inside and outside the drum has been recognized for a long time, and the great importance of catheterization and of the Valsalva and Politzer methods is that we can artificially force air through the narrow or blocked Eustachian tube and thus abolish the difference in air-pressure from time to time.

The use of bougies has been limited to those of filiform dimensions (Fig. 3 b), which are very small as compared with the size of the tube in the greater part of its length.

If the blocking of the tube is only temporary, it can be observed by

[1] My instruments can be had from F. L. Fischer, 113 Kaiserstrasse, Freiburg i. Br., Germany.

frequent inflation and sounding that the auditory organ remains less affected during the disease and that the healing is hastened. We can also influence chronic diseases in a satisfactory way with the Politzer method, by the catheter and bougie, if applied for some time; but in most cases we can preserve the hearing of patients to a certain degree, only if they are treated for many months or even years and there are

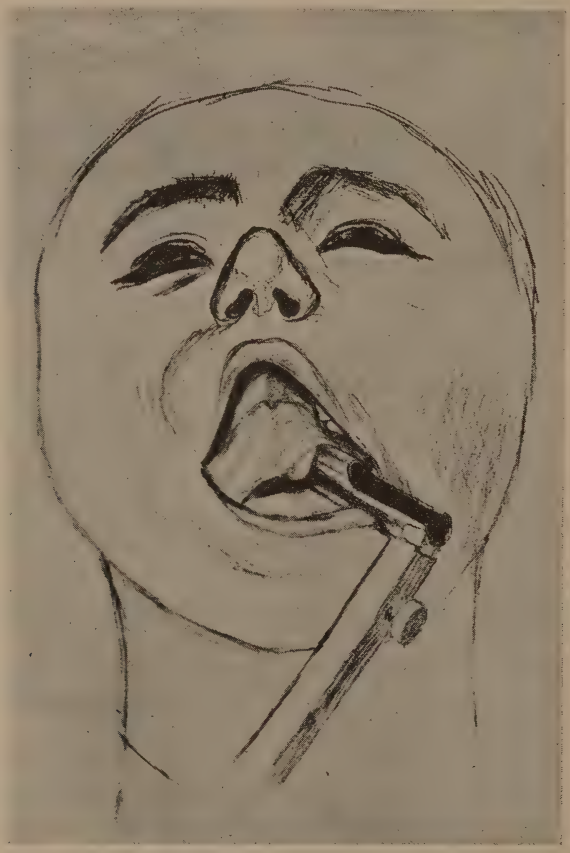


Fig. 1.

not long intervals between the treatments. There are some patients who need treatment during the whole course of their life on account of the blocking of the Eustachian tube. A too long duration of the blocking, in spite of inflation and sounding, in most cases gradually decreases hearing.

In the normal and pathological anatomical institutes, I have studied in several directions the anatomy and pathology of the parts and their surroundings explorable in a living person by my procedure. During these studies I have made, with Stent's composition, impressions of the cartilaginous tube (Fig. 3c) in dead bodies of various ages and sex.

From these impressions I have had metal casts made and handles attached to them (Fig. 3 a). The two sides are reversed specimens of each other, as is an image in a looking-glass. The inner side of the casts, where the cartilage lies, is flat and sometimes slightly concave, the outer side, where the wall of the tube is soft and elastic, is convex, the lower edge is concave and bent and the upper edge is narrow and convex.

I can easily introduce and push home into the Eustachian tube these large and smooth casts, which exactly correspond to its inner surface.

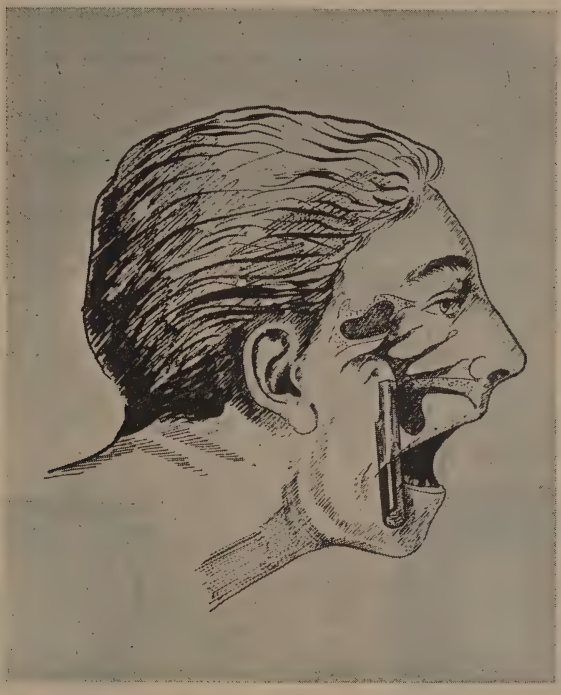


Fig. 2.

I place my straight tube through the backdrawn angle of the wide open mouth and put the upper grooved end on the back surface of the soft palate brought downwards and forwards by my hook; then the whole naso-pharynx can be thoroughly examined, and under the control of the eye medical treatment can be applied to all its parts (Figs. 4 and 5).

When I wish to examine the left Eustachian tube, I introduce my examining tube in the mouth from the right side, and vice versa. Before introducing the dilator, one places a straight plug steeped in a solution of cocaine in the lumen of the tube for a few seconds, and after taking out the same, one puts instantly the dilator inside. At a fresh insertion of the tube one can sometimes dispense with the application of cocaine.

As during the operation with the examining tube, the opening of the Eustachian tube appears in front and above, the point of the dilator tends a little downwards and backwards, the introduction is easier if its point looks upwards and also slightly forwards, until it has reached the

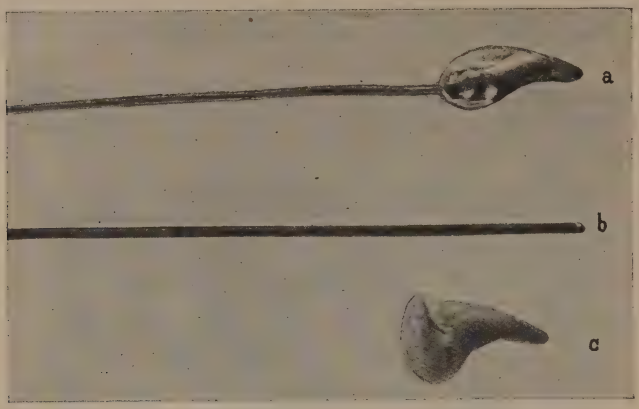
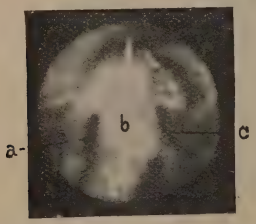
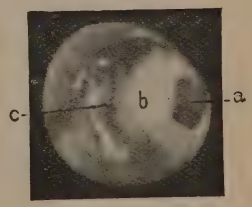


Fig. 3. (*a*) Dilator of the Eustachian tube; (*b*) usual bougie of the Eustachian tube; (*c*) cast of the dilated cartilaginous part of the Eustachian tube.

opening of the Eustachian tube. Now we must press it backwards in its original position and push it home. If one lessens pressure of the examining tube on the soft palate it is easier to place the dilator more deeply in the Eustachian tube.





Figs. 4 and 5. Photographs of normal pharyngeal orifice (*a*) of the Eustachian tube made on living persons under my method; (*b*) cushion of the tube; (*c*) fossa of Rosenmüller. My photographic apparatus, which is also suitable for making direct photographs of the larynx, is made by J. Leiter, 2 Mariannen-Gasse, Vienna.

I have used my dilator on 55 patients since February 15, on 32 patients only on one side, and on 23 patients on both sides; so that I dilated altogether 78 Eustachian tubes.

With	32	patients I dilated	42	tubes once only,
,,	12	,,	19	,, twice,
,,	2	,,	4	,, three times,
,,	3	,,	4	,, four times,
,,	1	,,	1	,, ten times,
,,	1	,,	1	,, fourteen times,
,,	1	,,	2	,, thirty-one times,
,,	1	,,	2	,, thirty-five times

Altogether I made the dilation 273 times. With the last 4 patients and 3 others I dispensed with the application of cocaine after the first few times.

I have undertaken the dilatation of the tube mostly with such patients, who, above everything, had depended on the Politzer treatment, catheterizing and sounding, and on massage, and with whom the sense of hearing had not improved much, or only for a short time.

In the following list I have made comparisons with the hearing of patients after they were treated under the mentioned methods and under my new treatment. Where not otherwise mentioned, the test of the sense of hearing was in a loud whisper.

The sense of hearing became almost or quite normal in 7 cases. After the other treatments one of the patients heard from a distance of only $2\frac{1}{2}$ metres, one from 6 metres, and three from the end of the room, say 6 metres, with the back turned. The other three heard before dilatation the ticking of a watch less than half a metre, but after my treatment from 2, 4, and lastly more than 5 metres!

After the dilatation 12 of my patients heard a normal whisper from the end of the room with the back turned. After the other treatment the same patients heard from only 3 metres, 3–5 metres, and 8 patients only loud whispering from the end of the room with the back turned.

Loud whispering from a distance of about 6 metres with the back turned was understood after dilatation by the following patients: one who heard before the operation only at a distance of 20–30 centimetres, 4 who could hear from a distance of 1–3 metres, 3 from 4–5 metres.

Five patients after other treatment could only hear loud whispering at a distance of less than 50 centimetres, after my treatment could hear 80–200 centimetres and 2 of them 400 centimetres. Seven patients heard from $\frac{1}{2}$ to 1 metre, and after the treatment 5 cases heard from 2–4 metres, and 2 from 2–6 metres. Two cases who heard from 1–2 metres improved to 3–$4\frac{1}{2}$ metres. One case improved from 3–5 metres. The noises in the ear generally decrease with improved hearing. In one case the trouble was only a noise in the ear, which ceased after dilatation. One case, where the bad hearing did not improve, the singing in the ear almost ceased. Only 10 cases did not improve.

After expansion by my dilator, the great improvement in the sense of hearing begins generally immediately, often after one treatment. During the next few days it often improves of itself without any further treatment, but after inflation or fresh expansion it improves still more markedly. Only in two cases was there no immediate good result. The patients became better only on the second or third day, and then made a great improvement.

In 10 cases with the ear drums perforated I dilated 16 tubes. The result was nearly equal to the cases where the drum was not perforated. In 8 ears there was some discharge before treatment, in 1 the improvement in hearing was from 10 centimetres to 6 metres with the back turned and in another 1–6 metres with the back turned. In other cases the improve-

ment was somewhat less, except one where the disease was of 35 years' standing and no improvement could be obtained. In 8 cases of perforation without discharge, 1 showed no improvement, 3 showed from half a metre to 2–4 metres improvement. In one, who had a radical mastoid operation some years before, the hearing improved from $1\frac{1}{2}$–5 metres.

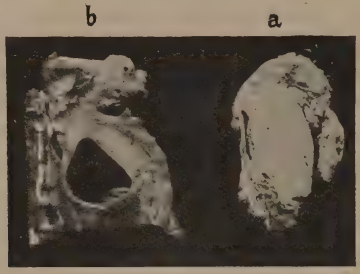


FIG. 6. (*a*) Cross cut through the Eustachian tube near its pharyngeal orifice; (*b*) the same after dilatation with my tube-dilator.

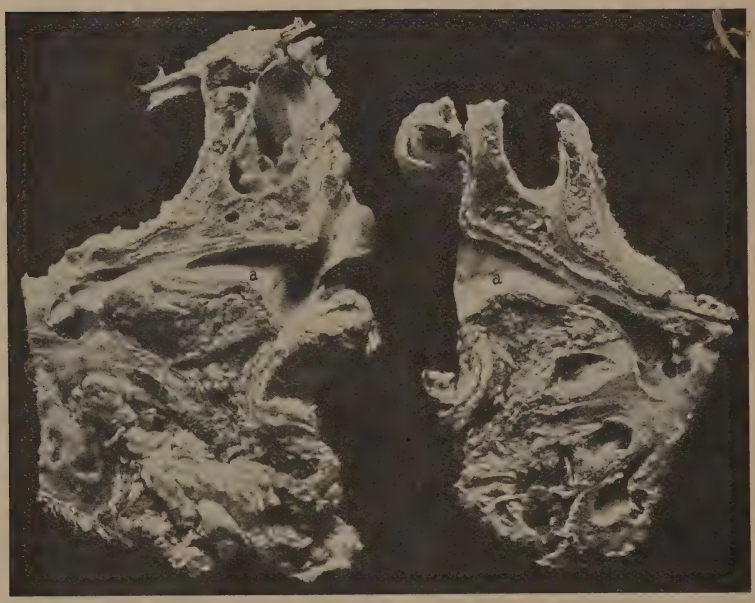


FIG. 7. Length cut through the Eustachian tube after dilatation of its cartilaginous part (*a*) with my dilator. Reduced as 5 : 4.

Another very important result of the dilatation of the tube is that the improvement in the sense of hearing is a lasting one. In many cases in which, for example, the improvement which has begun after the usual treatment, within 1 or 2 days, nay, from $\frac{1}{2}$ to 1 hour, commenced again to get worse, the improvement of the sense of hearing remained even after the first dilation 5–8 days or even longer at the same stage. Sometimes

the improvement of the hearing remained after the second or more frequent dilatation for weeks and is also good even to-day. Naturally I have also patients where the improvement was not so lasting. It is not a very long time since I began the dilatation, therefore I prefer to report later more fully the duration of the improvement in all my cases.

In order to avoid misunderstandings, I will remark that in diseases of the nerves of hearing of the labyrinth, dilatation is of no use. In otosclerosis the success is very small or nothing. The old chronic adhesive processes are suitable for dilatation, yet a lesser or less quick improvement of the sense of hearing is to be expected, than with middle-ear tube diseases, of some months' or some years' standing, in which the improvement is the most striking; above all, if they, during their illness, have been treated with the hitherto usual methods. I found no harmful effect whatever result from tube dilatation.

How is this favourable result of tube dilatation to be explained? That the Eustachian tube in consequence of the use of the tube dilator has a through passage and that the effect is a lasting one, is easily understood. The torus tubarius is pushed from the front lip of the ostium far behind, the elastic mucous membrane is greatly widened (Figs. 6 and 7); consequently the folds become smooth, the openings of the glands become wider, the contents empty themselves more easily, if there are perhaps adhesions which are seldom visible they separate, the negative pressure and the hyperæmia ceases, in short the passage becomes wider.

It is more difficult to explain why we get a better result as regards the sense of hearing with my method than after simple inflation of air, both with non-perforated and especially with perforated ear-drum. It is certainly known that an inflation can produce improvement of the sense of hearing also in cases where the drum is perforated. The reason for this is probably analogous in both cases.

When I see the result of my procedure, I think that I have every reason to hope that, as an accessory to other treatments, it will be helpful for many cases of ear diseases.

DISCUSSION

Professor ALEXANDER (Wien) kann die Methode Gyergais aus eigener Erfahrung bestens empfehlen. Die Methode leistet in der Hand des damit Vertrauten sehr viel in Fällen von chronischem Adhäsivprozess, die jeder anderen Behandlung trotzen.

Dr. MAURICE (Paris). Les manœuvres directes pour voir ou traiter les affections de la trompe sont forcément assez pénibles. J'ai fait construire un pharyngoscope assez simple qui relève automatiquement le voile du palais et qui permet de voir et de traiter la trompe d'Eustache sans que le malade soit placé dans une position gênante ou pénible. Le pharyngoscope est placé dans la bouche comme un abaisse-langue et l'on fait les manœuvres par le nez.

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7390 DONALDSON, MALCOLM, St. Bartholomew's Hospital, E.C.
3675 DONELAN, JAMES, 6 Manchester Square, W.
3810 DORAN, ALBAN H. G., 6, Palace Mansions, West Kensington, S.W.
7397 DORE, S. ERNEST, 26 New Cavendish Street, W.
2031 DOVE, AUG. C., Brightside, Crouch End Hill, N.
1506 DOVE, EMILY LOUISA, 1 Vincent Square, Westminster, S.W.
268 DOWSETT, ERNEST BLAIR, 1 Gloucester Street, Portman Square, W.
351 DRAKE-BROCKMAN, Lieut.-Col. EDWARD FORSTER, 18 Hanover Street, Hanover Square, W.
1060 †DREW, DOUGLAS, 6 Wimpole Street, W.
1845 DREWITT, F. D., 14 Palace Gardens Terrace, Kensington.
1868 DRYSDALE, JOHN HANNAH, 11 Devonshire Place, W.
1071 †DUCKWORTH, Sir DYCE, 28 Grosvenor Place, S.W.
1420 DUDFIELD, REGINALD, 19 Blomfield Road, Maida Vale, W.
1418 DUDGEON, LEONARD S., 6 Stanhope Street, W.
3877 DUGGAN, NORMAN, St. Marylebone Infirmary, Ladbroke Grove, W.
7411 DUNLOP, BINNIE, Alexandra Court, S.W.
7361 DUNN, HUGH PERCY, 54 Wimpole Street, W.
1195 DUNN, WILLIAM EDWARD NICKOLLS, Junior Athenæum Club, Piccadilly, W.
266 DURHAM, FREDERIC, 34 Dover Street, W.
2093 †DUTCH, MYER AKIBA, Linden House, 197 Walm Lane, Cricklewood, N.W.
3904 DWYER, JAMES, 10 Sunny Gardens, Hendon, N.W.

E

1222 EBERSTADT, FRITZ, German Hospital, Dalston, N.E.
739 †ECCLES, WILLIAM MCADAM, 124 Harley Street, W.
989 ECKENSTEIN, KENNETH, 8 Bentinck Street, Cavendish Square, W.
3796 †EDDOWES, ALFRED, 28 Wimpole Street, W.
224 EDEN, THEO. WATTS, 26 Queen Anne Street, W.
658 †EDER, MONTAGU DAVID, 7 Welbeck Street, W.
3575 EDHOLM, GUSTAF, 16 Hobart Place, Eaton Square, S.W.
1376 †EDKINS, J. SYDNEY, Bedford College, Regent's Park, N.W.
802 †EDMUNDS, ARTHUR, 57 Queen Anne Street, W.
1642 EDRIDGE-GREEN, F. W., 99 Walm Lane, Willesden Green.
1483 EDWARDS, FRANCIS HENRY, Camberwell House, Peckham, S.E.
1761 EDWARDS, FREDERICK SWINFORD, 68 Grosvenor Street, W.

4543 EDZARD, DIETZ L., German Hospital, Dalston, N.E.
1478 EHRMANN, ALBERT, 255 Camden Road, N.
3664 ELLIOTT, CHARLES COYNE, China Inland Mission, Newington Green.
719 ELLIOTT, THOMAS RENTON, 101 Harley Street, W.
1993 ELLIS, HENRY HAVELOCK, 14 Dover Mansions, Canterbury Road, Brixton, S.W.
1092 ELLISTON, GEORGE S., 36-38, Whitefriars Street, E.C.
460 ELMSLIE, REGINALD CHEYNE, 15 Devonshire Place, W.
7155 EMBLETON, DENNIS, 24 Weymouth Street, W.
958 †EMERY, W. D'ESTE, 48 Harley Street, W.
1340 †ENGLISH, THOMAS CRISP, 82 Brook Street, W.
7376 ENSOR, EDWIN T., 162 Ladbroke Grove, W.
386 †EVANS, ARTHUR HENRY, 28 Devonshire Place, W.
903 †EVANS, EVAN LAMING, 50 Seymour Street, W.
1891 EVANS, WILLMOTT, 121 Harley Street, W.
17 †EVE, Sir FREDERIC, 61 Harley Street, W.
476 ‡EYRE, JOHN W. H., Guy's Hospital, S.E.

F

411 FAGGE, CHARLES HERBERT, 3 Devonshire Place, Portland Place, W.
2047 FAIRBAIRN, JOHN SHIELDS, 42 Wimpole Street, W.
968 †FAIRBANK, HAROLD ARTHUR THOMAS, 84 Harley Street, W.
510 FAIRBANK, JOHN GERALD ATKINSON, 18 George Street, Hanover Square, W.
841 †FAIRFAX, EDWARD WILFRED, c/o Messrs. B. S. Floyd and Co., 40 King Street, Cheapside, E.C.
3633 FAIRLEY, JAMES FAIRBURN, Bloomsbury House Club, Cartwright Gardens, W.C.
772 FAULDER, THOMAS JEFFERSON, 6 Harley Street, W.
729 FAWCETT, JOHN, 66 Wimpole Street, W.
1075 FEARNSIDES, EDWIN GREAVES, London Hospital, E.
2069 ‡FENWICK, E. HURRY, 14 Savile Row, W.
680 †FERRIER, Sir DAVID, 34 Cavendish Square, W.
1895 †FERRIERE, LOUIS P., De Walden Court, New Cavendish Street, W.
7062 FILDES, PAUL, 11 Melbury Road, Kensington, S.W.
1390 FINEGAN, ARTHUR, 49 Albert Bridge Road, Battersea Park, S.W.
2017 FINK, LAWRENCE G., The Ruskin Manor Hotel, Denmark Hill, S.W.
3677 FINZI, NEVILLE SAMUEL, 107 Harley Street, W.
1404 †FIRTH, KARL J., 39 Harley Street, W.
1456 FISHER, JOHN HERBERT, 83 Wimpole Street, W.
1392 FISON, JAMES, Resident Staff Quarters, St. Bartholomew's Hospital, E.C.
1760 FLEMMING, Mrs. EMILY, 70 Harley Street, W.
1759 FLEMMING, PERCY, 70 Harley Street, W.
1153 FLETCHER, HERBERT MORLEY, 98 Harley Street, W.
2068 FORBES, JOHN, 106 Shaftesbury Avenue, W.
1099 FORD, ROSA, 30 Pepys Road, New Cross, S.E.
50 FORSTER, LAURA, 29 Stanley Gardens, Notting Hill, W.
6880 FORSTER, WILLIAM AUGUSTUS DE VERE, 20 Grosvenor Street, W.
1546 FORSYTH, DAVID, 74 Wimpole Street, W.
902 FORSYTH, J. A. CAIRNS, 152 Harley Street, W.
446 FORSYTH, LENNARD WILLIAM, 24 George Street, Hanover Square, W.
1974 FOULERTON, ALEXANDER G. R., 1 Morpeth Terrace, Victoria St., S.W.

6589 Fox, Hugh Clayton, 1 Weymouth Street, W.
429 †Fox, R. Fortescue, 145 Harley Street, W.
1471 Fox, Richard Hingston, 145 Harley Street, W.
1701 Francis, Lilian Angela, 9 Henrietta Street, Cavendish Square, W.
6756 Frazer, John Ernest, St. Mary's Hospital, London, W.
55 †French, Herbert, 62 Wimpole Street, W.
490 French, Major Herbert Cumming, c/o Messrs. Holt and Co., 3 Whitehall Place, S.W.
3907 Frenchman, Col. E. P., 8 Surat House, Loudoun Road, St. John's Wood.
812 Freshwater, Douglas, 13 Welbeck Street, W.
1509 Freyer, Peter Johnston, 27 Harley Street, W.
1997 Fry, Hubert John Burgess, St. Thomas's Hospital, S.E.
243 Funk, Casimir, Lister Institute, Chelsea Gardens, S.W.
1610 Furber, Edward P., 25 Welbeck Street, W.
1404 †Fürth, Karl J., 39 Harley Street, W.

G

1218 †Gabe, John Rees, 3 Mecklenburgh Square, W.C.
427 †Gabell, Douglas, 9 Portland Place, W.
6568 Gai, Nariman J., 107 Abbey Road, Hampstead.
1754 Galloway, James, 54 Harley Street, W.
1764 Gans, Friedrich, 17 Welbeck Street, W.
272 Gardner, Harold Bellamy, 126 Harley Street, W.
794 ‡Garrod, Archibald E., 9 Chandos Street, W.
1713 †Gask, George Ernest, 41 Devonshire Place, W.
1158 †Gaskell, John Foster, 23 Ladbroke Grove, W.
1011 †Gaster, Anghel, 33 Fitzjohn's Avenue, Hampstead, N.W.
3809 Gauvain, Henry John, 130 Harley Street, W.
1179 †Gay, John, 137 Upper Richmond Road, Putney, S.W.
7378 Gentle, James Alexander, 26 Colville Square, W.
937 Gibbard, Lieut.-Col. Thomas Wykes, 49 Prince of Wales Mansions, Battersea Park, S.W.
3654 Gibbon, Alfred Hugh, 4 Duke Street, Manchester Square, W.
7101 Gibbons, Robert Alexander, 29 Cadogan Place, S.W.
1902 †Gibbs-Smith, Edward Gibbs, 24 Alexander Square, South Kensington, S.W.
1094 †Giffard, Lieut.-Col. Gerald Godfray, c/o Grindlay and Co., 54 Parliament Street, S.W.
3696 Giles, Arthur, 10 Upper Wimpole Street, W.
1310 †Gill, Richard, 17 Albert Hall Mansions, Kensington Gate, S.W.
7037 Gilliatt, William, 27 Welbeck Street, W.
7160 Gilmour, Richard Withers, St. Luke's Hospital, E.C.
6803 Gimlette, Lieut.-Col. George Hart Desmond, E.I.U.S. Club, 16 St. James's Square, S.W.
1202 Gladstone, Reginald J., 22 Regent's Park Terrace, N.W.
1292 †Goadby, Kenneth W., 46 Harley Street, W.
220 Godding, James, 56, Leadenhall Street, E.C.
1431 †Godlee, Sir Rickman John, 19 Wimpole Street, W.
620 Godson, Clement, 5M Montagu Mansions, Portman Square, W.
6770 Goil, Capt. Dwarka Prasad, 14 Tanza Road, Hampstead.
3583 Goldsbrough, Giles Forward, 82 Wimpole Street, W.
523 †Golla, Frederick, 46 Welbeck Street, W.

1072 GOODALL, EDWARD WILBERFORCE, Eastern Hospital, Homerton, N.E.
684 †GOODBODY, FRANCIS WOODCOCK, 6 Chandos Street, Cavendish Square, W.
3955 GOODE, CHRISTINA LOVE, Haywood, Finchley Road, Golder's Green, N.W.
1554 GOODHART, GORDON, 25 Portland Place, W.
2002 GOODHART, Sir JAMES F., 25 Portland Place, W.
941 †GOSSAGE, ALFRED MILNE, 54 Upper Berkeley Street, Portman Square, W.
1329 GOULD, ALFRED LESLIE PEARCE, 10 Queen Anne Street, Cavendish Square, W.
433 ‡GOULD, Sir ALFRED PEARCE, 10 Queen Anne Street, W.
1584 GOULD, ERIC LUSH PEARCE, 10 Queen Anne Street, W.
1047 GOULDESBROUGH, CLAUDE, 56 Welbeck Street, W.
836 GOW, ALEXANDER EDWARD, 50 Welbeck Street, W.
1819 GRACE, JOHN J., 38 Harley Street, W.
2092 GRAHAM, CECIL IRVING, 17 Upper Wimpole Street, W.
1283 GRAHAM, GEORGE, 12 Ladbroke Gardens, Notting Hill, W.
592 †GRANT, JAMES DUNDAS, 18 Cavendish Square, W.
1172 GRAY, ARCHIBALD MONTAGUE HENRY, 30 New Cavendish Street, W.
1006 †GRAY, H. TYRRELL, 60 Harley Street, W.
207 GRAYLING, ARTHUR, 145 Sunderland Road, Forest Hill, S.E.
145 GREENWOOD, Major (Jun.), Lister Institute, Chelsea Gardens, S.W.
7026 †GREER, WILLIAM WELLS, Lancaster Mansions, 9 Lancaster Gate, W.
3627 GREEVES, REGINALD AFFLECK, 53 Upper Brook Street, W.
193 †GRIFFITH, WALTER SPENCER ANDERSON, 96 Harley Street, W.
786 †GRIFFITH, WILLIAM, 1 Harley Street, W.
1151 GRIFFITHS, JOHN NEVILLE, 47B Welbeck Street, W.
1180 †GRIMSDALE, HAROLD, 3 Harley Place, Marylebone Road, N.W.
1846 GROGAN, A. GERTRUDE, 289 Camden Road, N.
2050 GRÜNBAUM, OTTO, 92 Portland Place, W.
807 GUTHRIE, LEONARD, 15 Upper Berkeley Street, W.

H

1617 HAARBLEICHER, E. B. MEAKIN, 28 Grove End Road, St. John's Wood, N.W.
601 HAIG, ALEXANDER, 7 Brook Street, Hanover Square, W.
3591 HAKE, Dr. HENRY WILSON, Westminster Hospital Medical School, Caxton Street, Westminster.
38 †HALL, FRANCIS DE HAVILLAND, 47 Wimpole Street, W.
3961 HALL, HELENA ADELAIDE, 101 Church Hill, Walthamstow, N.E.
86 †HALLIBURTON, WILLIAM DOBINSON, Church Cottage, 17 Marylebone Road, N.W.
1568 HAM, B. BURNETT, 43 Lithos Road, South Hampstead, N.W.
1692 †HAMER, WILLIAM HEATON, 55 Dartmouth Park Hill, N.W.
924 HAMILL, PHILIP, 16 South Parade, Bedford Park, W.
227 †HANDLEY, WILLIAM SAMPSON, 12 New Cavendish Street, W.
1999 HANKINS, H. G., 20 Ribblesdale Road, Hornsey, N.
1156 HANNAY, MAURICE G., 1 Duke Street, Manchester Square, W.
546 HARDY, HORATIO NELSON, 43 Chisholm Road, Croydon.
7372 HARDY, SYLVIA FOWLER, 1A Southampton Row, W.C.
1965 HARFORD, CHARLES FORBES, Livingstone College, Knott's Green, Leyton, E.

76 †Harley, Vaughan, 25 Harley Street, W.
3880 Harman, N. Bishop, 108 Harley Street, W.
735 †Harmer, William Douglas, 45a Weymouth Street, W.
1859 Harper, Cecil Randle, Brockley House, 239 Peckham Rye, S.E.
7359 Harper, Gerald, 40 Curzon Street, Mayfair, W.
1776 Harris, Col. George F. A., 69 Harcourt Terrace, S.W.
390 Harris, Wilfred, 73 Wimpole Street, W.
918 Harrison, Lawrence Whitaker, 77 Prince of Wales Mansions, Battersea Park, S.W.
3596 Harrison, Major William Sandelands, Royal Army Medical College, Grosvenor Road, S.W.
382 Harrower, Henry R., "Alarneda," 20 Ribblesdale Road, Hornsey, N.
2053 Hartley, Percival, Lister Institute, Chelsea Gardens, S.W.
3881 Harvey, Capt. G. A. Duncan, Grosvenor Road, S.W.
1975 Harvey, John Owen, 33 Campden House Road, Kensington, W.
1044 Harwood-Hardman, Frederick, 16 College Court, Hammersmith, W.
1124 Haslam, Kate, 10 Warltersville Road, Crouch Hill, N.
1710 Haslip, G. E., 8 Suffolk Place, Pall Mall, S.W.
938 Hastings, Somerville, 43 Devonshire Street, Portland Place, W.
3815 Haughton, Edward, Spring Grove House, Upper Norwood, S.E.
2072 †Havelock-Charles, Sir Richard, 9 Manchester Square, W.
205 Hawkins, Herbert P., 56 Portland Place, W.
1939 Hawthorne, C. O., 63 Harley Street, W.
1938 Hawthorne, Janie Lorimer, 63 Harley Street, W.
6775 Haydon, Arthur George, 23 Henrietta Street, Cavendish Square.
1957 †Hayman, Albert Stephen, 20 Thurloe Place, S.W.
683 †Head, Henry, 4 Montagu Square, Hyde Park, W.
1482 Heath, Charles Joseph, 34 Devonshire Place, W.
842 Hebb, R. G., 50 Ridgmount Gardens, Gower Street, W.C.
1529 Hedley, E. W., 56 Hans Place, S.W.
265 †Hedley, John Prescott, 11 John Street, Berkeley Square, W.
2154 Henderson, Edward Erskine, 6 Lower Berkeley St., Portman Sq., W.
1480 †Hendley, Col. Thomas Holbein, 4 Loudoun Road, N.W.
984 Herman, C. Lawrence, c/o Standard Bank of South Africa, 10 Clement's Lane, Lombard Street, E.C.
447 Hern, George, 7 Stratford Place, Oxford Street, W.
425 ‡Hern, William, 7 Stratford Place, Oxford Street, W.
1219 †Herring, Herbert T., 50 Harley Street, W.
972 Herringham, Wilmot Parker, 40 Wimpole Street, W.
462 †Hertz, Arthur F., 1 Weymouth Street, W.
1781 †Hewitt, Fleet-Surgeon David Walker, Medical Dept., Admiralty, London.
1028 Hewlett, Richard Tanner, 12 Colinette Road, Putney, S.W.
2173 Hey, Dr. C. Granville, 138 Earl's Court Road, Kensington, W.
1038 Hicks, John Athelstan Braxton, 48 Queen Anne Street, Cavendish Square, W.
1969 Hicks, Sydney, 30 Bennett Road, Blackheath, S.E.
6551 Higgins, T. Shadick, Town Hall, Pancras Road, N.W.
6766 Hill, Walter, 45b Connaught Square, W.
868 Hill, William, 26 Weymouth Street, Portland Place, W.
3665 Hogg, Arthur J., 30 Haven Green, Ealing, W.
169 Holland, Eardley, 55 Queen Anne Street, W.
377 Holmes, Gordon M., 58 Harley Street, W.
7120 Holt, Arthur, 50 Shell Road, Lewisham, S.E.

1667 HOMER, ANNIE, Lister Institute, Chelsea Gardens, S.W.
1671 HOOD, DONALD W., Green Street, Park Lane.
894 HOPE, CHARLES WILLIAM MENELAUS, 58 Wimpole Street, W.
813 HOPEWELL-SMITH, ARTHUR, 58 Park Street, Park Lane, W.
756 HORNE, WALTER JOBSON, 23 Weymouth Street, Portland Place, W.
1063 HORSFORD, CYRIL, 41 Wimpole Street, W.
1685 HORSLEY, Sir VICTOR, 25 Cavendish Square, W.
7383 HORSMAN, GODFREY C., 22 King Street, Portman Square, W.
6596 HOSFORD, JOHN STROUD, 33 St. James's Place, S.W.
1771 HOWARTH, WALTER GOLDIE, 109 Harley Street, W.
975 †HOVELL, T. MARK, 105 Harley Street, Cavendish Square, W.
410 †HOWELL, CONRAD MEREDYTH HINDS, 53 Queen Anne Street, Cavendish Square, W.
2030 HOWELL, T. A. IVES, Oriel Lodge, Upper Richmond Road, Putney, S.W.
1883 HOWITT, ALFRED BAKEWELL, 18 Chapel Street, Belgrave Square, S.W.
1767 HUDSON, ARTHUR CYRIL, 50 Queen Anne Street, W.
6774 HUGHES, E. C., 37 Queen Anne Street, Cavendish Square, W.
1081 HUMPHRIS, FRANCIS HOWARD, 8 West Chapel Street, Mayfair, W.
7429 HUNT, EDWIN GUY, 71 Charlwood Street, S.W.
1155 HUNTER, HOWARD TOMLIN, 30 St. Petersburgh Place, Bayswater, W.
751 †HUNTER, WILLIAM, 103 Harley Street, W.
1576 HURTLEY, W. H., 122 Christchurch Road, Tulse Hill, S.W.
316 HUTCHINSON, F. H. G., c/o Messrs. H. S. King and Co., 9 Pall Mall, S.W.

I

1604 †IBBOTSON, WILLIAM, 18 Weymouth Street, Portland Place, W.
7047 INGEN-WINTER, ALICE VAN, 41 Willoughby Road, Hornsey, N.
1814 †INMAN, ARTHUR CONYERS, The Laboratory, Brompton Hospital, Fulham Road, S.W.
7098 INSTONE, BRADNEY, 68 Addison Road, Kensington, W.
461 IRONSIDE, ARTHUR EDMUND, 44 Devonshire Street, Portland Place, W.

J

3671 JACKSON, J. CHARLES, Town Hall, Fulham, S.W.
1550 †JAFFE, CHARLES SAMSON, 138 Sutherland Avenue, Maida Vale.
1923 †JAMES, Col. WALTER CULVER, 15 Marloes Road, Kensington, W.
875 JAMES, HERBERT ELLISON RHODES, 5 Wilbraham Place, Sloane St., S.W.
37 JAMES, WILLIAM WARWICK, 2 Park Crescent, W.
1991 JEFFERSON, GEOFFREY, The Cancer Hospital (Free), Fulham Rd., S.W.
437 JENKINS, GEORGE J., 48 Wimpole Street, W.
1477 †JENKINS, JOHN, Hammersmith Infirmary, Shepherd's Bush.
670 ‡JESSOP, WALTER HAMILTON HYLTON, 73 Harley Street, W.
1507 JEVONS, M. SOPHIA, 10 Hamilton House, Hall Road, St. John's Wood, N.W.
1810 JEWELL, WILLIAM HENRY, 27 New Cavendish St., Cavendish Sq., W.
3678 †JEWESBURY, REGINALD CHARLES, 5 Wimpole Street, W.
1840 JEX-BLAKE, A. J., 13 Ennismore Gardens, S.W.
2063 †JOHN, WILLIAM, 24 Belsize Park Gardens, N.W.
502 †JOHNSON, ALFRED EDWARD, 35 Grosvenor Street, W.
75 ‡JOHNSON, RAYMOND, 11 Wimpole Street, W.

1673 JOHNSON, WILLIAM, Pathological Department, Guy's Hospital, S.E.
1084 †JOHNSTON, GEORGE FRANCIS, 23 Seymour Street, Portman Square, W.
1527 †JOHNSTON, GERALD HERBERT, Brooke House, Clapton, N.E.
151 †JOLY, JOHN SWIFT, 48 Harley Street, W.
1871 JONA, J. LEON, Lister Institute, Chelsea, S.W.
1252 JONES, ARTHUR ROCYN, Royal National Orthopædic Hospital, Great Portland Street, W.
1562 JONES, CECIL PRICE, Cancer Research Laboratory, Middlesex Hospital.
1013 JONES, DUDLEY WILLIAM CARMALT, 78 Wimpole Street, W.
3814 †JONES, Col. G. C., Savoy Mansions, W.C.
7394 JONES, HENRY LEWIS, 143 Harley Street, W.
6809 JONES, L. VERNON, 7 Arlington Street, St. James's, S.W.
1454 JONES, Staff-Surgeon K. H., c/o G.P.O., London.
1042 †JORDAN, ALFRED C., 11 Bentinck Street, W.
566 †JULER, FRANK ANDERSON, 24 Cavendish Square, W.
7063 ‡JULER, HENRY E., 23 Cavendish Square, W.
1020 JUST, THEODORE HARTMANN, St. Bartholomew's Hospital, E.C.

K

3653 KARANJIA, KHURSEDJI N., 208 Upper Clapton Road, N.E.
1273 KEITH, A., Royal College of Surgeons, Lincoln's Inn Fields, W.C.
3987 KELLOCK, THOMAS H., 2 Upper Wimpole Street, Cavendish Square, W.
1201 KELSON, W. H., 17 Cavendish Place, W.
1261 KELYNACK, THEO. N., 139 Harley Street, W.
1133 KENNAWAY, ERNEST LAWRENCE, Guy's Hospital, S.E.
1129 KENNEDY, Major JAMES CRAWFORD, R.A.M. College, Grosvenor Road, S.W.
1956 KERR, JAMES, 15 Hanger Lane, Ealing, W.
370 KETTLE, EDGAR HARTLEY, 36 Addison Gardens, Kensington, W.
7031 KEYNES, GEOFFREY, 38 Brunswick Square, W.C.
1069 †KIDD, FRANCIS SEYMOUR, 55 Harley Street, W.
1752 KING, C. A., 22 Dalebury Road, Upper Tooting, S.W.
771 †KING, DAVID BARTY, 49 Hill Street, Berkeley Square, W.
1417 KINGSFORD, BERESFORD, 19 Upper George Street, Bryanston Sq., W.
2006 †KISCH, ALBERT, 3D Bickenhall Mansions, Gloucester Place, Portman Square, W.
2010 KITTELSEN, NILS, 137 Denmark Hill, S.E.
1321 KNOX, ROBERT, 7 Harley Street, W.
1996 KRISHNASWAMI-IYER, E. S., 95 Brook Green, W.

L

3621 LACK, H. LAMBERT, 16 Devonshire Place, W.
3584 LAKE, RICHARD, 60 Harley Street, W.
1815 LANE, JAMES ERNEST, 47 Queen Anne Street, Cavendish Square, W.
90 ‡LANE, Sir WILLIAM ARBUTHNOT, 21 Cavendish Square, W.
851 LANE-CLAYPON, JANET, Local Government Board, Whitehall, S.W.
6834 LANG, B. THORN, 22 Cavendish Square, W.
1370 LANGMEAD, FREDERICK S., 53 Queen Anne Street, Cavendish Square, W.
1498 LANKESTER, A. OWEN, 5 Upper Wimpole Street, W.
7134 †LAUZUN-BROWN, WILLIAM, 297 Goswell Road, E.C.
1100 †LAW, EDWARD, 8 Wimpole Street, W.
420 †LAWFORD, JOHN BOWRING, 99 Harley Street, W.

6757 LAWRENCE, LAURIE ASHER, 32 Devonshire Place, W.
440 LAWSON, ARNOLD, 12 Harley Street, W.
1215 LAYTON, T. B., 48 Queen Anne Street, Cavendish Square, W.
1297 LAZARUS-BARLOW, WALTER SYDNEY, Middlesex Hospital, W.
215 LEAHY, Lieut.-Col. ALBERT WILLIAM DENIS, 4 Iddesleigh Mansions, Victoria Street, S.W.
1607 †LEDINGHAM, J. C. G., Lister Institute, Chelsea Gardens, S.W.
2013 †LEES, DAVID BRIDGE, 22 Weymouth Street, Portland Place, W.
4544 LEESER, FELIX, German Hospital, Dalston, N.E.
6849 LEGG, T. P., 139 Harley Street, W.
7400 LEIPER, ROBERT THOMSON, 103 Corringham Road, Golder's Green, N.W.
1705 LEISHMAN, Col. Sir WILLIAM, Royal Army Medical College, Grosvenor Road, S.W.
1742 LEITCH, ARCHIBALD, 11 Overstrand Mansions, Battersea Park, S.W.
3968 ‡LENNANE, VINCENT, St. John's, Queen's Road, Battersea Park.
1408 LEPPER, ELIZABETH HERDMAN, 42 Manor House, Marylebone Road, N.W.
1287 †LESLIE, ROBERT MURRAY, 152 Harley Street, W.
691 LETT, HUGH, 8 Lower Berkeley Street, W.
241 †LEVY, AARON HAROLD, 67 Wimpole Street, W.
1648 LEVY, ALFRED GOODMAN, 2 Manchester Square, W.
131 LEWIS, THOMAS, 27 Queen Anne Street, W.
3965 LEWKOWITSCH, HUGO, Esperanza, Meadway, Hampstead Garden Suburb, N.W.
1992 LEWTAS, Lieut.-Col. J., 15 Cheyne Place, S.W.
1380 LINDSAY, LIONEL M., 8 Radnor Place, Hyde Park, W.
396 †LISTER, THOMAS DAVID, 31 Harley Street, W.
1832 LISTER, WILLIAM TINDALL, 24 Devonshire Place, W.
1627 LITTLE, ERNEST GRAHAM, 61 Wimpole Street, W.
788 †LITTLE, ERNEST MUIRHEAD, 40 Seymour Street, Portman Square, W.
1777 LLOYD, RICHARD WILLIAM, 103 Oakwood Court, Kensington, W.
935 LLOYD-WILLIAMS, EDWARD, 11 Buckingham Gate, S.W.
1670 LOCKYER, CUTHBERT, 117A, Harley Street, W.
1123 LONG, CONSTANCE E., 2 Harley Place, W.
623 LONNON, FREDERICK, 77 Denmark Hill, S.E.
1119 †LOTZ, HENRY JOHN, "Arnwood," 22 Lichfield Road, Kew Gardens, S.W.
192 LOW, GEORGE CARMICHAEL, 6 Bentinck Street, W.
707 LOW, HAROLD, 10 Eveyln Gardens, S.W.
1658 LOW, ROBERT BRUCE, 98 York Mansions, Battersea Park, S.W.
721 LOW, VINCENT WARREN, 146 Harley Street, W.
6836 LUCAS, RICHARD CLEMENT, 30 Wimpole Street, W.
653 LUKIS, THEODORE STEWART, Toynbee Hall, Commercial Street, E.
1737 LUSH, PERCY JOHN FREDERICK, 4 Maresfield Gardens, Hampstead, N.W.
1804 LYNCH, MAURICE EDMUND, 80 Antrim Mansions, Haverstock Hill, N.W.
853 LYNDEN-BELL, E. H., Horse Guards, S.W.
2082 †LYON-SMITH, HARRY, 2 Bentinck Street, Manchester Square, W.
1829 LYSTER, CECIL R. CHAWORTH, 70 Wimpole Street, W.

M

1825 MACALISTER, GEORGE HUGH KIDD, 85 Prince of Wales Mansions, Battersea Park, S.W.
783 MACALISTER, J. Y. W., 1 Wimpole Street, W.
345 ‡MCCALL, Sir JOHN, 30 Nevern Mansions, Warwick Road, S.W.
638 †MCCANN, FREDERICK JOHN, 14 Wimpole Street, W.

3843 †MacCarthy, E. F. Talbot, 124 Gower Street, W.C.
6823 McCleary, George Frederick, Wellington House, Buckingham Gate, S.W.
1192 †McClure, Henry, 30 Portland Court, W.
¶162 MacCormac, Henry, 15 Harley Street, W.
776 McCulloch, Henry Daniel, 152 Harley Street, W.
330 †McDonagh, J. E. R., 4 Wimpole Street, W.
¶144 MacDonald, Greville, 85 Harley Street, W.
1925 MacDonald, Isabella M., 47 Seymour Street, Portman Square, W.
130 MacDonald, Sydney Gray, 27 Wimpole Street, W.
¶519 McDonnell-Kelly, W., 15 High Road, South Tottenham, N.
¶260 McFadyean, Sir John, Royal Veterinary College, Camden Town, N.W.
421 McKay, Robert, 11 Queen Anne Street, W.
1619 MacKeith, John, 15 Cavendish Place, Cavendish Square, W.
1806 Mackenzie, James, 133 Harley Street, W.
1587 ‡McKenzie, Dan, 62 Brook Street, W.
7125 †Mackinnon, Murdoch, 93 Cromwell Road, S.W.
1857 MacLean, Hugh, 68 Overstrand Mansions, Battersea Park, S.W.
1578 MacLennan, S. J. (of Canada), c/o Royal Bank of Canada, Princes Street, E.C.
1196 MacLeod, J. M. H., 11 Harley Street, W.
¶893 McLeod, James Walter, 47 South Hill Park, Hampstead, N.W.
603 †McMullen, William Halliburton, 4 Chandos Street, Cavendish Square, W.
976 McNabb, Fleet-Surgeon Daniel Joseph Patrick, Medical Department, The Admiralty, Whitehall, S.W.
595 †Macnamara, Eric Danvers, 54 Welbeck Street, W.
519 †McNamara, John, 3 Holland Road, Kensington, W.
1361 †Macnaughton-Jones, H., 111 Harley Street, W.
999 Macphail, Alexander, 2 Harley Place, N.W.
1508 Madge, Edward D., 11 Mount Street, Grosvenor Square, W.
6242 Magathaes, Joaquin dos Reis, 20 Copthall Avenue, E.C.
52 †Maggs, William Adolphus, 14 Upper Wimpole Street, W.
3626 Maitland, Pelham C., 17 Gwendwr Road, West Kensington, W.
194 †Makins, George H., 45 Upper Brook Street, W.
1820 Malabari, Phiroza B., City House Hotel, 158 City Road, E.C.
701 †Malcolm, John D., 33 Wimpole Street, W.
3980 Manuel, Alexander, 1 Harley Street, W.
1150 Mark, Leonard Portal, 49 Oxford Terrace, Hyde Park, W.
1968 Marriage, Herbert James, 109 Harley Street, Cavendish Square, W.
3970 Marshall, Geoffrey, 38 St. Mary Abbott's Terrace, Kensington, W.
7438 Martin, Edward Kenneth, 10 Harley Street, W.
1514 †Martin, Charles J., Lister Institute, Chelsea Gardens, S.W.
1114 Martin, W. L., Medical Department, Admiralty, S.W.
6240 Maruyama, Surg.-Major T., 31, Wemyss Road, Blackheath, S.E.
1638 †Masters, Major John Alfred, 94 Knightsbridge, S.W.
3964 †Matthews, Arthur Kenward, 159 Lancaster Road, Kensington, W.
775 Matthews, Guy, 22 Suffolk Street, Pall Mall, S.W.
774 †Matthews, Valentine, 22 Suffolk Street, Pall Mall, S.W.
2036 †Maughan, James, 56 Albany Street, N.W.
741 †May, Surg.-General Arthur William, Medical Department, Admiralty, Whitehall, S.W.
1286 Mayer, Ernest, 25 Hamilton Road, Ealing, W.
1241 †Meachen, George Norman, 11 Devonshire Street, Portland Place, W.

1171 MEHTA, N. M., 79 Linden Gardens, Bayswater, W.
1399 †MELANDRI, FILIPPO, 18 Gower Street, Bedford Square, W.C.
7119 MELLANBY, EDWARD, St. Thomas's Hospital, S.W.
1216 MELVILLE, STANLEY, 10 Welbeck Street, W.
723 †MENNELL, JAMES BEAVER, 1 Royal Crescent, W.
1652 MENNELL, ZEBULON, 8 Hyde Park Terrace, W.
3913 MENZIES, JAMES HERBERT, 10 Wetherby Place, South Kensington.
1204 †METCALFE, JAMES, 123 Harley Street, W.
1437 MEYER, Lieut.-Col. CHARLES HARDWICK LOUIS, 4 Queen's Gate, S.W.
1623 MICHELS, G., 48 Wimpole Street, W.
1738 MILES, W. ERNEST, 16 Upper Wimpole Street, W.
6593 MILLIGAN, WYNDHAM A., 11 Upper Brook Street, W.
260 †MILLS, HERBERT HENRY, 21 St. Mary Abbott's Terrace, Kensington.
2023 MILLS, WESLEY, 45 Warrington Crescent, Maida Vale, W.
1035 MILNE, ROBERT, 75 Windsor Road, Forest Gate, E.
2061 MILNE, ROBERT, 29 Weymouth Street, Portland Place, W.
778 MINCHIN, Prof. E. A., 53 Cheyne Court, Chelsea, S.W.
997 MITRA, S. M., 47 Warrington Crescent, Maida Vale, W.
264 †MIVART, FREDERICK ST. GEORGE, 13 Stafford Terrace, Kensington, W.
3647 MODY, R. D. P., 216 Stockwell Road, Brixton, S.W.
3873 MÖLLER, Dr. THEODOR, Cleveland House, St. James's Square, S.W.
1994 MOLLISON, W. M., 9 Upper Brook Street, W.
1763 MONCKTON, R. V. G., 1 Emperor's Gate, S.W.
6137 MONRAD-KROHN, G. H., 29 Pennard Road, Shepherd's Bush, W.
640 MONTUSCHI, FREDERIC G., 106 Shaftesbury Avenue, W.
936 MOON, ROBERT OSWALD, 45 Green Street, Park Lane, W.
1426 MOORE, ALAN, 94 Gloucester Place, Portman Square, W.
3681 MOORE, IRWIN, 30A Wimpole Street, W.
398 †MOORE, NORMAN, 94 Gloucester Place, Portman Square, W.
7024 MORETON, ADRIAN LEONARD, 197 Albany Street, N.W.
1238 MORGAN, JOHN HAMMOND, 3 Connaught Square, W.
6760 MORGAN, WILLIAM PARRY, 9 Stafford House, Maida Hill West, W.
469 ‡MORISON, ALEXANDER, 14 Upper Berkeley Street, W.
1019 MORISON, ARCHIBALD ALEXANDER, Brook Hospital, Shooters Hill, Woolwich, S.E.
674 †MORISON, BASIL GORDON, 115 Green Lanes, Highbury, N.
3995 MORISON, JOHN, 1 Jackson's Lane, Highgate, N.
1841 MORLEY, ARTHUR SOLOMON, 52 Gordon Square, W.C.
1332 MORRIS, Sir HENRY, Bart., 8 Cavendish Square, W.
1203 †MORRIS, MALCOLM, 8 Harley Street, W.
1448 MORSON, ALBERT CLIFFORD, 43 Queen Anne Street, Cavendish Sq., W.
1185 MORTIMER, JOHN DESMOND, 15 St. Leonard's Terrace, Sloane Sq., S.W.
801 †MORTON, REGINALD, 66 Harley Street, W.
1639 †MOTT, F. W., 25 Nottingham Place, W.
759 MOXON, FRANK, 56 Seymour Street, Portman Square, W.
3578 MULLALLY, GERALD THOMAS, Guy's Hospital, S.E.
1090 MURPHY, Sir SHIRLEY, 9 Bentinck Terrace, Regent's Park, N.W.
6835 MURRAY, FLORA, 60 Bedford Gardens, Campden Hill, W.
289 MURRAY, JAMES ALEXANDER, Imperial Cancer Research Fund, 8-11 Queen Square, W.C.
3643 †MURRAY, JESSIE M., 14 Endsleigh Street, Tavistock Square.
203 MURRELL, CHRISTINE M., 86 Porchester Terrace, Hyde Park, W.
1255 MUTCH, NATHAN, 8 St. Thomas Street, Southwark, S.E.

N

1694 NEATBY, EDWIN A., 82 Wimpole Street, W.
848 †NEWSHOLME, ARTHUR, 112 Ashley Gardens, S.W.
7159 NEWTON, CHARLES JOHN, 5 The Grove, Hammersmith.
2062 †NIAS, J. B., 5 Rosary Gardens, S.W.
1978 NICHOLS, C. H. F., "The Evening News," Carmelite House, E.C.
1326 NICHOLSON, G. W., Cancer Hospital Research Institute, Brompton, S.W.
22 †NITCH, CYRIL ALFRED RANKIN, 69 Harley Street, W.
212 ‡NIX, EDWARD JAMES, 11 Weymouth Street, W.
708 NOBEL, HUGH PERCY, 24A Portland Place, W.
7363 †NOBLE, WILLIAM GEORGE, 851 High Road, Leyton, N.E.
891 NORBURY, LIONEL EDWARD CLOSE, 49 Wimpole Street, Cavendish Square, W.
1523 NORRIS, WILLIAM PERRIN, 72 Victoria Street, Westminster, S.W.
597 †NOURSE, WILLIAM JOHN CHICHELE, 20 Weymouth Street, W.
423 †NOWELL, WALTER SALMON, 4 Seymour Street, Portman Square, W.

O

1438 O'BRIEN, R. A., 100 Prince of Wales Mansions, Battersea Park, S.W.
3663 O'CONNOR, F. W., London School of Tropical Medicine, Albert Docks, E.
600 †OGILVIE, GEORGE, 22 Welbeck Street, W.
230 OGLE, CYRIL, 96 Gloucester Place, Portman Square, W.
1879 OLIVER, M. W. B., 67 Wimpole Street, Cavendish Square, W.
758 OLIVER, WILLIAM JENKINS, London Hospital, E.
392 O'MALLEY, JOHN F., 16 Weymouth Street, W.
1034 †OPENSHAW, THOMAS HORROCKS, 16 Wimpole Street, W.
1856 OPPENHEIMER, HEINRICH, 19 College Crescent, Fitzjohn's Avenue, N.W.
388 ‡ORMEROD, JOSEPH ARDERNE, 25 Upper Wimpole Street, Cavendish Square, W.
543 †ORMOND, ARTHUR WILLIAM, 7 Devonshire Place, W.
705 †ORR, VIVIAN B., 28 Lowndes Street, S.W.
1059 †ORTON, GEORGE HARRISON, 67 Upper Berkeley St., Portman Square, W.
3981 OWEN, EDMUND, 24 Upper Berkeley Street, Portman Square.
449 OWEN, SYDNEY A., 15 Queen Anne Street, W.

P

1251 PAGE, CHARLES MAX, 48 Welbeck Street, W.
713 PAGE, HARRY MARMADUKE, 53 Welbeck Street, W.
7375 PAGE, HERBERT W., 146 Harley Street, W.
1779 PAINE, ALEXANDER, The Cancer Hospital Research Institute, Brompton, S.W.
149 ‡PALMER, FREDERICK STEPHEN, 27 Queen Anne Street, W.
6569 PALMER, HARRY, 65 Alexandra Road, St. John's Wood, N.W.
1869 †PARDOE, JOHN, 77 Wimpole Street, W.
6755 PARKER, CHARLES ARTHUR, 24 Upper Wimpole Street, W.
591 PARKINSON, JOHN, London Hospital, Whitechapel, E.
1197 †PARSONS, CHRISTOPHER THACKERAY, Fulham Infirmary, Hammersmith, W.
1379 PARSONS, F. G., St. Thomas's Hospital, S.E.
700 PARSONS, JOHN HERBERT, 54 Queen Anne Street, Cavendish Square, W.

1813 PARSONS, RICHARD A., 52 Wellington Road, St. John's Wood, N.W.
2012 PART, JOHN SHEPLEY, 30 Bath Road, Bedford Park, W.
1076 ‡PASTEUR, WILLIAM, 4 Chandos Street, Cavendish Square, W.
318 †PATERSON, HERBERT J., 9 Upper Wimpole Street, W.
1512 PATERSON, WILLIAM BROMFIELD, 7A Manchester Square, W.
364 †PATON, LESLIE JOHNSTON, 29 Harley Street, W.
1098 PATTERSON, NORMAN, 42 Brook Street, W.
1141 PATTERSON, SYDNEY WENTWORTH, 64 Torrington Square, W.C.
779 PAYNE, JOSEPH LEWIN, 18 Portland Place, W.
3776 PEARSON, STELLA MESSINGER, 95 Guilford Street, W.C.
3685 PEART, JOSEPH FREDERICK, 10 Harley Street, W.
1440 †PEGLER, L. HEMINGTON, 58 Harley Street, W.
7140 PENBER, Fleet-Surgeon FREDERICK W., Junior United Service Club, Regent Street, W.
3635 PENFOLD, WILLIAM JAMES, 25 Melrose Avenue, Wimbledon Park, S.W.
1444 PENNELL, ALICE MAUD, 26 Russell Square, W.C.
7115 PENNY, Lieut.-Col. J., c/o Grindlay and Co., 54 Parliament Street, S.W.
338 PERNET, GEORGE, 20 Devonshire Place, W.
3652 PERRY, HUGH HAMILTON, 3 Upper Bedford Place, W.C.
2022 PETRIE, GEORGE F., Lister Institute, Chelsea Gardens, S.W.
3826 PETTY, M. J., St. Thomas's Hospital, S.E.
7145 PHILLIPS, C. EDMUND STANLEY, Cancer Hospital, Brompton, S.W.
79 PHILLIPS, SIDNEY PHILIP, 3 Upper Brook Street, Grosvenor Square, W.
2193 PINCH, ALBERT EDWIN HAYWARD, The Radium Institute, Langham Place, W.
463 †PINTO-LEITE, HUBERT, 24 Fellows Road, N.W.
3775 PIRIE, ALEXANDER HOWARD, 133 Harley Street, W.
1985 PISANI, L. J., 49 Wimpole Street, W.
1031 †PITT, GEORGE NEWTON, 15 Portland Place, W.
1231 PITTS, ARTHUR T., 26 New Cavendish Street, W.
1768 PLAYFAIR, ERNEST, 57 Gloucester Terrace, Hyde Park.
730 †PLAYFAIR, HUGH, 7 Upper Brook Street, W.
1862 POLLARD, BILTON, 24 Harley Street, W.
1719 POLLARD, JOSEPH, 51 Queen Anne Street, Cavendish Square, W.
755 POLLOCK, C. E., War Office, Whitehall, S.W.
1664 PORTER, AGATHA, 18 Kensington Park Road, Notting Hill Gate, W.
1616 POTTER, BERNARD, 58 Park Street, Park Lane, W.
6587 POWELL, H. FITZGERALD, 5 Harley Street, W.
3783 POWELL, Sir RICHARD DOUGLAS, 11B Portland Place, W.
964 POWELL, W. WYNDHAM, 75 Wimpole Street, W.
56 †POWER, D'ARCY, 10A Chandos Street, Cavendish Square, W.
2005 †POYNTON, F. J., 35 Devonshire Place, W.
632 PRASAD, Lieut.-Col. KANTA, 47 Barton Street, West Kensington.
402 †PRATT, Lieut.-Col. JAMES JOHN, 54 Parliament Street, S.W.
1807 PRICE, FREDERICK W., 133 Harley Street, W.
1805 PRICKETT, MARMADUKE, 20 Fitzgeorge Avenue, West Kensington.
1388 PRIDMORE, Lieut.-Col. WALTER GEORGE, c/o Grindlay and Co., 54 Parliament Street, S.W.
3823 PRIESTLEY, Capt. H. E., 102 Oxford Gardens, North Kensington.
1827 PRIESTLY, JOHN GILLIES, 3 Buckingham Gate, S.W.
35 PRINGLE, JOHN JAMES, 23 Lower Seymour Street, W.
1743 PRITCHARD, ERIC, 63 Harley Street, W.
1056 ‡PRITCHARD, URBAN, 26 Wimpole Street, W.
787 †PROBYN-WILLIAMS, ROBERT JAMES, 13 Welbeck Street, W.

R

3984 RAFFAN, GEORGE, 128 Harley Street, W.
1620 RAJCHMAN, LUDWIK, 16 Hartswood Road, Stamford Brook Road, W.
1644 RAU, B. K. NARAYANA, 116 South Hill Park, Hampstead, N.W.
1621 RAWES, WILLIAM, St. Luke's Hospital, Old Street, E.C.
3954 RAWLING, L. BATHE, 16 Montagu Street, Portman Square, W.
2091 RAYNER, HENRY, 16 Queen Anne Street, W.
2070 REANEY, Capt. M. FOSTER, 59 Coleraine Road, Blackheath, S.E.
973 †REID, ARCHIBALD DOUGLAS, 30 Welbeck Street, W.
1213 REID, Sir JAMES, 72 Grosvenor Street, W.
660 †RELPH, HERBERT J., 24 Wimpole Street, W.
2011 REYNOLDS, AUSTIN EDWARD, 15 Finsbury Circus, E.C.
2189 REYNOLDS, Dr. BRYAN ELLIS, Chedworth, Orleans Rd., Hornsey Lane.
7352 REYNOLDS, JOHN, 11 Brixton Hill, S.W.
7353 REYNOLDS, RUSSELL JOHN, Rossendals Lodge, 17 Streatham Hill, S.W.
373 †RHODES, HERBERT, 30 Brechin Place, South Kensington, S.W.
1229 RIAD, MAHMOUD, Royal London Ophthalmic Hospital, City Road, E.C.
1785 RIGBY, HUGH MALLINSON, 24 Queen Anne Street, W.
470 RILOT, CHARLES FREDERICK, 22 Wimpole Street, W.
1293 †RIVIERE, CLIVE, 19 Queen Anne Street, W.
406 ROAF, HERBERT ELDON, 44 Rotherwick Road, Golder's Green, N.W.
925 ROBERTS, JAMES ERNEST HELME, 15 Devonshire Place, W.
7078 †ROBINSON, ARTHUR HENRY, St. Mary's Infirmary, Highgate Hill, N.
7434 †ROBINSON, Surg.-Major EDMUND FIELD, 26 Bedford Place.
904 †ROBINSON, HENRY BETHAM, 1 Upper Wimpole Street, W.
1936 ROBINSON, Lieut.-Col. OLIVER L., Royal Army Medical College, Millbank, S.W.
1039 †ROBINSON, Lieut.-Col. W. H. B., 5 Cleveland Road, Ealing.
3645 †ROBISON, ROBERT, 78 Primrose Mansions, Battersea Park.
1284 †ROLLESTON, HUMPHRY DAVY, 55 Upper Brook Street, W.
1091 ROLLESTON, J. D., Grove Hospital, Tooting, S.W.
7401 ROOME, HENRY A., 58 Harley Street, W.
2038 ROSHER, ARTHUR BURCH, Charing Cross Hospital, W.C.
170 ROSS, ALEXANDER, 56 Maida Vale, W.
174 ROSS, HUGH C., McFadden Research Fund, Lister Institute, Chelsea, S.W.
279 ‡ROTH, BERNARD, 38 Harley Street, W.
298 †ROTH, PAUL BERNARD, 38 Harley Street, W.
1921 ROUND, JOHN CORNWELL, Purbrook, Crescent Wood Road, Sydenham Hill, S.E.
1812 ROUSE, ROLLA EDWARD, 126 Harley Street, W.
92 †ROUTH, AMAND J., 14A Manchester Square, W.
332 †ROUTH, LAURENCE MELVILLE, 39 Upper George Street, Bryanston Square, W.
277 ROUW, ROBERT WYNNE, 8 Upper Wimpole Street, W.
1821 ROWELL, GEORGE, 6 Cavendish Place, Cavendish Square, W.
1077 ROWLANDS, RICHARD ALUN, London Hospital, E.
1800 †ROWLANDS, ROBERT PUGH, 12 Queen Anne St., Cavendish Square, W.
3911 RUPP, KARL, 86 London Road, Forest Hill, S.E.
3906 RUSS, SIDNEY, Cancer Research Laboratories, Middlesex Hospital, W.
247 RUSSELL, ALFRED ERNEST, 7 Upper Wimpole Street, W.
290 RUSSELL, BERTIE RONALD GORDON, Imperial Cancer Research Fund, 8-11 Queen Square, W.C.

736 ‡Russell, J. S. Risien, 44 Wimpole Street, W.
7100 Russell, Lieut.-Col. Michael William H., Horse Guards, Whitehall, S.W.
3786 †Rutherford, Alexander Hamilton, 18 Birchin Lane, Lombard Street, E.C.
7416 Rutherford, Norman Cecil, Frith Manor, Mill Hill, N.W.
2096 ‡Ryall, Charles, 62 Harley Street, W.
7104 †Ryall, Edward Canny, 77 Harley Street, W.
3837 †Ryan, Edward, Imperial Hotel, Russell Square, W.C.
1460 Ryffel, John Henry, Guy's Hospital, S.E.
1585 Ryland, Archer, Central London Throat and Ear Hospital, Gray's Inn Road, W.C.

S

917 †Sachs, Alfred L., 62 Wimpole Street, W.
3648 Sadler, Samuel Champernowne, Church House, 9-11 Dean's Yard, Westminster, S.W.
3818 Saigol, Capt. Raghuber Dayal, 80 Gower Street, W.C.
3887 Sambon, Louis W., 68 Shoot-up Hill, N.W.
2058 Sanders, Charles, 29 Windsor Road, Forest Gate, E.
1339 †Sandwith, Fleming Mant, 31 Cavendish Square, W.
2169 Saner, John Godfrey, The Warden's House, Guy's Hospital, E.C.
1559 Sanguinetti, Harold H., 19 Campden House Road, Kensington, W.
374 Sargent, Percy, 20 Harley Street, W.
7711 Saunders, Percy W., 63 Wimpole Street, W.
1237 †Savage, Arthur Harold, c/o Sir George Savage, 26 Devonshire Place, W.
1032 Savage, Sir George Henry, 26 Devonshire Place, W.
7065 Sayer, Ettie, 35 Upper Brook Street, W.
1487 †Scharlieb, Herbert J., 49 Wimpole Street, W.
4 Scharlieb, Mary Ann Dacomb, 149 Harley Street, W.
3602 Scharlieb, Mary Ethel, 149 Harley Street, W.
1581 Schlesinger, Edward G., The College, Guy's Hospital.
1526 Schryver, S. B., The Cancer Hospital Research Institute, Brompton, S.W.
3782 Schütze, Harrie, Lister Institute, Chelsea Gardens, S.W.
7007 Scott, Alexander T., 8 Parkhurst Road, Holloway.
863 Scott, James, 98 Barons Court Road, West Kensington, W.
990 Scott, Kenneth, 7 Manchester Square, W.
362 †Scott, Sebastian Gilbert, 6 Bentinck Street, W.
1230 †Scott, Sydney, 130 Harley Street, W.
2079 Seaton, Edward Cox, 14 Walpole Street, Chelsea, S.W.
1733 Sefton, Basil, 61 Selsdon Road, West Norwood, S.E.
4272 Seifert, Ernst, German Hospital, Dalston, N.E.
815 Selfridge, Grant (of San Francisco), c/o Bank of Montreal, Threadneedle Street, E.C.
1641 Semon, Henry, 15 Queen Anne Street, Cavendish Square, W.
2001 Semple, Lieut.-Col. Sir David, Mayfield, 33 Madeley Road, Ealing, W.
1009 Sequeira, George William, 34 Jewry Street, E.C.
190 †Sequeira, James Harry, 8A Manchester Square, W.
1473 †Sergeant, John Noel, Newlands House, Tooting Bec Common, S.W.
959 Shaheen, B. I., Guy's Hospital, S.E.
1716 Sharkey, Seymour J., 22 Harley Street, W.

336 Shattock, Clement Edward, 4 Crescent Road, Wimbledon, Surrey.
5 Shattock, Samuel George, 4 Crescent Road, Wimbledon, S.W.
223 Shaw, Harold Batty, 122 Harley Street, W.
6571 Shaw, Thomas Claye, 33 Weymouth Street, W.
7423 Shaw-Mackenzie, John Alexander, 45 Cumberland Mansions, Bryanston Square, W.
1933 Sheppard, Amy, 13 Upper Berkeley Street, Portman Square, W.
249 †Sherren, James, 6 Devonshire Place, W.
2019 †Shields, Douglas A., 17 Park Lane, W.
349 Shipway, Francis E., 24A Portland Place, W.
2060 †Shore, Lieut.-Col. Robert, Colston, Gayton Road, Harrow.
1372 Shuttleworth, George Edward, 8 Lancaster Place, Hampstead, N.W.
320 Sibley, W. Knowsley, 6 Cavendish Place, W.
18 †Silk, John Frederick William, 47 Devonshire Street, W.
1017 †Simmons, Gerald Allpress, 42 Welbeck St., Cavendish Square, W.
442 Simpson, John Pollock, 8 Upper Montagu St., Montagu Square, W.
263 Sims, Mrs. J. Dillwyn, 17 Bentinck Street, W.
221 †Singer, Charles, 53 Upper Brook Street, W.
360 †Skinner, Col. Bruce Morland, Royal Army Medical College, Millbank, S.W.
164 Slater, Charles, St. Ermins Hotel, Westminster, S.W.
163 †Smale, Herbert, 22A Cavendish Square, W.
3 ‡Smale, Morton Alfred, 22A Cavendish Square, W.
1079 Smith, Eustace, 19 Queen Anne Street, W.
1900 †Smith, Frederick John, 138 Harley Street, W.
1637 †Smith, George Frederick Darwall, 8 Grosvenor Street, W.
325 †Smith, Heywood, 40 Portland Court, W.
1394 Smith, J. Percy, 26 Eaton Terrace, S.W.
1745 †Smith, Richard Thomas, 117 Haverstock Hill, N.W.
59 †Smith, Robert Percy, 36 Queen Anne Street, W.
1516 †Smith, William Robert, 36 Russell Square, W.C.
478 Smyth, Francis Radway, 9 Upper Wimpole Street, W.
619 Snowman, Jacob, 11 Shoot-up Hill, Brondesbury, N.W.
1304 Southern, Walter Duckett, 22 Wimpole Street, Cavendish Sq., W.
1889 †Souttar, Henry Sessions, 46 Queen Anne Street, Cavendish Sq., W.
453 Spearman, C., 71 Kensington Gardens Square, W.
267 Spencer, Herbert Ritchie, 104 Harley Street, W.
172 Spencer, Walter George, 2 Portland Place, W.
6594 Spicer, Arthur H., 3 Cleveland Terrace, Hyde Park, W.
1611 †Spicer, W. T. Holmes, 5 Manchester Square, W.
1704 Spilsbury, Bernard Henry, 31 Marlborough Hill, St. John's Wood, N.W.
51 Spokes, Peter Sidney, 4 Portland Place, W.
588 Sprawson, Evelyn C., 26 New Cavendish Street, W.
871 †Sprigge, J. E. Squire, 423 Strand, W.C.
1410 †Squire, J. Edward, 5 Harley Street, W.
2951 Stainer, Dr. Edward, 10 Gloucester Square, W.
6575 Starling, Ernest H., 40 West End Lane, N.W.
573 Stawell, Miss F. Melian, 33 Ladbroke Square, Notting Hill Gate, W.
1134 †Steadman, Frank St. Jermain, 7 Effra Road, Brixton, S.W.
628 †Steeves, George Walter, 9 Cavendish Square, W.
1093 Stephenson, Sydney, 33 Welbeck Street, W.
487 †Stevens, Thomas George, 20 Queen Anne Street, W.

3827 Stewart, Arthur Hastings, 40 Pall Mall, S.W.
7048 Stewart, James, Killydonnell, 28 Glebe Road, Barnes, S.W.
7441 Stewart, Kenneth Trevor, 15 Upper Wimpole Street, W.
368 †Stewart, Purves, 94 Harley Street, W.
824 †Stewart, Staff-Surgeon Robert Wm. Glennan, Admiralty, S.W.
531 Stewart, Thomas Grainger, 54 Queen Anne Street, W.
927 Still, George Frederic, 28 Queen Anne Street, W.
1351 Stoddart, William Henry Butter, Bethlem Royal Hospital, S.E.
7143 Stoney, Florence, 4 Nottingham Place, W.
3834 Stonham, Henry Archibald, 1A College Crescent, Swiss Cottage, N.W.
7040 Stout, Robert, The College, Guy's Hospital, S.E.
7039 Stout, T. Duncan M., The College, Guy's Hospital, S.E.
1242 Stowers, J. H., 128 Harley Street, W.
1193 Stuart-Low, William, 118 Harley Street, W.
3983 Sunderland, Septimus, 11 Cavendish Place, Cavendish Square, W.
1147 Sutherland, George Alexander, 29B Wimpole Street, W.
876 †Swaffield, Walter, 33 Curzon Street, W.
706 Swainson, James Midleton Graham, 9 Welbeck Street, W.
1486 †Swan, Russell Henry Joceyln, 75 Wimpole Street, W.
761 Swanton, James Hutchinson, 40 Harley Street, W.
245 ‡Sykes, Matthew Carrington, 50 Brook Street, Grosvenor Square, W.
1108 Symonds, Charters J., 58 Portland Place, W.

T

3967 Tabuteau, Augustus William, 228A Romford Road, Forest Gate, E.
1772 ‡Tait, Edward Sabine, 48 Highbury Park, N.
3885 ‡Tait, Henry Brewer, 22 Sunningside Road, N.
1316 †Talbot, Alfred George, 13 Willifield Way, Golder's Green, N.W.
1983 Talwalkar, Gajarran Ramchandra, 56 Tavistock Road, Westbourne Park, W.
1649 †Tate, Walter William Hunt, 32 Queen Anne Street, Cavendish Sq., W.
1352 Taylor, Alice, 16 Dover Street, W.
1995 †Taylor, Frederick, 20 Wimpole Street, Cavendish Square, W.
276 Taylor, Gordon, 15 Harley Street, W.
353 Taylor, James, 49 Welbeck Street, W.
524 Taylor, Seymour, 16 Seymour Street, Portman Square, W.
1876 Thackrah, Margaret G., 33 Woodberry Grove, Finsbury Park, N.
7151 Thiele, Francis Hugo, 24 Weymouth Street, W.
2080 Thomas, Edmund Fairfield, 38 Balls Pond Road, N.
236 †Thompson, Arthur Hugh, 36 Weymouth Street, W.
711 †Thompson, Theodore, 94 Portland Place, W.
709 Thomson, H. Campbell, 34 Queen Anne Street, W.
1402 Thomson, Sir StClair, 64 Wimpole Street, London, W.
1684 Thorne, Fred John, Middlesex Hospital, W.
1522 Thorne, May, 148 Harley Street, W.
183 Thorne, William Bezly, 16 Harley Street, W.
250 Thursfield, Hugh, 84 Wimpole Street, W.
3576 Tidy, Dr. H. L., 39 Devonshire Place, W.
1890 †Tilley, Herbert, 72 Harley Street, W.
1594 Timberg, Richard, 150 Harley Street, W.
1909 †Tirard, Nestor, 74 Harley Street, W.
2055 Tod, Hunter F., 11 Upper Wimpole Street, W.
7147 Tomes, Charles S., Welbeck House, Wigmore Street, W.

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1515 TUDOR, DOROTHEA MARY, 14 Endsleigh Street, Tavistock Sq., W.C.
1630 ‡TUKE, THOMAS SEYMOUR, Chiswick House, Chiswick, W.
1712 †TUNNICLIFFE, FRANCIS WHITTAKER, 129 Harley Street, W.
1797 †TURNBULL, GEORGE LINDSAY, 47 Ladbroke Square, W.
714 †TURNER, HENRY WATSON, 59 Wimpole Street, W.
436 TURNER, JOSEPH GEORGE, 59 Wimpole Street, W.
1839 †TURNER, PHILIP, 73 Wimpole Street, W.
3830 TURNER, ROSE, 87 Gower Street, W.C.
367 †TURNER, W. ALDREN, 18 Harley Street, W.
1233 TURNER, WILLIAM, 17 Harley Street, W.
167 ‡TURNEY, HORACE GEORGE, 68 Portland Place, W.
189 TURTLE, GODFREY DE BEC, 81 Cambridge Terrace, Hyde Park, W.
3789 †TWEEDY, MAY, Bedford College, Regent's Park, N.W.

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522 †UNDERWOOD, ARTHUR SWAYNE, 26 Wimpole Street, W.

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3848 WALLACE, ABRAHAM, 39 Harley Street, W.
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767 †WARING, HOLBURT JACOB, 37 Wimpole Street, W.
826 ‡WATERHOUSE, HERBERT FURNIVALL, 7 Wimpole Street, W.
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7447 WEBB, HELEN, 14 Nottingham Place, W.
3630 WEBER, FREDERICK PARKES, 19 Harley Street, W.
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186 †WHITE, CHARLES PERCIVAL, 22 Cadogan Gardens, S.W.
677 †WHITE, E. F., 388 Upper Richmond Road, Putney, S.W.
171 WHITE, WILLIAM HALE, 38 Wimpole Street, W.
7374 WHITEHEAD, Surg.-General HAYWARD READE, Horse Guards, Whitehall, S.W.
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285 ‡THOMAS, Sir A. GARROD, Clytha Park, Newport, Mon.
3605 THOMAS, JABEZ, Swansea.
6837 THOMAS, J. RAGLAN, Exeter.
66 †THOMAS, WILLIAM THELWALL, 84 Rodney Street, Liverpool.
992 THOMPSON, ARTHUR, The Museum, Oxford.
1057 †THOMPSON, CHARLES J. SEATON, Grey Friars, Harrow-on-the-Hill.
7016 THOMPSON, PETER, Tai Bach, Penmaenmawr, North Wales.
717 THOMPSON, WALTER, 30 Park Square, Leeds.
1422 THOMPSON, WILLIAM HENRY, School of Physiology, Trinity College, Dublin.
3908 †THOMPSON, Sir WILLIAM J., 59 Fitzwilliam Square, Dublin.
32 THOMSON, ALEXIS, 39 Drumsheugh Gardens, Edinburgh.
1224 THOMSON, H. TORRANCE, 3 Hillside Crescent, Edinburgh.
47 THOMSON, JOHN, 14 Coates Crescent, Edinburgh.
1045 THOMSON, WILLIAM ERNEST FRANCIS, 2 Somerset Place, Glasgow.
3757 THOMSON, WILLIAM W. D., Pathological Department, Queen's University, Belfast.
7128 †THORBURN, WILLIAM, 2 St. Peter's Square, Manchester.
656 TOMKINSON, JOSEPH GOODWIN, 9 Sandyford Place, Glasgow.
811 TOMLINSON, J. H., Tudor Lodge, Egham.
1851 TOZER, FRANCES M., The Cottage, Betchworth, Surrey.
7058 TREADWELL, OLIVER F. N., Brookfield, Parkhurst, Isle of Wight.
252 TREVES, Sir FREDERICK, Thatched House Lodge, Richmond Park, Kingston-on-Thames.
1980 TRIMBLE, ANDREW, 1 Thorndale Terrace, Belfast.
6769 TURNBULL, ARTHUR, 9 Grosvenor Crescent, Glasgow.
6532 TURNER, A. LOGAN, 27 Walker Street, Edinburgh.
1176 TURNER, GEORGE GREY, The Hawthorns, Osborne Road, Newcastle-on-Tyne.
1007 TURRELL, WALTER JOHN, Cherwell Lodge, Magdalen Bridge, Oxford.
6841 TWEEDIE, ALEXANDER ROBERT, 14 Oxford Street, Nottingham.

1808 †TWEEDY, E. HASTINGS, 6 Fitzwilliam Place, Dublin.
726 ‡TYRRELL, WALTER, Penwith, Shottermill, Surrey.
24 TYSON, WILLIAM JOSEPH, 10 Langhorne Gardens, Folkestone.

U

258 †UHTHOFF, JOHN CALDWELL, Wavertree House, Furze Hill, Brighton.
1288 USHER, C. H., 3 Bon Accord Square, Aberdeen.

V

884 VERNON, HORACE MIDDLETON, 5 Park Town, Oxford.

W

1479 WADE, HENRY, 6 Manor Place, Edinburgh.
1861 WAINWRIGHT, LENNOX, Earls Avenue, Folkestone.
1070 WALKER, HENRY SECKER, 45 Park Square, Leeds.
3851 WALTERS, FREDERICK RUFENACHT, Pinecroft, Farnham, Surrey.
518 WARRINGTON, W. B., 63 Rodney Street, Liverpool.
173 WATERHOUSE, AMYAS THEODORE, 35 Beaumont Street, Oxford.
196 WATERHOUSE, RUPERT, 25 The Circus, Bath.
1205 †WATERSTON, DAVID, Harlow, Northwood, Middlesex.
1824 †WATSON, JOHN HARRY, 68 Bank Parade, Burnley, Lancs.
1393 ‡WATSON-WILLIAMS, PATRICK, Rodney Place, Clifton, Bristol.
1657 WEBSTER, ARTHUR DOUGLAS, 18 Minto Street, Edinburgh.
763 WEBSTER, JOHN HENRY DOUGLAS, Ellesmere, Conway Road, Colwyn Bay.
2027 WELCH, CYRIL H., The Schiff Home of Recovery, Knowle Hill Park, Cobham, Surrey.
1452 †WERNER, L., 31 Merrion Square, Dublin.
7366 †WESTMACOTT, FREDERICK HIBBERT, 8 St. John Street, Manchester.
273 †WHEELER, WILLIAM IRELAND DE COURCY, 59 Lower Mount Street, Merrion Square, Dublin.
694 WHILLIS, SAMUEL S., 66 Jesmond Road, Newcastle-on-Tyne.
262 WHITAKER, SYDNEY, Waterloo, Liverpool.
1535 WHITE, ARTHUR H., Royal College of Surgeons, Dublin.
509 WHITE, CHARLES POWELL, 1 Albemarle Rd., Withington, Manchester.
1126 WHITE, J. DOUGLAS C., Beech Bank, Harrow-on-the-Hill.
7099 †WHITE, SINCLAIR, Dunluce, Ranmoor, Sheffield.
234 †WHITEHEAD, ARTHUR LONGLEY, 31 Park Square, Leeds.
888 WHITELOCKE, RICHARD HENRY ANGLIN, St. Giles' Gate, Oxford.
3780 WHITLA, Sir WILLIAM, College Square North, Belfast, Ireland.
181 WHITNALL, S. ERNEST, The Old Rectory Farm, Kidlington, Oxford.
6709 WHYTE, ROBERT PROSSER, Florence House, Wigan.
284 WIGHAM, JOSEPH T., Albany House, Monkstown, Co. Dublin.
305 WIGMORE, JAMES, 16 Queen Square, Bath.
728 WILD, ROBERT BRIGGS, Broome House, Fallowfield, Manchester.
3858 WILKINSON, Lieut.-Col. EDMUND, 26 Montpelier Road, Ealing.
2191 WILLAN, R. J., 25 Ellison Place, Newcastle-on-Tyne.
6780 WILLETT, EDGAR, Farmleigh, Worth, Sussex.
6752 WILLIAMS, ARNOLD WINKELREID, 1 Belvedere Terrace, Brighton.
3806 WILLIAMS, FREDERIC N., 181 High Street, Brentford, Middlesex.

1330 WILLIAMS, RALPH PAUL, 6 Severn Road, Broomhill, Sheffield.
1128 WILLOUGHBY, WILLIAM GEORGE, Town Hall, Eastbourne.
1622 †WILSON, ALEXANDER, 5 Anson Road, Victoria Park, Manchester.
211 WILSON, CLAUDE, 9 Church Road, Tunbridge Wells.
1881 WILSON, FREDERICK PERERA, 1A Rodney Street, Liverpool.
3992 WILSON, HELEN MARY, Osgathorpe Hills, Sheffield.
1536 WILSON, J. MITCHELL, Westholme, Beverley, East Yorks.
42 †WILSON, THOMAS, 87 Cornwall Street, Birmingham.
184 †WITHINGTON, EDWARD THEODORE, Petit Tor House, St. Mary Church, Torquay, South Devon.
1263 WOOD, LEWIS H., Physiological Laboratory, University, Liverpool.
6776 WOODCOCK, H. DE CARLE, 32 Park Street, Leeds.
7392 †WOODHEAD, GERMAN L. SIMS, Dysart House, Luard Rd., Cambridge.
7111 WOODS, Sir ROBERT H., 39 Merrion Square, Dublin.
718 †WORTHINGTON, ROBERT ALFRED, 30 East Southernhay, Exeter.
565 WRIGHT, ANDREW JOHN METFORD, 60 St. Paul's Rd., Clifton, Bristol.
39 WRIGHT, GEORGE A., The Old Orchard, Bickwell Valley, Sidmouth, South Devon.
769 WRIGLEY, PHILIP ROSCOE, 11 St. John Street, Manchester.
7448 WYNNE, J. DARLEY, Ashbourne, Clonmel.

Y

1168 YOUNG, ERIC ERNEST, 6 Stoke Road, Stoke-on-Trent.

DIVISION III.

OVERSEAS DOMINIONS.

A

1967 ABBOTT, MAUD ELIZABETH SEYMOUR, The Medical Museum, McGill University, Montreal.
1078 ADAMI, J. GEORGE, McGill University, Montreal, Canada.
1223 ADEY, JOHN KELLERMANN (of Melbourne), c/o English, Scottish, and Australian Bank, 38 Lombard Street, E.C.
129 †AIKINS, WILLIAM HENRY BEAUFORT, 134 Bloor Street West, Toronto, Ont., Canada.
3838 †ANDERSON, GEORGE ALLAN, Calgary, Alberta, Canada.
147 †ANDERSON, H. B., 184 Bloor Street East, Toronto, Ont., Canada.
339 ARCHIBALD, EDWARD, 160 Metcalfe Street, Montreal, Quebec, Canada.
137 ARMSTRONG, GEORGE E., 320 Mountain Street, Montreal, Canada.

B

1488 BALFE, JOSEPH HAMILTON, Government Hospital, Durban.
311 BANNERMAN, Surg.-General WILLIAM BURNEY, Madras, India.
439 BARNETT, L. E., 83 Stafford Street, Dunedin, New Zealand.
576 BATCHELOR, FERDINAND C., Otago University, Dunedin, New Zealand.
7180 BEEMER, NELSON H., Mimica, Ont., Canada.
668 BEVAN, LLEWELLYN E. W., Government Veterinary Bacteriologist, Southern Rhodesia.
733 †BINNEY, E. H., 195 Macquarie Street, Sydney, N.S.W.
7369 BIRD, FREDERIC DOUGAN, 41 Spring St. Melbourne, Victoria, Australia.
84 †BIRKETT, HERBERT STANLEY, McGill University, and 252 Mountain Street, Montreal, Canada.
7391 †BISSETT, ERNEST, Port Morien, Nova Scotia, Canada.
134 ‡BLACKADER, ALEXANDER DOUGALL, 236 Mountain Street, Montreal, Canada.
6598 BOOTH, MARY, 77 King Street, Sydney, N.S.W.
7413 †BOULANGER, JOSEPH, Edmonton, Alberta, Canada.
3634 BOWKER, ROBERT STEER, 207 Macquarie Street, Sydney, N.S.W.
281 BRADY, ANDREW JOHN, "Wyoming," 175 Macquarie Street, Sydney, N.S.W., Australia.
441 BRAHMACHARI, UPENDRANATH, 10 Nimtola Ghat St., Calcutta, India.
583 †BRINTON, ARTHUR GREENE, P.O. Box 5852 Johannesburg.
3857 BRODIE, T. GREGOR, University, Toronto, Ont., Canada.
1143 BRUCE, HERBERT A., 64 East Bloor Street, Toronto, Ont., Canada.
198 BURNETT, PHILIP, 161 Stanley Street, Montreal, Canada.
1896 BURNETT, WILLIAM BRENTON, 1301 Cornox Street, Vancouver, Canada.

2168 BURNHAM, FREDERICK W. E., 373 Broadway, Winnipeg.
1433 BURRIDGE, ARTHUR JAMES, 306 Somerset Buildings, Winnipeg, Manitoba, Canada.
299 †BUTLER, GAMALIEL HENRY, 140 Macquarie Street, Hobart, Tasmania.

C

1451 CALVERT, Lieut.-Col. J. T., Medical College, Calcutta, India.
1220 CAMERON, J. H., The University, Toronto.
3795 †CAMPBELL, ALEXANDER M., Winnipeg, Canada.
1165 CAMPBELL, GEORGE ALEXANDER, 459 Avenue Road, Toronto, Ont., Canada.
499 †CAMPBELL, G. GORDON, 123 Crescent Street, Montreal, Quebec, Canada.
561 †CAMPBELL, J. W., 150 Clergy Street, Kingston, Ont., Canada.
2198 CAMPBELL, RANALD DOUGLAS, Hobart, Tasmania.
3603 CARMICHAEL, DUNCAN A., Oshawa, Canada.
7117 CHABOT, JOHN LEO, Ottawa.
622 CHAPMAN, HENRY G., University of Sydney, Sydney, N.S.W.
328 CHATTERJEE, G. C., Medical College, Calcutta, India.
1419 †CHEADLE, BERNARD ARTHUR, Salisbury, Rhodesia, South Africa.
3847 CHISHOLM, WILLIAM, 127 Macquarie Street, Sydney, N.S.W.
1107 CHOKSY, KHAN BAHADUR N. H., Altamont Road, Bombay.
475 CLENDINNEN, FREDERICK JOHN, Haven, Hawksburn, Melbourne, Victoria, Australia.
1803 COKER, ALFRED PHILIP, Auckland, New Zealand.
3619 COLLIE, JOHN R. M., Halifax, Nova Scotia.
3637 COLLISON, JOHN, Red Deer, Alberta, Canada.
321 COLQUHOUN, DANIEL, 44 High Street, Dunedin, New Zealand.
135 †CONNELL, JAMES CAMERON, Queen's University, Kingston, Ont., Canada.
558 CONNELL, WALTER THOMAS, Queen's University, Kingston, Ont., Canada.
3864 COPELAND, GORDON GROTE, 160 Spadina Road, Toronto, Ont., Canada.
2054 COTTON, JAMES MILTON, 20 Bloor Street East, Toronto.
1597 COTTON, THOMAS F., University Club, Montreal, Canada.
1189 CRUICKSHANK, MARGARET BARNETT, "Whareora," Waimate, New Zealand.

D

340 DAS, KEDARNATH, 22 Bethune Row, Calcutta, India.
2051 DIGBY, REGINALD W., 64 Wellington Street, Brantford, Canada.
6761 DOHERTY, MARY AGATHA, 2160 Yonge Street, Toronto, Ont.
1446 †DRIER, EZRA NEWTON, 434 Pinder Street West, Vancouver, Canada.

E

257 EBERTS, EDMOND MELCHIOR VON, 219 Peel Street, Montreal.
7008 EDGAR, JOHN JAMES, Napier, New Zealand.
7018 ‡ELDER, JOHN M., McGill University, Montreal, Canada.
407 ELLIOT, Lieut.-Col. ROBERT HENRY, Shawfield, Egmore, Madras, S. India.

399 ‡Embley, Edward Henry, 245 Latrobe Street, Melbourne, Victoria, Australia.
7042 Ethier, Z. Hormisdas, 21 St. Louis Square, Montreal.
271 Evans, S. Colin, Marine Lines, Bombay, India.

F

7095 Farish, James C., Vancouver, B.C., Canada.
515 Ferguson, H. Lindo, Lower Stuart Street, Dunedin, New Zealand.
7443 Ferguson, J. Young, Renfrew, Ont., Canada.
379 Finley, Frederick G., 273 Bishop St., Montreal, Quebec, Canada.
636 Flashman, J. Froude, 183 Macquarie Street, Sydney, N.S.W.
7182 Fleming-Jones, R., Papua, British New Guinea.
125 Fotheringham, Lieut.-Col. John Taylor, 20 Wellesley St., Toronto, Ont., Canada.

G

306 †Galloway, Herbert P. H., 661 Broadway, Winnipeg, Canada.
3642 Gibson, Frederick Goulbourn, Christchurch, New Zealand.
2084 †Gibson, Harry A., Calgary, Alberta, Canada.
295 Gibson, John Lockhart, Wickham Terrace, Brisbane, Queensland, Australia.
3657 Gibson, Thomas, 164 Metcalfe Street, Ottawa.
6574 Gillespie, Alexander, Edmonton, Alberta, Canada.
814 †Gillies, Sinclair, 153 Macquarie Street N., Sydney, N.S.W.
1345 Goldsmith, Perry, 84 Carlton Street, Toronto, Canada.
3960 Gordon, Andrew R., Toronto, Ont., Canada.
7079 Graham, A. L. Duncan, University of Toronto, Toronto, Canada.
3690 †Graham, Colin, 532 Granville Street, Vancouver.
1625 Gray, George Douglas, British Legation, Peking, China.
2098 Gruner, Oskar C., McGill University, Montreal, Canada.
1499 Guerin, James J., Edgehill Avenue, Montreal, Canada.
1102 †Gurd, David Frazer, 124 Bishop Street, Montreal, Canada.

H

1715 Hamilton, Herbert J., 220 Bloor Street West, Toronto, Canada.
6590 Hamilton, Hubert Douglas, 203-5 Birk's Buildings, Montreal, Canada.
403 Harris, David Fraser, Dalhousie University, Halifax, N.S., Canada.
158 Harrison, Frederick Christopher, 134 Bloor Street West, Toronto, Ont., Canada.
1826 Hattie, William H., Nova Scotia Hospital, Halifax, N.S., Canada.
537 †Hehir, Col. P., Assistant Director of Medical Services, Burma Division, Maymyo, Burma.
6826 Henderson, David A., Toronto, Canada.
1583 †Herbert, William Edward, Wellington, New Zealand.
3903 Hoare, Charles W., Walkerville, Ont., Canada.
789 Hodgetts, Charles Alfred, Ottawa, Ont., Canada.
881 Hughes, George Osborne, 5A Fort Garry Court, Winnipeg, Manitoba.
1613 ‡Hugo, D. de V., Adderley House, Worcester, Cape Colony.
1311 Hutchinson, Harry Stewart, 11 Staveley Road, Poona, India.

I

1661 Iles, Mary M. G., Hazaribagh, Bengal, India.

J

3700 Jagose, Jamshed Rustomji, opposite Grant Road Station, Bombay. India.
1254 ‡Jamieson, James, Toorak, Melbourne, Victoria.
2174 Johnston, Herbert Huff, Wyoming, Macquarie Street, Sydney.
148 Johnston, Samuel, 169 Carlton Street, Toronto, Ont., Canada.
126 †Jones, Ernest, 407 Brunswick Avenue, Toronto, Ont., Canada.
852 †Jones, Guy Carleton, Militia Department, Ottawa, Canada.
152 †Jones, Oswald Meredith, 711 Fort Street, Victoria, B.C.
1037 Jones, W. Ernest, Victoria, Australia.

K

7399 Keith, Robert Donald, Singapore.
985 †Kerry, Richard A., 374 Mountain Street, Montreal, Canada.
301 Kilvington, Basil, "Cronton," Sefton Place, East Camberwell, Melbourne, Victoria, Australia.
3590 †King, F. Truby, Seacliffe, Otago, New Zealand.

L

1920 Labrecque, J. J. Alphonse, Prince Albert, Saskatchewan, Canada.
1769 Lackner, Henry George, Berlin, Ont., Canada.
1532 Langevin, Stephen, Montreal, Canada.
1378 Lapierre, Joseph, Buckingham, Province of Quebec, Canada.
1062 Lassonde, A., Drummondville, Province of Quebec, Canada.
162 †Lawrence, Herman, 82 Collins Street, Melbourne, Australia.
1468 Leathes, John Beresford, The University, Toronto, Canada.
395 ‡Leavy, Edward P., Government Medical Officer, Innisfail, Johnstone River, North Queensland, Australia.
302 Lendon, Alfred Austin, North Terrace, Adelaide, South Australia.
1266 †Lewis, Thomas Hope, Renillera Road, Auckland, N.Z.
3839 †Lincoln, William A., Calgary, Alberta, Canada.
1469 Lindsay, H. C. L., 326 Hastings Street W., Vancouver, B.C.
517 †Liston, Major William Glen, Bombay Bacteriological Laboratory, Parel, Bombay, India.
3849 Lukis, Surg.-General Sir C. Pardey, Calcutta, India.

M

1564 †Macallum, Archibald Bruce, 50 St. George Street, Toronto, Ont.
1449 Macallum, Archibald Byron, The University of Toronto, Toronto, Canada.
687 McCay, Major David, Medical College, Calcutta.
400 MacCormick, A., 185 Macquarie St. North, Sydney, N.S.W., Australia.
1164 †M'Coy, Samuel Harvey, 459 Avenue Road, Toronto, Ont., Canada.
3629 McCrae, John, 160 Metcalfe Street, Montreal.
971 McCullough, John W. S., Parliament Buildings, Toronto, Ont., Canada.

535 †MACDONALD, ANGUS GRAHAM, Medical Officer of Health, Kingston, Jamaica, West Indies.
2161 MCGIBBON, SALTON, Forest, Canada.
529 †MACLAREN, MURRAY, 75 Coburg Street, St. John, N.B., Canada.
2057 MCMAHON, THOMAS F., 116, St. George Street, Toronto, Ont., Canada.
300 MCMURRAY, Dr. WAHAB, "Wyoming," Macquarie Street, Sydney, N.S.W., Australia.
599 ‡MCMURRICH, J. PLAYFAIR, University of Toronto, Toronto, Ont., Canada.
7092 †MACNEILL, JAMES WALTER, Battleford, Sask., Canada.
120 ‡MCPHEDRAN, ALEXANDER, 151 Bloor Street West, Toronto, Ont., Canada.
3884 MCPHEDRAN, FREDERIC MAURICE, 151 Bloor Street West, Toronto, Ont., Canada.
1198 MACTAGGART, DUNCAN DONALD, 1075 Mount Royal Avenue W., Montreal, Quebec, Canada.
923 †MARKS, HERBERT J., Macquarie Street, Sydney, N.S.W.
7030 †MARTIN, ROBERT, Octagon, Dunedin, New Zealand.
1566 †MASINA, H. M., Masina Hospital, Bombay, India.
282 MAUDSLEY, HENRY CARR, 8 Collins St., Melbourne, Victoria, Australia.
101 ‡MAYNARD, Lieut.-Col. FREDERIC PINSENT, 13 Harington Street, Calcutta, India.
3611 †MEEK, HARRY, 330 Queen's Avenue, London, Ontario, Canada.
3615 †MELLISH, JOHN THOMAS, Alberni, Vancouver Island, B.C., Canada.
1947 MENKEL, HERMAN C., 17 Abbott Road, Lucknow, India.
7123 MOORE, SAMUEL, 1203 Bloor Street West, Toronto, Ont., Canada.
1682 MORISON, Capt. JOHN, 18A Civil Lines, Poona, India.
238 MUKERJI, RAJ KRISNA, Inspector of Hospitals, Nepal, North India.
1299 †MÜLLER, CHARLES ALBERT, 263 Elizabeth Street, Sydney, N.S.W.
2074 MURRAY, CHARLES F. K., Kenilworth House, Kenilworth, Cape Town.
942 MURRAY, G. A. E., 24 Plein Street, Johannesburg, Transvaal.

N

872 NAGLE, FRANCIS WILLARD, 104 Sherbrooke Street W., Montreal, Quebec, Canada.

O

3570 O'BRIEN, MARY, Dufferin Hospital, Lucknow, India.
1874 OCHS, ANTHONY, Preston, Ont., Canada.
1040 OGDEN, WILLIAM EDWARD, University Club, 82 King Street, W. Toronto, Ont., Canada.
1356 O'KINEALY, Lieut.-Col. F., Superintendent's House, General Hospital, Calcutta, India.

P

1608 †PATON, DAVID DUNCAN, Breenu, Western Australia.
7108 PEEK, JOHN HAROLD, Sydney, N.S.W.
1485 PRESTON, CHARLES EDWARD, 280 Elgin Street, Ottawa, Canada.
112 †PRIMROSE, ALEXANDER, 100 College Street, Toronto, Ont., Canada.
352 †PURCHAS, ARTHUR CHALLINER, Park Rd. East, Auckland, New Zealand.
1492 PUTTANNA, DODBALLAPUR SIVAPPA, Dodballapur, Mysore Province, India.

R

1935 RAMSEY, G. STUART, 6 St. Famille Street, Quebec, Canada.
1748 RANDLE, JOHN, Lagos, West Africa.
1971 RAO, K. V. RAMANA, 24 Chandrappa Mudali Street, Sowcarpet, Madras, India.
7004 †REASON, CLIFFORD HAMILTON, London, Ont., Canada.
7005 †REASON, HENRY THOMAS, London, Ontario, Canada.
1122 †REDMAN, WILLIAM EDWARD, Picton, New Zealand.
940 REEVE, RICHARD A., 48 Bloor Street E. (corner of Park Road), Toronto, Canada.
309 ROBERTSON, Major J. C., Sanitary Commission with the Government of India, Simla, India.
341 †ROW, R., Greylands, Marine Lines, Fort, Bombay, India.
1199 RUDOLF, ROBERT DAWSON, 302 Bloor Street W., Toronto, Ont., Canada.
1109 †RUSSEL, COLIN K., 218 Bishop Street, Montreal, Canada.
618 †RYAN, EDWARD, Queen's University, Kingston, Ont., Canada.

S

4320 SAUNDERS, JOHN H., Box 142, Perth, Western Australia.
1793 SCOTT, JOHN GEORGE, 265 Elgin Street, Ottawa, Canada.
153 †SHEPHERD, FRANCIS J., 152 Mansfield Street, Montreal, Quebec, Canada.
106 SHIVDAS, S. P., Director Vaccine Therapy Institute, Empire Buildings, Hornby Road, Fort, Bombay, India.
850 †SMITH, ARTHUR DALTON, Mitchell, Ont., Canada.
310 SMITH, DAVID KING, 22 Wellesley Street, Toronto, Ont., Canada.
845 †SMITH, HUGH, The Somerset Hospital, Cape Town.
7091 SMITH, ISSACHAR REUBEL, Toronto, Ont., Canada.
483 SMITH, R. W. BRUCE, Parliament Buildings, Toronto, Ont., Canada.
222 †STARR, CLARENCE L., 224 Bloor Street West, Toronto, Ont., Canada.
3632 STENHOUSE, JOHN, 175 Bloor Street East, Toronto, Ont., Canada.
7105 STEWART, ALEXANDER DRUMMOND, 243 Prince Arthur Street West, Montreal, Canada.
1811 STEWART, JOHN, 28 South Street, Halifax, Nova Scotia.
7109 STEWART, W. GRANT, 61 Sherbrooke Street West, Montreal, Canada.
556 STUART, T. P. ANDERSON, Dean of the Faculty of Medicine, University, Sydney, N.S.W., Australia.
574 SURVEYOR, N. F., 145 Queen's Road, Bombay, India.
283 SYME, GEORGE ADLINGTON, 19 Collins Street, Melbourne, Victoria, Australia.

T

1316 †TALBOT, ALFRED GEORGE, Christchurch, New Zealand.
1809 TALBOT, LEONARD SMITH, Timaru, New Zealand.
144 THIRD, JAMES, 67 West Street, Kingston, Ont., Canada.
474 THOMPSON, J. ASHBURTON, Department of Public Health, Sydney, N.S.W., Australia.
124 TODD, J. L., Medical Building, McGill University, Montreal, Quebec, Canada.
3902 TROTT, DUDLEY COX, Hamilton, Bermuda.
6764 TUCKER, BENJAMIN ALEXANDER, Eighth Avenue, Mayfair, Johannesburg, South Africa.

V

348 VINCENT, SWALE, University of Manitoba, Winnipeg, Canada.

W

176 WALKER, DUNBAR, Army and Navy Stores, Bombay, India.
3604 WALLACE, WILLIAM GORDON, Metcalfe, Ont., Canada.
1799 WALTON, HERBERT JAMES, King George's Medical College, Lucknow, India.
1662 †WARREN, CHARLES FRANK, Wyoming, Macquarie St., Sydney, N.S.W.
662 WATSON, BENJAMIN PHILP, 320 Bloor Street West, Toronto, Ont., Canada.
6560 WATSON, MALCOLM, Klang, Federated Malay States.
3769 WEEKES, WILLIAM JAMES, 436 Waterloo Street, London, Canada.
1843 WELLS, ROBERT BRUCE, Norwood Block, Edmonton, Canada.
296 WELSH, D. A., Department of Pathology, University, Sydney, N.S.W., Australia.
1384 WHITE, ERNEST HAMILTON, 298 Stanley Street, Montreal, Canada.
1880 WHITE, R. G., Turf Club, Cairo.
7118 WHITMORE, GEORGE H., 425 Clendenan Avenue West, Toronto, Canada.
1423 †WHITTLE, EDWARD DENIS, Singapore, Straits Settlements.
7433 WILLIAMS, ERNEST HARRY, Dunedin, New Zealand.
977 WILLIAMS, GRIFFITH JONES (of South Africa).
3609 †WILLIAMS, HADLEY, London, Ont., Canada.
1945 WILLIAMS, HORACE J., Plumstead, Cape Town, S. Africa.
3691 †WILSON, DAVID H., Vancouver, B.C., Canada.
303 ‡WILSON, JAMES THOMAS, University of Sydney, Sydney, N.S.W., Australia.
136 †WILSON, ROBERT, 596 Wellington Street, Montreal, Quebec, Canada.
155 WRIGHT, ADAM H., 30 Gerrard Street East, Toronto, Ont., Canada.
3793 WRIGHT, R. PERCY, 46 St. Louis Street, Quebec, Canada.

Y

1377 YOUNG, HUGH CORBITT TAYLOR, 221 Macquarie Street, Sydney, N.S.W., Australia.

DIVISION IV.

UNITED STATES OF AMERICA.

A

308 ABBE, ROBERT, 13 West 50th Street, New York City, U.S.A.
1934 ABBE, TRUMAN, Washington, D.C., U.S.A.
781 ABBOTT, EDVILLE GERHARDT, 310 Y.M.C.A. Building, Portland, Maine, U.S.A.
116 ABBOTT, WALLACE C., 4753 E. Ravenswood Park, Chicago, Ill., U.S.A.
879 ABEL, JOHN J., Johns Hopkins University, Baltimore, U.S.A.
1628 †ADAMS, W. HERBERT, Faison, North Carolina, U.S.A.
827 ADDISON, WILLIAM H. F., University of Pennsylvania, Philadelphia, U.S.A.
1301 ADLER, HARRY, 1718 Eutaw Place, Baltimore, Maryland, U.S.A.
117 ALBEE, FRED HOUDLETT, 40 East 41st Street, New York City, U.S.A.
81 ALBERT, HENRY, College of Medicine, University of Iowa, Iowa City, Iowa, U.S.A.
1350 ALDERSON, HARRY E., Stanford University, Medical Dept., San Francisco, California, U.S.A.
1187 ‡ALDERSON, MADISON E., Russellville, Kentucky, U.S.A.
378 ALKIER, STANISLAW JOSEPH, 299 Broadway, Brooklyn, N.Y., U.S.A.
7014 ALLABEN, JOHN ELMER, Rockford, Ill., U.S.A.
527 †ALLEN, ALFRED REGINALD, 2013 Spruce Street, Philadelphia, P.A., U.S.A.
6843 †ALLISON, NATHANIEL, St. Louis, U.S.A.
2177 ANDERS, HOWARD S., 115 South 20th Street, Philadelphia, U.S.A.
6786 ANDERSON, HENRY GRAY, Waterbury, Conn., U.S.A.
818 †ANDERSON, WINSLOW, 1065 Sutter Street, San Francisco, California.
7414 †ANDRIES, JOSEPH H., 75 Adelaide Street, Detroit, Michigan, U.S.A.
825 ARMSTRONG, ELWOOD, Greenleaf, Kansas, U.S.A.
1349 ARNOLD, HAROLD SEARS, 122 College Street, Newhaven, Conn., U.S.A.
3692 ASHTON, CHARLES H., Franklin, Pa., U.S.A.
1741 AUTEN, FRANK E., Belleville, Ill., U.S.A.

B

161 BABCOCK, W. WAYNE, 2033 Walnut Street, Philadelphia, Pa., U.S.A.
1103 †BACON, GORHAM, 47 West 54th Street, New York, U.S.A.
911 BAEHR, GEORGE, 1 East 100th Street, New York, U.S.A.
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7148 BALDWIN, WALTER F., University Hospital, San Francisco, U.S.A.
943 †BARKAN, ADOLF, c/o Mercantile National Bank, San Francisco, U.S.A.
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7068 ‡BARNHILL, JOHN F., Pennway Building, Indianapolis, Indiana, U.S.A.
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1866 BARUCH, SIMON, 51 West 70th Street, New York, U.S.A

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2090 Beedle, Gordon A., 706 Bryant Buildings, Kansas City, U.S.A.
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534 Bergener, Gustav J., 803 Head Building, San Francisco, Cal., U.S.A.
752 Bierhoff, Frederic, 10 West 61st Street, New York City, U.S.A.
102 ‡Biggs, Hermann M., 113 West 57th Street, New York City, U.S.A.
67 Billings, Frank, 335 East 22nd Street, Chicago, Ill., U.S.A.
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3959 †Blaine, Edward S., 710 Majestic Building, Milwaukee, Wis., U.S.A.
3774 †Blair, Edward G., 404 Bryant Buildings, Kansas City, Mo., U.S.A.
585 †Blair, William Wightman, Woodland Road, Pittsburg, Pa., U.S.A.
156 †Blake, Clarence John, 226 Marlborough St., Boston, Mass., U.S.A.
508 Bliss, M. A., Humboldt Building, St. Louis, Mo., U.S.A.
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7006 Botsford, Mary Elizabeth, 807 Francisco Street, San Francisco, California.
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7121 Bradley, Edward R., 317 Black Building, Los Angeles, U.S.A.
562 †Brewer, George Emerson, 61 West 48th St., New York City, U.S.A.
1494 Bridges, Willson Orton, Omaha, Nebraska, U.S.A.
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7122 Bucknell, Howard, Atlanta, Georgia, U.S.A.
3801 Buckner, Leigh, Roanoke, Virginia, U.S.A.
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7131 CAVE, HENRY W., Johns Hopkins Hospital, Baltimore, Maryland, U.S.A.
6845 CHAPOTON, E. A., 372 Woodward Avenue, Detroit, Mich., U.S.A.
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